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Can we predict absent fetal heart rate variability with recurrent late decelerations by using hematologic markers?

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Abstract

This study aimed to investigate the predictive utility of hematologic markers for absent fetal heart rate variability with recurrent late decelerations during uterine contractions. This retrospective case-control study was conducted at Selçuk University Faculty of Medicine between January 2019 and July 2026. A total of 546 pregnant women were enrolled: 182 patients with Category III fetal heart rate patterns (study group) and 364 women with Category I non-stress test patterns who underwent spontaneous vaginal delivery (control group), according to NICHD criteria. Hematologic markers including Hb, PDW, MPV, NLR, and PLR were compared between groups. ROC curve analysis and univariate and multivariate logistic regression analyses were performed.PDW was significantly lower in the study group (16.3±1.7 vs. 17.8±0.8, p<0.001), whereas MPV and NLR were significantly higher (p<0.001). ROC analysis revealed that PDW had the highest discriminative performance (AUC: 0.850, 95% CI: 0.812–0.889). Multivariate logistic regression identified PDW (OR: 0.15), MPV (OR: 2.19), and NLR (OR: 1.13) as independent predictors after adjusting for gestational age, gravida, and BMI. PDW and MPV were identified as independent predictors of absent fetal heart rate variability with recurrent late decelerations, with PDW demonstrating superior discriminative performance at a cut-off value of ≤17.05%. These readily available and inexpensive markers may facilitate antenatal identification of patients at risk for intrapartum fetal distress.

Keywords: Fetal heart rate variability, Late decelerations, Platelet distribution width, Mean platelet volume, Non-Stress Test

Introduction

Prediction and assessment of fetal well-being during labor is of paramount importance in intrapartum fetal monitoring. The primary methods used for evaluating fetal well-being include the Non-Stress Test (NST), biophysical profile, modified biophysical profile, contraction stress test, and fetal movement count. Among these, NST is the most widely used method due to its simplicity, low cost, and ease of application (1). During NST evaluation, multiple variables are assessed, including baseline Fetal Heart Rate (FHR), variability, accelerations, and decelerations, each of which corresponds to distinct clinical patterns. Among these patterns, absent FHR variability with recurrent late decelerations is considered the most ominous, as it primarily reflects fetal oxygen deficiency (1).

In healthy fetuses, transient reductions in fetal blood flow during uterine contractions can be readily compensated, leaving NST traces unaffected (2). The fetus normally tolerates this transient hypoxemia through several compensatory mechanisms, including the high oxygen affinity of fetal hemoglobin, elevated fetal hemoglobin levels, and adequate intervillous reserve capacity (3). However, in fetuses with compromised placental reserve, these compensatory mechanisms become insufficient, resulting in absent FHR variability accompanied by recurrent late decelerations during uterine contractions (3).

Hematologic markers, including the Neutrophil-To-Lymphocyte Ratio (NLR), Platelet-To-Lymphocyte Ratio (PLR), Platelet Distribution Width (PDW), Red Cell Distribution Width (RDW), and Mean Platelet Volume (MPV), represent simple, rapid, and cost-effective parameters that have recently been evaluated for their diagnostic and predictive utility across a wide range of clinical conditions (4–6).

Although hematologic markers have been investigated in various pregnancy-related conditions, including preeclampsia (4), preterm labor (7), recurrent pregnancy loss (8), and gestational diabetes mellitus (9), no study to date has examined their utility in predicting absent FHR variability with recurrent late decelerations. Early identification of patients at risk for this pattern prior to the onset of labor may reduce the likelihood of emergency cesarean delivery and its associated maternal complications, including endometritis and venous

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thromboembolism (10,11). Therefore, this study aimed to investigate whether hematologic markers can serve as predictors of absent FHR variability with recurrent late decelerations during uterine contractions.

