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Can platelet activation markers predict preeclampsia and/or its severity?

¿Pueden los marcadores de activación plaquetaria predecir la preeclampsia y/o su gravedad?

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Abstract

Objective: This study aimed to evaluate the value of platelet activation markers in predicting preeclampsia and its severity. Preeclampsia is a serious pregnancy complication that affects 3-5% of pregnancies and can lead to significant morbidity and mortality for both the mother and the fetus. **Methods:** The study included 99 patients diagnosed with preeclampsia and 60 healthy pregnant women as a control group. Platelet activation markers such as mean platelet volume (MPV), platelet distribution width (PDW), platelet count, and plateletcrit were evaluated along with other clinical parameters. **Results**: The results of the study showed that platelet activation markers, particularly PDW and MPV, are valuable in the diagnosis and follow-up of preeclampsia. However, they are not sufficient to predict the severity of the disease. **Conclusion:** The study suggests that platelet activation markers could aid in predicting, diagnosing, and managing preeclampsia. However, further research is needed to determine the role of these markers in predicting the severity of the disease. The findings of this study could contribute to the development of more effective strategies for the prevention and management of preeclampsia, which could ultimately improve maternal and fetal outcomes.

Keywords: Mean platelet volume. Plateletcrit. Platelet activation markers. Preeclampsia.

Resumen

Objetivo: El estudio tuvo como objetivo determinar el valor de los marcadores de activación plaquetaria en la predicción de la preeclampsia y su gravedad. **Método:** Se incluyeron 99 pacientes diagnosticadas con preeclampsia, incluyendo 36 casos graves, y un grupo control de 60 mujeres embarazadas sanas. Se evaluaron diversas variables, como el volumen plaquetario medio, el recuento de plaquetas, el hematocrito plaquetario y la amplitud de distribución plaquetaria. **Resultados:** Los resultados mostraron que el volumen plaquetario medio y la amplitud de distribución plaquetaria son parámetros valiosos en el diagnóstico y seguimiento de la preeclampsia, aunque no son suficientes para predecir su gravedad. El análisis estadístico reveló que la edad, el volumen plaquetario medio, la amplitud de distribución plaquetaria, la semana de gestación y los puntajes de Apgar al primer y quinto minuto fueron significativamente diferentes en el grupo de preeclampsia en comparación con el grupo control. **Conclusiones:** En conclusión, estos resultados sugieren que los marcadores de activación plaquetaria pueden ser útiles para el diagnóstico y seguimiento de la preeclampsia, y que el volumen plaquetario medio y la amplitud de distribución plaquetaria, por ser parámetros económicos y accesibles, podrían ayudar a predecir, diagnosticar y manejar esta complicación durante el embarazo.

Palabras clave: Volumen plaquetario medio. Critocitos plaquetarios. Marcadores de activación plaquetaria. Preeclampsia.

Introduction

Preeclampsia, the etiology and pathogenesis of which have not been fully elucidated, is a serious complication that is seen in 3-5% of all pregnancies and threatens the life of both the fetus and the mother[1]. One view locates its pathogenesis in placental ischemia due to abnormalities in the location of the placenta, remodeling of the spiral artery, and invasion of the extravillous trophoblast. As a result of this ischemia, vasoactive substances are released into the maternal circulation, which may cause maternal endothelial activation and endothelial dysfunction[2].

Microangiopathy in preeclampsia affects all cells circulating in the vessel, such as leukocytes, neutrophils, lymphocytes, and platelets. The hematological abnormality observed in preeclampsia depends on platelet consumption and the activation of the coagulation system. An increase in platelet function has also been reported in pregnant women with preeclampsia[3]. A damaged endothelium due to defective placental trophoblastic invasion leads to the activation of platelets and inflammation[4].

