

Research Article

# Assessing the transaminase complex-platelet ratio (TACPR) and the platelet-albumin ratio (PAR) as composite biomarkers in severe preeclampsia

## *Şiddetli preeklampside bileşik biyobelirteçler olarak transaminaz kompleks-platelet oranı (TACPR) ve platelet-albümin oranının (PAR) değerlendirilmesi*

 Gulcan Okutucu<sup>1\*</sup>,  Dilek Sahin<sup>2</sup>

<sup>1</sup>Department of Obstetrics and Gynecology, Division of Perinatology, Ministry of Health Ankara Bilkent City Hospital, Ankara, Turkey

<sup>2</sup>Department of Obstetrics and Gynecology, Division of Perinatology, University of Health Sciences, Ministry of Health Ankara Bilkent City Hospital, Ankara, Turkey

### Abstract

**Aim:** To evaluate the clinical significance of the transaminase complex-platelet ratio (TACPR) and the platelet-albumin ratio (PAR) in predicting obstetric and perinatal outcomes among women with severe preeclampsia (PE).

**Material and Methods:** A retrospective study was conducted at Ankara Bilkent City Hospital, including 60 pregnant women diagnosed with severe PE and 120 gestational age-matched healthy controls. TACPR was calculated as (AST×ALT)/PLT count, and PAR as PLT/Albumin. Clinical, laboratory, and perinatal outcomes were compared between groups. ROC curve analysis was used to assess the predictive performance of TACPR for preterm birth. Subgroup analysis was performed based on proteinuria severity (spot urine <+2 vs. ≥+2).

**Results:** No significant differences were observed between groups in demographic data ( $p > 0.05$ ). TACPR was significantly elevated in severe PE cases ( $p < 0.05$ ), while PAR did not differ significantly ( $p > 0.05$ ). Severe PE was associated with significantly higher rates of preterm birth, low birth weight (LBW), and NICU admission ( $p < 0.001$ ). ROC analysis identified a TACPR cut-off of 0.86 for predicting preterm birth (AUC = 0.701, sensitivity 63.6%, specificity 63.2%). Among severe PE patients, those with ≥+2 proteinuria exhibited higher blood pressures, creatinine, and albumin levels, along with increased rates of preterm birth and LBW. However, TACPR and PAR did not significantly differ across proteinuria levels.

**Conclusion:** TACPR is a novel and accessible composite biomarker that correlates with adverse perinatal outcomes in severe PE. Its integration into clinical assessment could enhance risk stratification. PAR showed limited utility near delivery. Further multicenter prospective studies are warranted.

**Keywords:** severe preeclampsia, TACPR, PAR, perinatal outcomes, composite biomarkers

Corresponding Author\*: Gulcan Okutucu, Department of Obstetrics and Gynecology, Division of Perinatology, Turkish Ministry of Health, Ankara Bilkent City Hospital, Ankara, Turkey.

E-mail: gulcanokutucu@gmail.com

Orcid: 0000-0003-4618-8312

Doi: 10.18663/tjcl.1735877

Received: 06.07.2025 Accepted: 18.08.2025

## Öz

**Amaç:** Şiddetli preeklampsia (PE) tanılı gebelerde, obstetrik ve perinatal sonuçları öngörmeye transaminaz kompleks-platelet oranı (TACPR) ve platelet-albümin oranının (PAR) klinik önemini değerlendirmektir.

**Gereç ve Yöntemler:** Ankara Bilkent Şehir Hastanesinde, şiddetli PE tanısı alan 60 gebe ve gebelik yaşı eşleştirilmiş 120 sağlıklı kadını içeren kontrol grubuyla retrospektif bir çalışma yapılmıştır. Bileşik biyobelirteçlerden TACPR, (AST×ALT) /PLT sayısı olarak, PAR ise PLT/Albümin olarak hesaplandı. Gruplar arasında klinik, laboratuvar ve perinatal sonuçlar karşılaştırıldı. TACPR'nin preterm doğum için öngörü performansını değerlendirmek için ROC eğrisi analizi kullanıldı. Proteinüri şiddetine göre (spot idrar <+2 vs. ≥+2) alt grup analizleri yapıldı.