Materials and methods

Study design

This retrospective case-control study was conducted at the Department of Obstetrics and Gynecology, Selçuk University Faculty of Medicine, between January 2019 and July 2026. The study was approved by the local institutional ethics committee (Approval number: 2021/270). Patient data were retrieved from hospital medical records.

Patient selection

A total of 546 pregnant women aged 18 to 40 years who were in the active phase of labor with regular uterine contractions were enrolled in this study. The study group comprised 182 patients who developed absent FHR variability with recurrent late decelerations following uterine contractions, either spontaneous or induced, and were classified as Category III according to the NICHD criteria (1,12). The control group consisted of 364 pregnant women with Category I NST patterns who underwent spontaneous vaginal delivery. All fetal distress cases admitted during the study period were consecutively enrolled regardless of gestational age, in order to reflect real clinical practice and avoid selection bias. Emergency cesarean delivery was performed in all study group patients due to non-reassuring FHR tracings.

The following exclusion criteria were applied: multifetal gestation, fetal arrhythmias, fetal growth restriction, fetal anomaly, preeclampsia, placental abruption, systemic diseases, smoking history, and uterine hyperstimulation on NST traces.

Data collection

A detailed medical history was obtained from all participants, and a physical examination was performed upon admission to the Labor and Delivery Room (LDR). Maternal weight and height were recorded, and Body Mass Index (BMI) was calculated as weight in kilograms divided by height in meters squared. Gestational age was determined according to the last menstrual period or first-trimester ultrasound findings.

Blood samples for complete blood count with differentials, including Hemoglobin (Hb), hematocrit (Hct), White Blood Cell Count (WBC), neutrophil count, lymphocyte count, platelet count, Red Cell Distribution Width (RDW), Platelet Distribution Width (PDW), Mean Platelet Volume (MPV), Neutrophil-To-Lymphocyte Ratio (NLR), and Platelet-To-Lymphocyte Ratio (PLR), were obtained upon admission to the LDR prior to initiation of any medical treatment. All samples were processed within two hours of venipuncture using an automated blood cell counter. NLR was calculated as the ratio of neutrophil count to lymphocyte count, and PLR was calculated as the ratio of platelet count to lymphocyte count.

Fhr pattern classification

FHR was continuously monitored using Sunray SRF618B6 electronic fetal monitors. FHR baseline, variability, accelerations, decelerations, and sinusoidal pattern were defined and classified according to the three-tier FHR classification system recommended by the National Institute of Child Health and Human Development (NICHD) in 2008 (1,12).

Category I FHR tracings include all of the following: baseline FHR of 110 to 160 beats per minute (bpm), moderate FHR variability (6 to 25 bpm), absence of late or variable decelerations, and presence or absence of early decelerations and accelerations.

Category II FHR tracings are indeterminate and do not meet criteria for either Category I or Category III.

Category III FHR tracings include at least one of the following: absent FHR variability with recurrent late decelerations, absent FHR variability with recurrent variable decelerations, absent FHR variability with bradycardia, or a sinusoidal pattern.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 21.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation or median (minimum-maximum) based on the distribution characteristics. The normality of continuous variables was assessed using the Shapiro-Wilk and Kolmogorov-Smirnov tests, as well as histogram inspection. Differences between the study and control groups were evaluated using the independent samples t-test for normally distributed variables and the Mann-Whitney U test for non-normally distributed variables.

Receiver Operating Characteristic (ROC) curve analysis was performed to determine the optimal cut-off values and discriminative performance of PDW, MPV, NLR, and PLR for predicting absent FHR variability with recurrent late decelerations. The Area Under the Curve (AUC) with 95% Confidence Intervals (CI) was calculated for each marker. Optimal cut-off values were determined based on the Youden index (sensitivity + specificity - 1).

Univariate and multivariate binary logistic regression analyses were performed to identify independent predictors of absent FHR variability with recurrent late decelerations. Variables that showed statistically significant differences between the groups in pairwise comparisons were included in the univariate analysis. Gestational age, gravida, and BMI, which differed significantly between the groups, were incorporated as covariates in the multivariate model to control for potential confounding effects. PCT was initially included in the analysis; however, it was excluded from the final model due to complete separation in the binary logistic regression, which yielded unreliable parameter estimates. Adjusted Odds Ratios (OR) and 95% Confidence Intervals (CI) were calculated for each variable.