The mean platelet volume (MPV) increases when platelets are activated. Large platelets are stickier than small ones and likelier to aggregate. Platelet activation markers include MPV; platelet distribution width (PDW), which corresponds to the size distribution of platelets; plateletcrit (PCT), which corresponds to the volume of platelets in 100 mL of total blood; and the platelet large cell ratio, which reflects the percentage of platelets > 12 fL indirectly by calculating various indices, including PDW, which measures platelet size distribution. MPV, PCT, and PDW are potential and easily measurable *in vivo* markers of platelet activation[5].

Platelet activation markers may be more sensitive than platelet counts, which may change during normal pregnancy and as a result of preeclampsia. Physiologically, MPV decreases from the 20th week of pregnancy to the 31st week and increases after the 38th week. Increased platelet activity may contribute to the formation of microthrombi in the placenta and exacerbate the vascular dysfunction seen in preeclampsia. Therefore, platelet activation markers may have the potential to aid in the early prediction of preeclampsia and prognosis[6].

Platelet activation markers are routine parameters in complete blood counts. Measurement of platelet activation markers is an easily accessible, reproducible, simple, and inexpensive test. In this study, we investigated the differences between platelet activation markers in healthy pregnant women and pregnant women with preeclampsia and these markers' prognostic effects on the group with preeclampsia.

Methods

This was a retrospective, descriptive, and cross-sectional study. A total of 99 patients diagnosed with preeclampsia were included in the study, 36 of whom were diagnosed with severe preeclampsia. The control group consisted of 60 patients. Patients diagnosed with preeclampsia between May 2020 and November 2022 were retrospectively included in the study. Relevant demographic data, such as maternal age, parity, complete blood count results, and gestational age at birth, were recorded from hospital records and hospital automation systems for analysis.

We compared platelet counts and platelet indices first MPV, and then PDW and PCT values in patients diagnosed with preeclampsia and the control group. We then investigated the differences in platelet activation markers between patients with severe and non-severe preeclampsia.

Venous blood samples of the patients were collected in tubes containing ethylenediaminetetraacetic acid (EDTA) in the first trimester of pregnancy (12-14 weeks) during routine controls, and complete blood counts of all patients included in the study were performed in our hospital laboratory using the same automatic analyzer. The blood sample was taken and studied at the first moment of diagnosis. Blood samples of 2 mL were collected in vacuum tubes (purple cap) containing 2.0 mg/mL EDTA and stored at 37°C for platelet analysis. The samples were measured using an LH 755 automated quantitative hematology analyzer (BECKMAN COULTER Inc., USA). All tubes were mixed by being inverted 5-10 times immediately after blood collection and analyzed within 1 h.

Urine protein levels were measured with a measuring stick at the time of admission. Complete urinalyses were studied using a FUS-200/H-800 Fully Automatic Urine Analysis System. Proteinuria was measured turbidimetrically in spot and 24-h urine samples. Neonatal outcomes at the 1st and 5th min of life were recorded. Apgar scores and delivery patterns were noted.

The diagnoses of preeclampsia and its severity were made according to the criteria in The American College of Obstetricians and Gynecologists Practice Bulletin No. 222[7].

Preeclampsia was diagnosed if a patient previously had normal blood pressure, a systolic blood pressure of ≥ 140 mmHg, and/or a diastolic blood pressure of 90 mmHg at least 4 h apart after the 20th gestational week in addition to one of the following criteria: proteinuria (with a dipstick reading ≥ 2+ and protein/creatinine ratio ≥ 0.3 or ≥ 0.3 g in a 24-h urine sample), thrombocytopenia (with a platelet count ≤ 100,000/mL), a renal failure serum creatinine concentration > 1.1 mg/dL (97.2 μmol/L), a blood concentration of liver transaminases at twice the normal concentration, pulmonary edema, visual symptoms (blurred vision, flashing lights or sparks, or scotoma), or the new onset of a persistent headache unexplained by alternative diagnoses and unresponsive to routine doses of analgesics.