**Bulgular:** Demografik verilerde gruplar arasında anlamlı farklar gözlenmedi ( $p > 0,05$ ). TACPR, şiddetli PE vakalarında anlamlı olarak yükselirken ( $p < 0,05$ ), PAR arasında anlamlı fark gözlenmedi ( $p > 0,05$ ). Şiddetli PE, preterm doğum, düşük doğum ağırlığı (DDA) ve YBÜ'ye yatış oranlarında anlamlı olarak daha yüksek oranlarla ilişkiliydi ( $p < 0,001$ ). ROC analizi, preterm doğumun tahmininde 0,86'lık bir TACPR kesme noktası belirlemiştir (AUC = 0,701, duyarlılık %63,6, özgüllük %63,2). Şiddetli PE hastaları arasında, ≥+2 proteinüri olanlar, preterm doğum ve DDA oranlarının artmasıyla birlikte daha yüksek kan basıncı, kreatinin ve albümin düzeyleri sergilemiştir. Ancak, TACPR ve PAR, proteinüri düzeyleri arasında önemli bir fark göstermemiştir.

**Sonuç:** TACPR, şiddetli PE'de olumsuz perinatal sonuçlarla ilişkili yeni ve erişilebilir bir bileşik biyobelirteçtir. Klinik değerlendirmeye dahil edilmesi, risk sınıflandırmasını iyileştirebilir. PAR, obstetrik ve perinatal sonuçların tahmininde sınırlı yarara sahiptir. Yine de bu konuda büyük popülasyona sahip çok merkezli prospektif çalışmalara ihtiyaç vardır.

**Anahtar kelimeler:** şiddetli preeklampsia, TACPR, PAR, perinatal sonuçlar, bileşik biyobelirteçler

## Introduction

Preeclampsia (PE) is a complex multisystem disorder of pregnancy characterized by new-onset hypertension and often proteinuria after 20 weeks of gestation [1]. It affects approximately 5-8% of pregnancies, with severe forms occurring in about 1%, contributing significantly to maternal and perinatal morbidity and mortality worldwide [2,3]. Severe PE is frequently associated with hepatic dysfunction, thrombocytopenia, endothelial injury, and adverse perinatal outcomes such as preterm birth, low birth weight (LBW), and neonatal intensive care unit (NICU) admission [1].

Aspartateaminotransferase (AST) and alanineaminotransferase (ALT) are enzymes widely used to assess hepatocellular injury. Both participate in transamination reactions involved in gluconeogenesis, but differ in tissue distribution and clinical specificity. AST exists in both cytosolic and mitochondrial forms and is present in various organs, including the heart, skeletal muscle, kidneys, brain, and red blood cells. As such, elevated AST levels can occur in both hepatic and non-hepatic conditions. In contrast, ALT is a cytosolic enzyme found predominantly in hepatocytes, making it a more specific

and sensitive marker of liver injury, particularly in conditions involving hepatocellular stress, such as acute hepatitis or PE-related liver dysfunction [4,5]. Building on these physiological understandings, we developed a novel composite biomarker, the transaminase complex-platelet ratio (TACPR), to reflect hepatic and hematologic dysfunction simultaneously. TACPR was calculated by multiplying the serum ALT level by the serum AST level and then dividing the result by the PLT count. Some recent evidence suggests that lower albumin levels and altered platelet (PLT) indices are associated with increased PE severity and poorer fetal outcomes [6]. AISheeha et al [7] showed that women with PE had significantly lower PLT counts and altered mean platelet volume (MPV), both of which correlated with disease severity. Another study further demonstrated that combining PLT parameters with serum albumin improved the early prediction of PE [8]. In addition to TACPR, this study also examined the platelet-albumin ratio (PAR) due to its theoretical importance in inflammatory and vascular conditions [9-11], and to the best of our knowledge, there are no studies in the literature regarding the role of these ratios in obstetric populations.

This research primarily aims to evaluate the clinical significance of TACPR and PAR in patients with severe PE, particularly in relation to adverse perinatal outcomes such as preterm delivery, LBW, and admission to the NICU.