Sample size calculation was performed using G*Power version 3.1.9.2 (13). For the independent samples t-test with a two-tailed design, an effect size of 0.5, an alpha level of 0.05, and a power of 80%, the minimum required sample size was calculated as 128 participants per group. A p-value of less than 0.05 was considered statistically significant.

Results

The demographic characteristics of the study and control groups are presented in Table 1. A total of 546 pregnant women were included in the study, of whom 182 constituted the study group and 364 constituted the control group. Maternal age and height were similar between the two groups (p>0.05). Gravida, parity, gestational age at delivery, weight, and body mass index differed significantly between the groups (p<0.05). The study group comprised both preterm and term cases, as all fetal distress cases admitted during the study period were consecutively enrolled regardless of gestational age (Table 1).

Table 1. Demographic data of groups

Variables	Study Groups n=182	Control Groups n=364	p Values
Maternal age (years)*	23.7 ± 3.5	23.6± 3.9	0.835
Gravida**	1 (0-7)	1 (1-4)	<0.001
Parity**	0 (0-0)	0 (0-4)	<0.001
Gestation at delivery (weeks)**	37 (27-41)	39 (36-40)	<0.001
Height (cm)*	161.7 ± 5.3	161.6 ± 5.9	0.850
Weight (kg)*	77.5 ± 13.1	74.5± 12.4	0.011
BMI (kg/m^2)*	29.6 ± 4.8	28.5 ± 4.4	0.008

*Independent samples t-test; **Mann-Whitney U test; BMI: body mass index. Bold values indicate statistical significance (p<0.05)

Hematologic variables of the study and control groups are presented in Table 2. Hct, RDW, MCV, platelet count, WBC, neutrophil count, and lymphocyte count were similar between the two groups (p>0.05). Hb, PDW, MPV, NLR, and PLR differed significantly between the groups (p<0.05). PDW values were significantly lower in the study group compared to the control group (16.3 ± 1.7 versus 17.8 ± 0.8, p<0.001). Hb values were

significantly lower in the study group than in the control group (11.9 ± 1.5 versus 12.3 ± 1.4, p<0.001). MPV, NLR, and PLR values were significantly higher in the study group than in the control group (p<0.05) (Table 2).

Table 2. Hematologic variables between groups

Variables	Study Groups (n=182)	Control Groups (n=364)	p value
Hb (g/dl)	11.9 ± 1.5	12.3 ± 1.4	<0.001
Hct (%)	36.1 ± 4.1	36.6 ± 4.2	0.147
RDW (%)	15.0 ± 2.2	14.7 ± 2.3	0.187
MCV (fL)*	86.7 ± 7.2	87.3 ± 6.8	0.325
Platelet count (×10³ mm⁻³)	237.9 ± 69.5	227.7 ± 72.4	0.113
PDW (%)	16.3 ± 1.7	17.8 ± 0.8	<0.001
MPV (fL)	10.3 ± 1.3	9.2 ± 1.5	<0.001
WBC(×10³ mm⁻³)	11.1 ± 3.9	10.7 ± 4.1	0.214
Neutrophil count (×10³ mm⁻³)	8.3 ± 3.4	7.7± 3.9	0.112
Lymphocyte count (×10³ mm⁻³)	1.91± 0.7	1.99 ± 0.6	0.169
NLR	5.1 ± 3.5	4.2 ± 2.7	<0.001
PLR	140.4 ± 79.3	120.3 ± 41.7	<0.001

Independent samples t-test. Hb: Hemoglobin; Hct: Hematocrit; RDW: Red cell distribution width; MCV: Mean corpuscular volume; PDW: Platelet distribution width; MPV: Mean platelet volume; WBC: White blood cell count; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio. Bold values indicate statistical significance (p<0.05)