A patient was diagnosed with severe preeclampsia if they had a systolic blood pressure ≥ 160 mmHg and/or a diastolic blood pressure ≥ 110 mmHg in two measurements at rest at least 4 h apart, symptoms of central nervous system dysfunction (new-onset cerebral or visual impairment), hepatic abnormality (unresponsive to medication), severe and persistent right upper quadrant or epigastric pain, a serum transaminase concentration ≥ 2 times the upper limit of the normal range or both, thrombocytopenia (< 100,000 platelets/microL), a renal abnormality (serum creatinine > 1.1 mg/dL), or pulmonary edema. If there was more than one, it was diagnosed as severe preeclampsia.

All of the control group members were healthy women with single pregnancies, no history of systemic disease, and no fetal or chromosomal abnormalities. The blood pressure of the women in this group was below 140/90 mmHg, and none had proteinuria. They all had healthy live births at term. This control group was randomly selected from patients who were followed up and treated in our hospital, were in their third trimesters of pregnancy, and did not have preeclampsia or systemic disease.

To avoid any possible interaction with the incidence of preeclampsia or fetal growth restriction (FGR) and platelet changes caused by the use of drugs that may affect platelet activity, such as aspirin, we excluded patients using these drugs. Patients with previous kidney disease, insulin-dependent diabetes, asthma requiring steroid therapy, chronic hepatitis (with or without liver dysfunction), chronic kidney disease, a history of severe trauma, a history of anticoagulant drug use, a history of oral contraceptive use, a history of smoking, immune thrombocytopenic purpura, hemolysis, elevated liver enzymes, and low platelets syndrome, gestational thrombocytopenia, or any hematological disease were excluded. The exclusion criteria were strictly maintained, and we attempted to increase the working power.
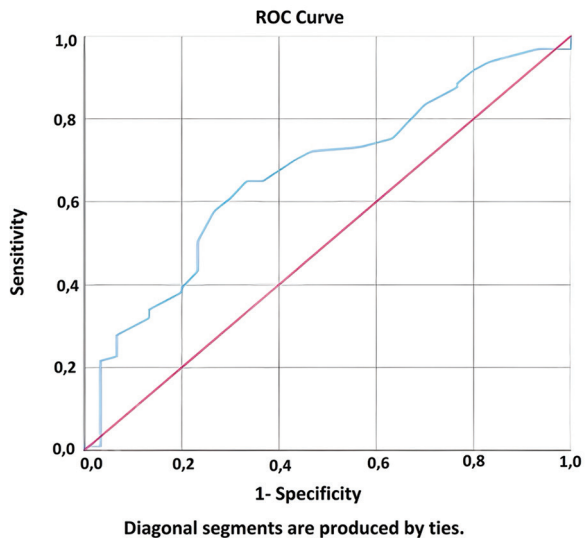
The study was carried out in accordance with the ethical regulations and permission of the hospital's Ethics Committee (2022-42) and conducted in accordance with the Declaration of Helsinki.

The Number Cruncher Statistical System 2007 (Kaysville, Utah, USA) program was used for the statistical analysis. When evaluating the study data, the descriptive statistical methods (mean, standard deviation, median, frequency, ratio, minimum, and maximum) and distribution of the data were determined using the Shapiro–Wilk test. The Mann–Whitney U test was used to compare two groups of quantitative data. Chi-square analysis was used to determine the relationships between the qualitative data. Receiver operating characteristic (ROC) analysis was used to determine the predictive value of the variables. Spearman's correlation test was used to determine the relationships between the quantitative data. Multiple logistic regression analysis was used to determine the factors affecting the dependent variable. Significance was evaluated at the $p < 0.01$ and $p < 0.05$ levels.

