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Evaluation of the Association Between Preeclampsia Severity and Hematological and Biochemical Parameters

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ABSTRACT

Objective: Preeclampsia is a multisystemic disorder characterized by inflammation, platelet dysfunction, and target-organ involvement. Early identification of disease severity is crucial for improving maternal and fetal outcomes. This study aimed to evaluate the association between preeclampsia severity and hematological, biochemical, and derived inflammatory parameters.

Methods: This retrospective study included 135 pregnant women diagnosed with preeclampsia between 2020 and 2025 at a single tertiary center. Patients were classified as having mild or severe preeclampsia based on American College of Obstetricians and Gynecologists criteria. Hematological markers [hemoglobin, white blood cell (WBC), platelet, mean platelet volume (MPV)], biochemical parameters [aspartate aminotransferase (AST), alanine aminotransferase (ALT), creatinine], and derived indices [neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), platelet-to-neutrophil ratio (PNR)] were recorded. Group comparisons were performed using Student's t-test, Mann-Whitney U test, or Fisher's exact test as appropriate. Statistical significance was set at $p<0.05$.

Results: Severe preeclampsia was associated with significantly higher hemoglobin ($p=0.019$), WBC ($p=0.005$), neutrophil counts ($p=0.004$), and AST, ALT, and creatinine levels ($p=0.003$, $p<0.001$, $p=0.002$, respectively). Platelet counts were lower in severe cases ($p=0.0411$). No significant differences were found in MPV ($p=0.833$), NLR ($p=0.614$), or PLR ($p=0.109$). PNR was significantly higher in the mild group ($p=0.001$), whereas NLR and PLR did not differ significantly.

Conclusion: Severe preeclampsia is characterized by intensified inflammation, increased platelet consumption, and more pronounced organ involvement. Among the inflammatory indices, PNR differed significantly between severe and mild disease groups, whereas NLR and PLR did not differ significantly. Combined assessment of hematological and biochemical markers may provide a more comprehensive and reliable approach for evaluating preeclampsia severity. PNR may represent a potential supportive parameter associated with disease severity; however, further studies, including diagnostic performance analyses, are required.

Keywords: Preeclampsia, disease severity, platelet-to-neutrophil ratio, inflammatory markers

INTRODUCTION

Preeclampsia is a multisystemic and heterogeneous syndrome affecting approximately 3-5% of pregnancies and is one of the leading causes of maternal and perinatal morbidity and mortality (1). The clinical spectrum ranges from mild cases to severe forms accompanied by hepatic and renal dysfunction, hematologic abnormalities, and neurological manifestations. Therefore, early and accurate assessment of disease severity is critically important for both maternal and fetal prognosis (1).

The most widely accepted mechanism in the etiopathogenesis of preeclampsia involves abnormal trophoblast invasion and insufficient spiral artery remodeling, leading to placental hypoperfusion and ischemia (2,3). This hypoxic process triggers the release of anti-angiogenic factors, oxidative stress products, and proinflammatory mediators from the placenta into the maternal circulation. These substances induce systemic endothelial dysfunction, vasoconstriction, and activation of coagulation pathways, thereby shaping the clinical presentation of the disease (3,4). Consequently, the magnitude of the inflammatory response becomes traceable through alterations in hematological and biochemical parameters (4,5).

This systemic inflammatory response during pathogenesis leads to measurable alterations in peripheral blood cells. Neutrophil activation, a decrease in lymphocyte count, and impaired platelet function constitute the hematologic reflections of the preeclamptic process (5,6). Therefore, complete blood count parameters are considered inexpensive and reproducible indicators that reflect

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the severity of inflammation. Derived inflammatory indices—particularly the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR)—have been reported to play a significant role in the pathophysiology of preeclampsia (6-8). Specifically, elevated NLR is associated with neutrophil-mediated oxidative stress and endothelial activation, whereas increased PLR may reflect platelet activation and the vascular response (7,8).