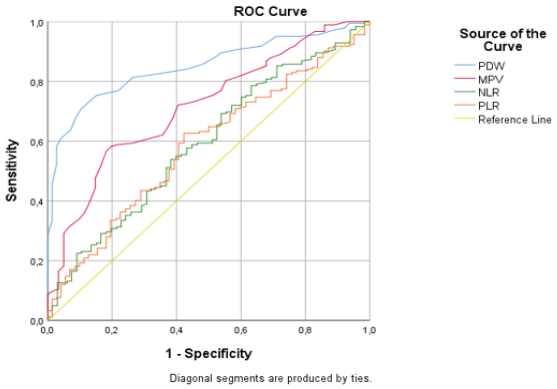

Figure 1. Receiver operating characteristic (ROC) curves of PDW, MPV, NLR, and PLR for predicting absent fetal heart rate variability with recurrent late decelerations. **PDW:** Platelet distribution width; MPV: Mean platelet volume; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; AUC: Area under the curve

The ROC curve analyses of hematologic markers for predicting absent fetal heart rate variability with recurrent late decelerations are presented in Table 3 and Figure 1. PDW demonstrated the highest discriminative performance among all markers evaluated, with an AUC of 0.850 (95% CI: 0.812-0.889, p<0.001). At the optimal cut-off value of ≤17.05%, PDW yielded a sensitivity of 70.9% and a specificity of 89.6%, as determined by the Youden index. MPV demonstrated acceptable discriminative ability, with an AUC of 0.716 (95% CI: 0.670-0.762, p<0.001). At the optimal cut-off value of ≥10.25 fL, MPV yielded a sensitivity of 56.6% and a specificity of 81.9%. NLR demonstrated limited discriminative ability, with an AUC of 0.593 (95% CI: 0.542-0.643, p<0.001). At the optimal cut-off value of ≥3.024, NLR yielded a sensitivity of 79.1% and a specificity of 35.2%. PLR demonstrated the lowest discriminative performance among the markers evaluated, with an AUC of 0.585 (95% CI: 0.533-0.636, p<0.001). At the optimal cut-off value of ≥115.06, PLR yielded a sensitivity of 59.3% and a specificity of 59.3%.

Table 3. ROC Curve analysis results

	Value	Sensitivity %	Specificity %	AUC (95% CI)	p value
PDW (%)	≤17.05%	70.9	89.6	0.850 (0.812-0.889)	<0.001
MPV (fL)	≥10.250fL	56.6	81.9	0.716 (0.670-0.762)	<0.001
NLR	≥3.024	79.1	35.2	0.593 (0.542-0.643)	<0.001
PLR	≥115.06	59.3	59.3	0.585 (0.533-0.636)	<0.001

AUC: Area under the curve; CI: Confidence interval; PDW: Platelet distribution width; MPV: Mean platelet volume; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio. Bold values indicate statistical significance (p<0.05).

The univariate and multivariate logistic regression analyses are presented in Table 4. In univariate analysis, gestational age, gravida, BMI, Hb, PDW, MPV, NLR, and PLR were significantly associated with absent fetal heart rate variability accompanied by recurrent late decelerations (p<0.05).

Table 4. Univariate and multivariate logistic regression analysis

	Univariate		Multivariate	
Variables	**OR (95% CI)**	**p value**	**OR (95% CI)**	**p value**
Covariates				
Gestational age (weeks)	0.59 (0.53-0.68)	<0.001	0.61 (0.51-0.72)	<0.001
Gravida	2.81 (2.16-3.64)	<0.001	2.19 (1.49-3.20)	<0.001
BMI	1.06 (1.02-1.09)	0.007	1.07 (1.00-1.15)	0.048
Hematologic Markers				
Hb (g/dl)	0.81 (0.72-0.92)	<0.001	0.90 (0.73-1.12)	0.341
PDW (%)	0.15 (0.11-0.21)	<0.001	0.15 (0.09-0.23)	<0.001
MPV (fL)	1.96 (1.66-2.32)	<0.001	2.19 (1.70-2.81)	<0.001
NLR	1.10 (1.032-1.162)	0.003	1.13 (1.02-1.26)	0.021
PLR	1.01 (1.003-1.010)	<0.001	0.99 (0.99-1.01)	0.185