Results

A total of 99 pregnant women diagnosed with preeclampsia were included in the study. Of these, 63.6% (n = 63) were not severe preeclampsia cases, while 36.4% (n = 36) were severe. In the preeclampsia group, 54.5% (n = 54) of the patients received magnesium treatment, while 45.5% (n = 45) did not. The rates of protein in the complete urinalyses of the preeclamptic pregnant women were as follows: 24.2% (n = 24) were negative, 29.3% (n = 29) were 1+, 7.1% (n = 7) were 2+, and 39.4% (n = 39) were 3+. Of the patients, 51.5% (n = 51) had primary cesarean sections, 28.3% (n = 28) had previous cesarean sections, 11.1% (n = 11) had induced labor, and 9.1% (n = 9) gave birth spontaneously. While 45.5% (n = 45) of the preeclamptic patients' babies were female, 54.5% (n = 54) were male, and 45.5% (n = 45) had FGR, while 54.5% (n = 54) did not.

The age, MPV, platelet count, PCT, PDW, red cell distribution width coefficient of variation (RDW-CV), red cell distribution width standard deviation (RDW-SD), hemoglobin, week of birth, gravida, parity, abortion number, and Apgar score 1st- and 5th-min values

Figure 1. *Receiver operating characteristic curve of mean platelet volume parameter.*

Table 1. Comparison of measurements by group

Parameters	n	Mean ± SD	Min-Max (Median)	p-value
Age (year)				
Preeclampsia	99	30.99 ± 6.06	18-45 (31)	0.001*
Control	60	26.77 ± 5.15	20-37 (26)	
MPV (fL)				
Preeclampsia	99	11.3 ± 1.1	9-14 (11.2)	0.006*
Control	60	10.71 ± 0.94	9.6-13.7 (10.5)	
PLT (10³/μL)				
Preeclampsia	99	256.13 ± 70.06	139-501 (245)	0.640
Control	60	247.43 ± 63.93	138-410 (234.5)	
PCT (%)				
Preeclampsia	99	0.29 ± 0.07	0.16-0.53 (0.28)	0.124
Control	60	0.26 ± 0.06	0.16-0.43 (0.26)	
PDW (%)				
Preeclampsia	99	13.91 ± 2.73	9.1-21.9 (13.6)	0.048*
Control	60	12.94 ± 2.41	10-19.4 (11.95)	
RDW-CV (%)				
Preeclampsia	99	14.22 ± 1.88	11.9-22.7 (13.7)	0.759
Control	60	14.77 ± 2.92	12.1-26 (13.9)	
RDW-SD (fL)				
Preeclampsia	99	43.16 ± 4.85	36.2-63.7 (42.5)	0.113
Control	60	44.73 ± 5.94	37.6-67.5 (44.3)	
Hemoglobin (g/dL)				
Preeclampsia	99	12.01 ± 1.44	7.1-15.4 (12)	0.290
Control	60	11.63 ± 1.76	6.9-14.7 (11.85)	
Birth week (day)				
Preeclampsia	99	188.57 ± 89.03	0-275 (222)	0.001*
Control	60	275.23 ± 9.79	254-294 (274.5)	
Gravida (number)				
Preeclampsia	99	2.35 ± 1.74	1-9 (2)	0.155
Control	60	2.67 ± 1.58	1-7 (2)	
Parity (number)				
Preeclampsia	99	0.89 ± 1.13	0-5 (0)	0.055
Control	60	1.33 ± 1.27	0-4 (1)	
Abortion (number)				
Preeclampsia	99	0.48 ± 1.06	0-6 (0)	0.646
Control	60	0.33 ± 0.71	0-3 (0)	
Apgar score 1st min				
Preeclampsia	99	5.99 ± 1.89	0-9 (6)	0.001*
Control	60	7.93 ± 0.87	5-9 (8)	
Apgar score 5th min				
Preeclampsia	99	7.78 ± 1.49	0-10 (8)	0.001*
Control	60	9.17 ± 0.59	8-10 (9)	

*Mann Whitney U Test p < 0.05.
fL: femtoliter; MPV: mean platelet volume; PLT: platelet; PCT: plateletcrit, PDW: platelet distribution width; RDW-CV: red cell distribution width coefficient of variation; RDW-SD: red cell distribution width standard deviation.

of the patients and control group are given in table 1. The age, MPV, PDW, week of birth, and APGAR score 1st- and 5th-min values were found to be statistically significant in the preeclampsia group compared to the control group (p = 0.001; p < 0.05). PLT, PCT, RDW-CV, RDW-SD, hemoglobin, gravida, parity, and number of abortions did not show any statistically significant difference (p > 0.05).