Furthermore, parameters associated with platelet function—such as mean platelet volume (MPV)—and newly derived indices, including the platelet-to-neutrophil ratio (PNR), have also been proposed to correlate with disease severity (8,9). However, evidence regarding the clinical utility of these ratios in distinguishing the severity of preeclampsia remains limited.

Biochemical parameters reflect the manifestation of the systemic inflammatory response in target organ injury. Elevations in aspartate aminotransferase (AST) and alanine aminotransferase (ALT) indicate hepatic microcirculatory disturbance and endothelial damage, whereas increased creatinine levels reflect reduced renal perfusion and glomerular endotheliosis (10,11). The fact that serum creatinine—which physiologically decreases during pregnancy—shows a significant rise in preeclampsia further supports the renal involvement of this systemic process (11).

Evaluating hematological parameters [hemoglobin, white blood cell (WBC), platelet, MPV] and derived inflammatory ratios (NLR, PLR, PNR), together with biochemical markers (AST, ALT, creatinine), enables comprehensive characterization of the inflammation-platelet activation-organ involvement axis of the disease. The present study aimed to assess the associations between hematological and biochemical parameters and disease severity in cases of mild and severe preeclampsia.

METHODS

Study Design and Ethical Approval

This retrospective study involved reviewing the medical records of pregnant women diagnosed with preeclampsia and followed between 2020 and 2025 in the Department of Obstetrics and Gynecology at University of Health Sciences Türkiye, Gaziosmanpaşa Training and Research Hospital. Ethical approval for the study was obtained from the University of Health Sciences Türkiye, University of Health Sciences Türkiye, Gaziosmanpaşa Training and Research Hospital, Clinical Research Ethics Committee (decision no: 17, date: 21.03.2018), and the scope of the approval was revised on 15 October 2025 to update the research period to 2020-2025. All procedures were carried out in accordance with the principles of the Declaration of Helsinki.

Inclusion and Exclusion Criteria

Pregnant women diagnosed with preeclampsia after the 20th week of gestation who had complete laboratory data were included in the study. Exclusion criteria consisted of chronic hypertension, diabetes mellitus, hematologic disorders, chronic renal or hepatic insufficiency, multiple pregnancy, and active infection. Based on these criteria, a total of 135 patients were included in the study.

Patient Grouping

The diagnosis of preeclampsia was established according to criteria defined by international guidelines. Patients were categorized into two groups—mild preeclampsia and severe preeclampsia—according to the classification of the American College of Obstetricians and Gynecologists (ACOG). Blood pressure values, proteinuria levels, and clinical findings were used solely for grouping purposes; therefore, they were neither included in the analytical evaluations nor reported in the tables.

Collected Variables

Demographic data (age, gestational week, and mode of delivery) and hematological parameters (hemoglobin, WBC, platelet count, MPV, MCV, neutrophils, lymphocytes, monocytes, eosinophils, and basophils) were recorded. In addition, derived inflammatory indices—including the NLR, PLR, and PNR—were calculated. As part of the biochemical assessment, AST, ALT, and creatinine levels were evaluated.

Statistical Analysis

Continuous variables were presented as mean ± standard deviation or median [interquartile range (IQR)] according to data distribution. Normality was assessed using the Shapiro-Wilk test, and homogeneity of variances was evaluated with Levene's test. For variables with a normal distribution, the Student's t-test was used; for those not normally distributed, the Mann-Whitney U test was applied. Categorical variables were expressed as numbers and percentages. Because expected cell counts fell below 5 in some categories, Fisher's exact test was preferred for comparing delivery modes. A p-value <0.05 was considered statistically significant. Based on the post-hoc power analysis performed using data from Mohamed and Ali (10), the current sample size was determined to provide approximately 80% statistical power for the analyses.