Logistic regression analysis. OR: Odds ratio; CI: Confidence interval; BMI: Body mass index; Hb: Hemoglobin; PDW: Platelet distribution width; MPV: Mean platelet volume; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio. Bold values indicate statistical significance (p<0.05)

In multivariate analysis, after adjusting for gestational age, gravida, and BMI, PDW (OR: 0.15, 95% CI: 0.09-0.23, p<0.001), MPV (OR: 2.19, 95% CI: 1.70-2.81, p<0.001), and NLR (OR: 1.13, 95% CI: 1.02-1.26, p=0.021) were identified as independent predictors of absent fetal heart rate variability with recurrent late decelerations. Gestational age (OR: 0.61, 95% CI: 0.51-0.72, p<0.001), gravida (OR: 2.19, 95% CI: 1.49-3.20, p<0.001), and BMI (OR: 1.07, 95% CI: 1.00-1.15, p=0.048) were retained as significant covariates in the multivariate model. Hb and PLR failed to retain statistical significance in the multivariate model (p>0.05).

Discussion

In this retrospective case-control study, we investigated the predictive utility of hematologic markers for absent fetal heart rate variability with recurrent late decelerations. The main finding of the present study was that PDW and MPV were identified as independent predictors of absent FHR variability with recurrent late decelerations in multivariate analysis. Additionally, NLR was found to be an

independent predictor, albeit with a modest effect size. To the best of our knowledge, this is the first study to evaluate the predictive role of hematologic markers specifically in the context of absent FHR variability with recurrent late decelerations. Fetal distress and non-reassuring NST findings represent the most common indications for emergency cesarean delivery (10,11). Late decelerations are considered a reflex fetal response to transient hypoxemia occurring during uterine contractions (1). Spiral arteries, which are essential for fetal nutrition, course between myometrial muscle fibers, and when intrauterine pressure exceeds 35 mmHg, blood flow in these vessels is temporarily interrupted (2,14). When uterine contractions are prolonged, excessively strong, or abnormally frequent, intervillous blood flow may decrease substantially, leading to a reduction in fetal PO2 levels. Under normal circumstances, the fetus tolerates this transient hypoxemia through several compensatory mechanisms, including the high oxygen affinity of fetal hemoglobin, elevated fetal hemoglobin concentrations, and adequate intervillous reserve capacity (3). When these compensatory mechanisms are overwhelmed, fetal hypoxia ensues, resulting in myocardial depression and the emergence of late decelerations (1). Late decelerations associated with severe hypoxia, metabolic acidemia, and myocardial depression have been shown to significantly increase the risk of adverse neonatal outcomes (15,16)

In the present study, PDW values were significantly lower in the study group compared to the control

group, and PDW demonstrated the highest discriminative performance among all markers evaluated, with an AUC of 0.850. At the optimal cut-off value of ≤17.05%, PDW yielded a sensitivity of 70.9% and a specificity of 89.6%. Furthermore, PDW was identified as the strongest independent predictor of absent FHR variability with recurrent late decelerations in multivariate analysis (OR: 0.15, 95% CI: 0.09-0.23, p<0.001).