As shown in table 2, when the patients with pre-eclampsia were divided into severe and non-severe groups, there were no statistically significant differences between MPV, PLT, PCT, PDW, RDW-CV, RDW SD, hemoglobin, or Apgar score 1st- and 5th-min values and the week of birth (p > 0.05). Moreover, there was no statistically significant relationship between the severity of preeclampsia and FGR (p > 0.05).

In this study, 64.8% (n = 35) of the patient group and 35.2% (n = 19) of the control group had female fetuses. There was no statistically significant relationship between the severity of preeclampsia and fetal gender (p > 0.05).

Upon applying Spearman's correlation analysis, no correlation was found between MPV, PLT, PCT, PDW, RDW-CV, RDW SD, hemoglobin, or APGAR 1st- and 5th-min scores and the week of birth (p > 0.05).

A statistically significant correlation in complete urinalysis protein was found between the groups (p = 0.001; p <0.01). In those with complete urinalysis protein 3+, the patients had higher values than the control group by a statistically significant amount (p = 0.001; p < 0.01).

Cutoff and AUC value of ROC analysis

When the cutoff point of the MPV value was taken as 10.85, the sensitivity was 98.7%. The specificity

Table 2. Comparison of measurements by preeclampsia severity

Parameter	n	Mean ± SD	Min-Max (Median)	p-value
MPV (fL)				
Non-severe	63	11.33 ± 1.09	9.4-13.6 (11.3)	0.922
Severe	36	11.32 ± 1.07	9-14 (11.25)	
PLT (10³/µL)				
Non-severe	63	256.48 ± 67.15	139-383 (245)	0.904
Severe	36	257.81 ± 71.91	164-501 (252)	
PCT (%)				
Non-severe	63	0.29 ± 0.07	0.16-0.45 (0.28)	0.968
Severe	36	0.29 ± 0.07	0.2-0.53 (0.28)	
PDW (%)				
Non-severe	63	13.87 ± 2.6	9.9-21.9 (13.8)	0.644
Severe	36	14.14 ± 2.83	9.1-21.5 (13.9)	
RDW-CV (%)				
Non-severe	63	14.56 ± 2.1	12.2-22.7 (14)	0.134
Severe	36	13.77 ± 1.1	11.9-17 (13.7)	
RDW-SD (fL)				
Non-severe	63	43.83 ± 5.35	36.8-63.7 (43.2)	0.174
Severe	36	42.15 ± 3.39	36.2-50 (41.75)	
Hemoglobin (g/dL)				
Non-severe	63	11.74 ± 1.45	7.1-14.1 (11.9)	0.059
Severe	36	12.39 ± 1.12	10.6-15.2 (12.4)	
Apgar score 1st min				
Non-severe	63	6.06 ± 1.51	1-9 (6)	0.727
Severe	36	5.83 ± 1.58	1-8 (6)	
Apagr score 5th min				
Non-severe	63	7.92 ± 1.2	3-10 (8)	0.342
Severe	36	7.83 ± 0.91	5-9 (8)	
Birth week				
Non-severe	63	33.07 ± 2.95	25.43-39.29 (33)	0.079
Severe	36	32.08 ± 2.13	25-35.57 (32.5)	

FL: femtoliter; MPV: mean platelet volume; PLT: platelet, PCT: plateletcrit, PDW: platelet distribution width; RDW-CV: red cell distribution width coefficient of variation; RDW-SD: red cell distribution width standard deviation.

was determined to be 98.5%, and a reliable cutoff point was determined (Fig. 1)

Table 3 shows, when multiple logistic regression analysis was performed to determine the effect of the independent variables on disease status, it was found to be statistically significant (X2 = 43,738; p < 0.001). There was a positive and weakly significant relationship between the independent variables and disease status (R = 0.29, p < 0.001). The independent variables in the model explain 8.5% of the total variance in disease status (p < 0.01).