## Material and Methods

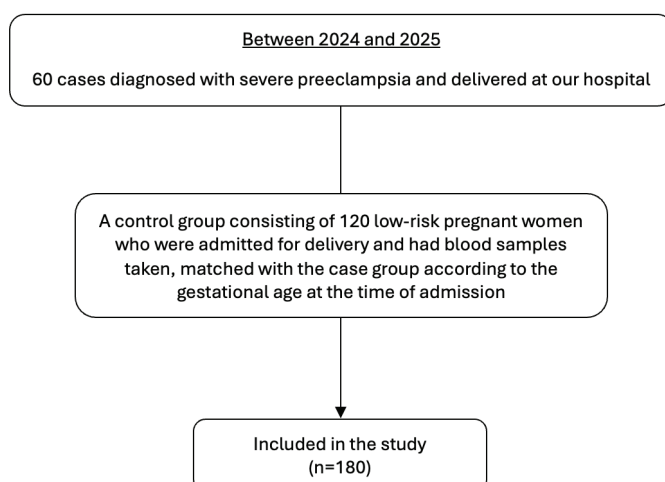
This retrospective study was conducted at the Perinatology Clinic of Ankara Bilkent City Hospital. Institutional review board approval was obtained from the Ethics Committee of the Republic of Turkey Ministry of Health Ankara Bilkent City Hospital (Approval number: TABED 2-25-1117). All stages of the study adhered to the principles of the Declaration of Helsinki.

The study population includes women aged 18-45 between 2024 and 2025. The case group consisted of 60 pregnant women who were hospitalized due to PE with severe features during the study period and gave birth in the maternity ward of our hospital. A control group consisting of 120 healthy, low-risk pregnant women was included, and demographic characteristics and blood sampling at admission for delivery were matched with the case group according to gestational age (Figure 1). Multiple gestations, pregnancies with known multisystemic diseases (malignancies, hepatobiliary, rheumatological, or cardiovascular diseases, etc.), obstetric pathologies other than PE (gestational diabetes, intrahepatic cholestasis of pregnancy, placental abruption, etc.), active viral or bacterial infections, and congenital anomalies were excluded from the study. If medication, medical intervention, or surgery was planned for pregnant women in the study population, these procedures were performed after blood sampling.

Preeclampsia was diagnosed in the presence of new-onset hypertension (systolic blood pressure (BP)  $\geq 140$  mmHg and/or diastolic BP  $\geq 90$  mmHg at least two times with an interval of at least four hours) and proteinuria or significant end-organ dysfunction with or without proteinuria associated with new-onset hypertension in a previously normotensive patient after 20 weeks' gestation [12,13]. Preeclampsia with severe features was diagnosed in the presence of severe hypertension (systolic BP  $\geq 160$  mmHg and/or diastolic BP  $\geq 110$  mmHg at bed rest), development of new-onset cerebral or visual disturbances (e.g. photopsia, scotomata, severe headache or headache that persists and progresses despite analgesic treatment and cannot be explained by alternative diagnoses, etc.), laboratory changes indicating end-organ dysfunction (e.g. thrombocytopenia, at least a two-fold increase in liver function tests or in maternal serum creatinine), or presence of pulmonary edema [12].

The researchers obtained the medical records of the study groups from the hospital database retrospectively. The recorded data included maternal age, body mass index (BMI, calculated by dividing weight in kilograms by the square of height in meters), gravidity, parity, systolic and diastolic BP (mmHg) at hospitalization, C-section rate, severe PE-related maternal symptoms (headache, visual symptoms and/or epigastric pain) and birth-time albumin, creatinine, ALT, AST, PLT, PAR, and TACPR values, as well as perinatal outcomes. Information on birth week and weight, 1- and 5-minute APGAR scores, umbilical cord arterial pH, preterm birth and LBW rates, and NICU admission were recorded as perinatal outcomes. The PAR value was calculated by dividing the PLT (109/L) count by the albumin level (mg/dL). The TACPR was calculated by dividing the multiplication of AST and ALT values by PLT [TACPR=AST (IU/L) X ALT (IU/L) / PLT (109/L)]. While births occurring before the 37th week of pregnancy are considered preterm (14), births weighing less than 2500 grams are considered LBW [15]. The severe PE and control groups were compared in terms of their clinical and demographic characteristics, birth-time PAR and TACPR indices, and perinatal outcomes.