RESULTS

A total of 135 patients were included in the study, comprising 82 mild and 53 severe preeclampsia cases. The two groups were similar in terms of maternal age [median (IQR): 30.00 (25.00-33.00) vs. 28.00 (24.00-34.75) years, p=0.535), gravidity [median (IQR): 2.00 (1.00-4.00) vs. 3.00 (2.00-4.00), p=0.089], and parity [median (IQR): 2.00 (1.00-2.00) vs. 2.00 (1.00-2.00), p=0.159). However, gestational age at delivery was significantly lower in the severe preeclampsia group compared with the mild preeclampsia group [median (IQR): 36.00 (36.00138.00) vs. 38.00 (37.00139.00) weeks, p<0.001]. The mode of delivery did not differ significantly between the severe and mild preeclampsia groups; cesarean section was the predominant route in both (96.2% vs. 93.9%, respectively; p=0.710) (Table 1).

The comparison of hematological and biochemical parameters is presented in Table 2. Hemoglobin levels were significantly higher in the severe preeclampsia group compared with the mild group (p=0.019). WBC values were also significantly elevated in the severe group (p=0.005), whereas platelet counts were significantly

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higher in the mild group (p=0.041). Among the biochemical parameters, the levels of AST, ALT, and creatinine were significantly higher in the severe preeclampsia group (p=0.003, p<0.001, and p=0.002, respectively). No significant difference in MPV values was observed between the groups (p=0.833).

Inflammatory cell counts and derived indices are presented in Table 3. Neutrophil counts were significantly higher in the severe preeclampsia group than in the mild group (p=0.004). There was no significant difference between the groups in terms of lymphocyte counts (p=0.535). NLR and PLR values were similar between the two groups, with no statistically significant differences (p=0.614 and p=0.109, respectively). In contrast, PNR levels were significantly higher in the mild preeclampsia group (p=0.001).

DISCUSSION

In this study, hematological, biochemical, and derived inflammatory indices were compared between mild and severe preeclampsia cases, and the biological manifestations of the disease along the inflammation-platelet activation-organ involvement axis were evaluated. The findings indicate that, as the severity of preeclampsia increases, marked deterioration occurs in both hematological and biochemical profiles.

The significantly higher hemoglobin levels observed in the severe preeclampsia group are consistent with the fundamental pathophysiological mechanisms of the disease. Placental hypoperfusion and widespread endothelial dysfunction lead to intravascular fluid loss and a reduction in plasma volume, resulting

Table 1. Demographic and obstetric characteristics of the study groups

Variable	Severe (n=53)	Mild (n=82)	p-value
Age (years)	30.00 (25.00-33.00)	28.00 (24.00-34.75)	0.535
Gravidity	2.00 (1.00-4.00)	3.00 (2.00-4.00)	0.089
Parity	2.00 (1.00-2.00)	2.00 (1.00-2.00)	0.159
Gestational age (weeks)	36.00 (36.00-38.00)	38.00 (37.00-39.00)	<0.001
Mode of delivery - CS	51 (96.2%)	77 (93.9%)	0.710*
Mode of delivery - NSD	2 (3.8%)	5 (6.1%)	-

Continuous variables are presented as medians (IQR, 25th-75th percentiles), and categorical variables are presented as numbers and percentages. Because the distributions were non-normal, the Mann-Whitney U test was used for between-group comparisons
*: The comparison of the mode of delivery was performed using Fisher's exact test, CS: Cesarean section, NSD: Normal spontaneous delivery

Table. 2. Hematological and biochemical laboratory parameters

Parameter	Severe (n=53)	Mild (n=82)	p-value
Hemoglobin (g/dL)	12.00±1.45	11.39±1.49	0.019
WBC (10^9/L)	12.10 (9.90-14.30)	9.91 (9.00-12.57)	0.005
Platelet (10^9/L)	204.06±72.45	229.56±65.93	0.041
MPV (fL)	10.00±1.08	9.96±1.14	0.833
AST (IU/L)	24.00 (19.00-71.00)	20.90 (16.00-26.85)	0.003
ALT (IU/L)	16.00 (10.00-50.25)	10.00 (8.25-17.75)	<0.001
Creatinine (mg/dL)	0.57 (0.51-0.71)	0.52 (0.48-0.59)	0.002