When the regression coefficients were examined, the complete urinalysis protein-negative (β = −0.085, p < 0.001) variable was a negative effect on disease status, while age (β = 1.177, p < 0.001) and MPV (β = 2.021, p < 0.001) seemed to have positive and significant effects.

Discussion

The results of our study are similar to those in the literature. We found platelet activation markers to be valuable parameters in the diagnosis and follow-up of preeclampsia but insufficient for predicting the intake of preeclampsia. After the continuous consumption of platelets in the peripheral blood in preeclamptic pregnant women, a rapid platelet turnover occurs as a result of the continuous production in the bone. Järemo and colleagues explained the increase in platelet volume by the presence of vessels rather than platelet changes and attributed it to those that dispersed to the surrounding platelet densities[8]. Highly dispersed platelets have high volumes, which limits the assumption that platelet lines and subsequent granule release disrupt platelet distribution[9].

After the release of vasoactive amines, the prolongation and swelling of platelets occur, and new platelets are released into the blood. This leads to an increase in MPV and PDW. A single platelet count may be misleading in early prognosis. Therefore, platelet activation parameters should be used. Reddy and Rajendra Prasad argued that the spread of preeclampsia aggressively occurs on 10.95 fL cut-off travelers in the MPV they bring[10,11]. In our study, the cut-off point for MPV surveillance between the patient and control groups was 10.85, leading to a protective value of 98.7%; ownership was determined as 98.5%, the reliable cutoff point. Our study is consistent with the literature and suggests that MPV is valuable in the diagnosis of preeclampsia in pregnancy.

Yang et al. stated that the PDW value in women with preeclampsia is a valid measurement tool for predicting severe preeclampsia[12]. In our study, the PDW values of the patient group were also higher than those of the control group and were statistically significant.

PCT is calculated by multiplying the platelet count by the MPV and dividing the result by 10,000. Thalor et al. reported in their study that PCT, a marker of platelet activation, showed a slight decrease in patients with preeclampsia, but not at a statistically significant level[13]. In our study, there was no statistically significant relationship between PCT values and preeclampsia. We believe that this may be because the platelet count tends to approach normal in preeclampsia patients, as stated in Thalor et al., and because PCT is calculated using platelet counts[13].

Çintesun et al. looked at the difference between PCT values in preeclamptic pregnant women with different degrees of preeclampsia and could not obtain a significant result[14]. In our study, we did not find a statistically significant difference between PCT values in the severe and

Table 3. Logistic regression analysis findings for ınterpretation of disease status with ındependent variables

Model	Variables	Univariable					Multivariable				
		B	SD	Wald	Exp (B)	p	B	SD	Wald	Exp (B)	p
1	Age (year)	0.129	0.040	10.148	1.137	0.001*	0.163	0.052	9.659	1.177	0.001*
	MPV (fL)	0.568	2.454	6.347	1.765	0.001*	0.703	0.275	6.528	2.021	0.001*
	Complete Urinalysis Protein-Negative	−2.264	0.478	22.400	0.104	0.001*	−2.462	0.552	19.867	−0.085	0.001*

*p < 0.05.

mild preeclampsia groups. Although these differences are well known and defined, one factor that may be relevant here is the activation of the bone marrow with an unknown stimulus due to the individuality of the inflammatory and coagulant responses of each pregnant woman.

In their meta-analysis, Bellos et al. suggested that MPV should be evaluated together with conventional markers of preeclampsia and included in combined models that would provide optimum efficiency in the prediction of the disease[15,16]. Based on our results, we think that platelet activation markers, especially MPV and PDW, are valuable. However, we believe that it is necessary to be aware that they are systemic markers.