PDW reflects the heterogeneity of platelet size and serves as an indicator of platelet activation (17). Placental hypoxia, which underlies the pathophysiology of fetal distress, is known to trigger endothelial injury and subsequent platelet activation (18).Activated platelets undergo morphological changes and exhibit a more uniform size distribution, which paradoxically results in a decrease in PDW(17). This mechanism is analogous to that proposed in preeclampsia, where placental hypoxia and endothelial dysfunction drive systemic inflammatory and thrombotic responses (18). A significant relationship between PDW and threatened preterm labor has previously been reported by Artunc Ulkumen et al., who suggested that platelet activation may serve as a marker of underlying placental dysfunction (19). Similarly, Dundar et al. demonstrated elevated PDW values in patients with recurrent pregnancy loss, further supporting the role of platelet activation in adverse pregnancy outcomes (20).

MPV, which reflects the mean size of circulating platelets, is a well-established marker of platelet activation and function (21). In the present study, MPV values were significantly higher in the study group compared to the control group, and MPV demonstrated acceptable discriminative ability with an AUC of 0.716. At the optimal cut-off value of ≥10.25 fL, MPV yielded a sensitivity of 56.6% and a specificity of 81.9%. In multivariate analysis, MPV was identified as an independent predictor of absent FHR variability with recurrent late decelerations (OR: 2.19, 95% CI: 1.70-2.81, p<0.001).

The elevation of MPV observed in the study group may be attributed to the release of larger, more metabolically active platelets from the bone marrow in response to increased thrombopoietin levels triggered by hypoxia-induced endothelial stress (18,21). Larger platelets are known to be more reactive and prothrombotic, potentially contributing to the microvascular changes observed in placental insufficiency (21–23). The concurrent decrease in PDW alongside the increase in MPV observed in our study suggests a pattern consistent with platelet activation, whereby platelets become larger yet more uniform in size. Çintesun et al. previously demonstrated significantly elevated MPV values in patients with preeclampsia compared to healthy controls, proposing that platelet activation plays a central role in the pathophysiology of placental dysfunction (4,5). The findings of the present study extend this observation to the context of intrapartum fetal distress.

NLR, a marker of systemic inflammatory response, was found to be significantly higher in the study group compared to the control gr oup. In multivariate analysis, NLR was identified as an independent predictor of absent FHR variability with recurrent late decelerations (OR: 1.13, 95% CI: 1.02-1.26, p=0.021). At the optimal cut-off value of ≥3.024, NLR yielded a sensitivity of 79.1% and a specificity of 35.2%, with an AUC of 0.593, indicating limited discriminative ability despite its statistical significance.

The elevation of NLR in the study group may reflect the systemic inflammatory response triggered by placental hypoxia and endothelial injury. Placental ischemia is known to activate innate immune pathways, leading to neutrophil recruitment and relative lymphocyte suppression, thereby increasing the NLR (18).This inflammatory mechanism is well established in the context of preeclampsia, where placental hypoperfusion drives a systemic inflammatory cascade (18). Although NLR has been investigated in various obstetric conditions, its utility as a standalone predictor of intrapartum fetal distress appears limited, as evidenced by the low specificity observed in the present study. Christoforaki et al. similarly reported that first-trimester NLR was not a significant predictor of adverse pregnancy outcomes, with the exception of an association with increased miscarriage rates (6). Yücel et al. found that NLR was similar between preeclamptic and healthy pregnant women, suggesting that the inflammatory response captured by NLR may not be specific to placental dysfunction

(24). The modest OR value observed in our study further supports the notion that NLR, while statistically significant, has limited clinical utility as an independent predictor in this context.

PLR was significantly higher in the study group in univariate analysis; however, it failed to retain statistical significance in the multivariate model (OR: 0.99, 95% CI: 0.99-1.01, p=0.185). The loss of significance of PLR in multivariate analysis is likely attributable to its collinearity with NLR, as both markers share the lymphocyte count as a common denominator. The AUC of PLR was 0.585 (95% CI: 0.533-0.636), reflecting poor discriminative ability. These findings are consistent with those of Yücel et al., who demonstrated elevated PLR values in preeclamptic patients but noted considerable overlap between groups (24). The limited independent predictive value of PLR observed in the present study suggests that NLR may be a more informative inflammatory marker in this clinical context.