Hemoglobin, platelet count, and MPV were compared using Student's t-test; all other parameters were compared using the Mann-Whitney U test due to their non-normal distribution. Parameters analyzed with Student's t-test are presented as mean ± standard deviation, whereas those analyzed with the Mann-Whitney U test are presented as median (interquartile range, 25th-75th percentile)
WBC: White blood cell, MPV: Mean platelet volume, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase

Table 3. Inflammatory cell counts and derived indices

Parameter	Severe (n=53)	Mild (n=82)	p-value
Neutrophils (10^3/μL)	8.60 (6.71-10.30)	7.09 (5.85-8.68)	0.004
Lymphocytes (10^3/μL)	2.07 (1.66-2.58)	2.07 (1.64-2.44)	0.535
NLR	3.00 (2.00-5.00)	3.00 (2.00-4.00)	0.614
PLR	93.00 (70.00-135.00)	108.50 (86.00-139.75)	0.109
PNR	24.00 (16.00-33.00)	29.00 (25.00-38.00)	0.001

All variables are presented as medians (interquartile range, 25th-75th percentiles). All parameters in this table were compared using the Mann-Whitney U test because the data were not normally distributed
NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, PNR: Platelet-to-neutrophil ratio

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in relative hemoconcentration and consequently elevated hemoglobin levels (1). This mechanism is considered part of the vasoconstrictive and hypovolemic response frequently seen in severe preeclampsia. However, findings in the literature regarding the relationship between hemoglobin levels and preeclampsia severity are inconsistent; while some studies have reported no significant association (10), others have indicated that hemoglobin levels do not parallel the clinical presentation (12). The increase in hemoglobin observed in our study aligns with the volume contraction seen in severe cases, suggesting that this parameter may provide supportive information for assessing disease severity.

The marked elevation of WBC and neutrophil counts in the severe preeclampsia group reflects the systemic inflammatory component of the disorder. Neutrophil activation, oxidative stress generation, and endothelial injury play central roles in the pathogenesis (2,4). Neutrophilia has been associated with preeclampsia severity in numerous studies, consistent with the findings of our study (5,10,13). The wide standard deviation of neutrophil counts in the mild group may indicate variability in individual inflammatory responses. The absence of a significant difference in lymphocyte counts suggests that the neutrophil-dominant inflammatory response may not have resulted in pronounced lymphocyte suppression.

The significantly lower platelet counts observed in the severe group may be explained by platelet consumption secondary to coagulation activation in preeclampsia. Endothelial injury, exposure of the subendothelial matrix, and increased microthrombus formation lead to the withdrawal of platelets from the circulation (3,8). This finding is consistent with reports in the literature indicating that thrombocytopenia is associated with disease severity (8,14,15).

The absence of a significant difference in MPV levels aligns with the conflicting findings reported in the literature. While some studies have suggested that MPV may indicate increased platelet activation, others have demonstrated that it is not a reliable discriminator (7,14). Therefore, MPV alone may not serve as a dependable marker.

The markedly elevated AST, ALT, and creatinine levels observed in the severe preeclampsia group represent biochemical evidence of endothelial injury and target-organ involvement. Hepatocellular impairment and microcirculatory disturbance lead to increases in hepatic enzymes, whereas glomerular endotheliosis and reduced filtration contribute to elevated creatinine levels (1,10,11). Our findings are largely consistent with the literature reporting more pronounced renal and hepatic dysfunction in severe preeclampsia (11,12).

The absence of significant differences in NLR and PLR values between the two groups suggests these ratios may be influenced by population characteristics, the timing of sampling, and individual variability in inflammatory burden. Although several studies have reported increased NLR and PLR levels in severe preeclampsia (6,7,13), other series have found no significant differences (4,9). Moreover, evidence indicates that the performance of derived inflammatory indices in distinguishing disease severity is not consistently reliable (16,17). Our findings align with the latter group of studies, suggesting that NLR and PLR may have limited value in differentiating severe preeclampsia.