The MPV measurement method should be standardized to eliminate inconsistent results. It is known that MPV increases with time when the sample is exposed to EDTA[9]. We tried to achieve standardization by sending our samples to the laboratory to be examined as soon as possible. In addition, we made sure to take all measurements on the same device and tried to increase the working power by eliminating possible differences.

The relationship between RDW and preeclampsia has been investigated in many studies. Yücel et al. found that the RDW was significantly higher in severely preeclamptic pregnant women compared to their control group. RDW indicates a change in erythrocyte volume called anisocytosis. High RDW levels are believed to reflect increased inflammation, but this mechanism has not been elucidated[17]. The RDW-CV is found by dividing the histogram width of erythrocytes in 1 SD by the MCV. The RDW-SD is the difference in volume between the largest and smallest erythrocytes at the level of 20% of the erythrocyte population in the erythrocyte histogram. RDW-SD and RDW-CV are parameters that can generally be used to evaluate RDW[18]. We did not find a statistically significant difference in RDW-SD, RDW-CV, or hemoglobin between preeclampsia patients and the control group. Likewise, when we divided the preeclampsia group into severe and non-severe cases, there was no statistical difference. Since the data on

the patients' folate and iron levels were not routinely checked, we attributed the different levels to the activation of the patients' red blood cell series.

MPV is a measurement of the average size of platelets. Under conditions of increased platelet turnover, an increase in MPV produces larger and more reactive platelets, possibly affecting megakaryocyte ploidy. Because the MPV increase reflects increased platelet turnover, it precedes the clinical onset of disease symptoms. An identified increase in the MPV of these platelets in the pathogeneses of hypertensive disorders of pregnancy should be carefully monitored to aid in early diagnosis so that appropriate management can be enacted and a thrombotic event leading to maternal and neonatal morbidity or mortality can be prevented[19,20]. We administered magnesium treatment to 54.5% (n = 54) of the patients to avoid complications.

Several studies have investigated the relationship between preeclampsia and neonatal respiratuar distress syndrom in preterm infants, but definitive conclusions have not been reached. The mechanism underlying the association between preeclampsia and neonatal respiratory disorders is unclear. It has been suggested that preeclampsia causes a degenerative change in placental villi and a continuous spasm of maternal whole-body arterioles. In this case, it may cause long-term chronic hypoxia in the fetus, leading to neonatal respiratory disorders. As a second mechanism, it may cause pulmonary hypertension by causing neonatal pulmonary vasculature and systemic vascular dysfunction[21,22]. We found that Apgar scores were significantly lower at the 1st and 5th min in the preeclampsia group than in the control group. However, there was no statistically significant difference between the severe and non-severe groups. When we looked at the correlations of these Apgar scores with platelet activation markers, we could once again find no statistical significance. The increased risk of preeclampsia is caused by incomplete lung maturation due to preterm labor, which may also exacerbate the severity of the preeclampsia.

Limitations

Platelet indices have different normal ranges, as they are determined by different laboratory machines used for analysis. Therefore, the data need to be adjusted to allow for these differences in the normal ranges determined by different machines. Further multicenter studies with larger numbers of patients are required to resolve the contradictions in the literature and achieve definitive results.

Conclusion

Preeclampsia can lead to serious complications if not properly diagnosed. It is a multi-organ disease; therefore, it is difficult to establish severity markers for its development. However, the development of these markers may assist clinicians in determining the timing of delivery for women with preeclampsia. PDW and MPV, which are platelet activation markers that are economical and easily available, can be used in the prediction, early diagnosis, and management of the disease. It would be ideal to create functional systemic indices to predict the onset and severity of PE, which is a systemic disease.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

Confidentiality of data. The authors declare that they have followed the protocols of their work center on the publication of patient data.

Right to privacy and informed consent. The authors have obtained approval from the Ethics Committee for analysis and publication of routinely acquired clinical data and informed consent was not required for this retrospective observational study.

Use of artificial intelligence for generating text. The authors declare that they have not used any type of generative artificial intelligence for the writing of this manuscript nor for the creation of images, graphics, tables, or their corresponding captions.

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