Similarly, Hb was significantly lower in the study group in univariate analysis (OR: 0.81, 95% CI: 0.72-0.92, p<0.001); however, it did not retain statistical significance in the multivariate model (OR: 0.90, 95% CI: 0.73-1.12, p=0.341). This finding suggests that the lower Hb levels observed in the study group may be confounded by gestational age differences between the groups, given that preterm pregnancies were included in the study group. The loss of significance of Hb in the adjusted model indicates that it does not serve as an independent predictor of absent FHR variability with recurrent late decelerations when other clinical and hematologic variables are accounted for.

The clinical implications of the present study are noteworthy. Cesarean section is one of the most frequently performed surgical procedures worldwide, and emergency cesarean delivery is associated with a significantly higher risk of maternal complications compared to elective procedures, including endometritis, wound complications, and venous thromboembolism (10,11,25). The ability to identify patients at risk for absent FHR variability with recurrent late decelerations prior to the onset of labor could allow for more judicious planning of delivery, potentially reducing the rate of emergency cesarean delivery and its associated morbidity.

PDW and MPV, as simple and inexpensive components of the routine complete blood count, are readily available in clinical practice without additional cost or complexity. The identification of PDW ≤17.05% and MPV ≥10.25 fL as independent predictors of intrapartum fetal distress may facilitate the antenatal stratification of patients at higher risk, enabling closer fetal surveillance and more timely clinical intervention. Furthermore, the adverse neonatal outcomes associated with late decelerations due to severe hypoxia and metabolic acidemia underscore the importance of early prediction and intervention in this population (15,16).

The present study has several strengths. To the best of our knowledge, this is the first study to systematically evaluate the predictive utility of hematologic markers for absent FHR variability with recurrent late decelerations. The relatively large sample size of 546 participants, the consecutive enrollment of all eligible cases during the study period, and the use of multivariate logistic regression to control for potential confounding variables represent methodological strengths of this study.

However, several limitations must be acknowledged. First, the retrospective design of the study introduces the possibility of selection bias and limits the ability to establish causal relationships. Second, the inclusion of both preterm and term cases in the study group may have introduced heterogeneity, although this approach was intentional to reflect real clinical practice. Third, fetal blood gas results were not available for the study population, which precluded direct confirmation of fetal acidemia in the study group. Fourth, the single-center design of the study may limit the generalizability of the findings to other clinical settings. Future prospective multicenter studies with larger sample sizes incorporating additional biomarkers and ultrasonographic parameters are warranted to validate and expand upon these findings.

Conclusion

In conclusion, the present study demonstrated that PDW and MPV are independent predictors of absent fetal heart rate variability with recurrent late decelerations, with PDW exhibiting the highest discriminative performance (AUC: 0.850). NLR was

also identified as an independent predictor; however, its discriminative ability was limited (AUC: 0.593). These hematologic markers are readily available, inexpensive, and easily applicable in clinical practice as routine components of the complete blood count. Patients with PDW ≤17.05% and MPV ≥10.25 fL may represent a high-risk group warranting closer intrapartum fetal surveillance. Early identification of such patients may contribute to more timely clinical decision-making, potentially reducing the rate of emergency cesarean delivery and its associated maternal and neonatal complications. Prospective multicenter studies incorporating larger sample sizes and additional biomarkers are needed to validate these findings and to explore the integration of hematologic markers into comprehensive clinical prediction models for intrapartum fetal distress.

Ethics statement: This study was approved by the Institutional Ethics Committee of Selcuk University Faculty of Medicine (Approval number: 2021/270). The study was conducted in accordance with the Declaration of Helsinki.

Conflict of Interest Statement: The authors declare no conflicts of interest.

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Author contribution statement: EC: Conceptualization, study design, data collection, statistical analysis, writing original draft, review and editing. NDS: Data collection, writing original draft, review and editing. ET: Data collection, review and editing. CC: Conceptualization, supervision, review and editing.

Data availability statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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