One of the most notable findings of this study is that PNR values were significantly higher in the mild group compared with the severe group. Since PNR represents the ratio of platelet count to neutrophil count, it simultaneously reflects platelet consumption and neutrophil elevation. Although data on PNR in the literature are limited, it has been suggested that this index may decrease in conditions where inflammation and platelet activation increase concurrently (14,18). Our results indicate that PNR values differed significantly between mild and severe preeclampsia groups, suggesting a potential association with disease severity. However, since diagnostic performance analyses such as ROC/AUC were not performed in the present study, the true discriminatory value of PNR cannot be determined. Further prospective studies are required to evaluate its diagnostic accuracy.

Moreover, the ease of calculating PNR from routine complete blood count parameters enhances its applicability in clinical practice.

Overall, the severe preeclampsia group demonstrated markedly increased inflammation (WBC, neutrophils), enhanced platelet consumption (reduced platelet count, decreased PNR), and more pronounced organ involvement (AST, ALT, creatinine). These findings support the progressive, multisystemic nature of preeclampsia. The lack of significant differences in NLR and PLR reflects the inter-population variability of these biomarkers (19,20) and suggests that PNR may represent a potentially useful adjunct parameter. A combined assessment of hematological and biochemical markers may offer a more comprehensive and reliable approach for determining disease severity.

This study has several notable strengths. First, all patients were followed at the same center, and laboratory measurements were performed under a single standardized protocol, which reduced biological and technical variability and thereby enhanced the internal validity of the results. The classification of mild and severe preeclampsia according to ACOG criteria further strengthened the accuracy of group stratification. Additionally, the simultaneous evaluation of hematological, biochemical, and derived inflammatory indices provided a comprehensive approach—integrating inflammation, platelet activation, and organ involvement—and enabled a multidimensional assessment of disease severity. Furthermore, the analysis of PNR as a severity marker, despite the limited data in the literature, represents one of this study's original contributions.

Study Limitations

This study has several limitations. Due to its retrospective design, the study's ability to establish causal relationships is limited, and missing records may introduce potential bias. The sample size—particularly the relatively small number of patients in the severe

preeclampsia group—may restrict the statistical power of the study. Measuring laboratory parameters at a single time point may not fully capture the dynamic nature of the inflammatory response. Despite strict exclusion criteria, residual confounding related to subclinical inflammation or unmeasured variables cannot be completely ruled out. Although gestational age was included as a baseline characteristic, the absence of multivariable analysis limits the ability to fully adjust for potential confounders. Additionally, the single-center design and a lack of evaluation across populations with different demographic characteristics limit the generalizability of the findings.

CONCLUSION

Severe preeclampsia is characterized by markedly increased inflammation, enhanced platelet consumption, and more pronounced organ involvement. While NLR and PLR may not always be sufficient to distinguish disease severity, PNR differed significantly between mild and severe preeclampsia groups and may serve as a potential adjunct marker. However, its clinical diagnostic value should be confirmed in larger prospective studies. A combined assessment of hematological and biochemical markers may provide a more comprehensive and reliable approach for determining preeclampsia severity. Supporting these findings with larger sample sizes and prospective study designs will strengthen their applicability in clinical practice.

Ethics

Ethics Committee Approval: Ethical approval for the study was obtained from the University of Health Sciences Türkiye, Gaziosmanpaşa Training and Research Hospital, Clinical Research Ethics Committee (decision no: 17, date: 21.03.2018).

Informed Consent: Retrospective study.

Footnotes

Author Contributions: Concept - H.B.B., S.K.; Design - H.B.B., S.S.; Data Collection and/or Processing - E.S.C., B.B.; Analysis and/or Interpretation - S.S., S.K.; Literature Search - E.S.C., S.K.; Writing - H.B.B.

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