The protein value in the urine of pregnant women with severe PE was evaluated by dipstick and/or 24-hour test. The presence of proteinuria of +2 or more in dipstick test and 300 mg or more in 24-hour urine was considered significant for the diagnosis of PE [12,13]. Severe PE cases were divided into two groups based on the level of proteinuria in spot urine: less than +2 and +2 or higher. The clinical characteristics, laboratory parameters, and perinatal characteristics of these groups were compared.



**Figure 1.** Flowchart of the study population

## Statistical Analysis

The study's sample size was calculated using G Power software (version 3.1; Heinrich-Heine- Universität Düsseldorf). The effect size of 0.80 was determined with a P-value of 0.05 and a power of 95%, and a minimum of 42 cases was planned for each group. Statistical Package for Social Sciences (SPSS version 26.0; Chicago, IL, USA) was utilized for data analysis. Median (interquartile range [IQR]) or mean $\pm$ standard deviation represented continuous variables, while counts (percentages) measured categorical variables. The study assessed the normal distribution of variables through the Kolmogorov– Smirnov test. Two groups' normally distributed continuous variables were compared using the independent t test, while the Mann–Whitney U test was used for non-normally distributed variables. Pearson chi-square or Fisher's exact test was used to compare categorical variables. Receiver operating characteristic (ROC) curve analysis was performed to estimate optimal cut-off values, maximizing sensitivity and specificity according to the Youden index. ROC analysis was performed to evaluate the performance of the TACPR index in predicting preterm birth in cases of severe PE. A p-value of less than 0.05 was deemed significant across all analyses.

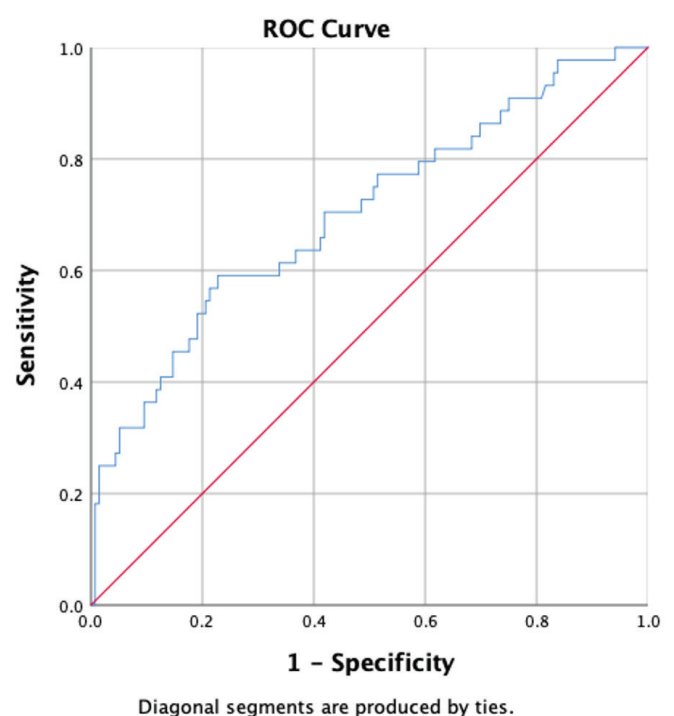
## Results

There was no significant difference in demographic data between severe PE cases and the control group ( $p > 0.05$ ). In the severe PE group, hospitalization systolic and diastolic BP and C-section rates were significantly higher ( $p < 0.001$ , all). The presence of +2 or higher proteinuria in spot urine ( $n = 32$ , 53.3%) and 300 mg or higher proteinuria in 24-hour urine ( $n = 26$ , 43.3%) was present in the majority of cases. The symptoms observed in cases of severe PE were headache ( $n = 55$ , 91.7%), visual symptoms ( $n = 14$ , 23.3%), and epigastric pain ( $n = 4$ , 6.7%), in order of frequency. Creatinine, ALT, AST, and TACPR values were significantly higher in cases of severe PE ( $p < 0.05$ , all). There was no significant difference between the study groups in terms of PLT, albumin, or PAR values ( $p > 0.05$ , all). In cases of severe PE, preterm birth, LBW, and NICU admission rates were found to be significantly higher ( $p < 0.001$ , all). Detailed data on the comparison of the clinical-demographic characteristics, laboratory parameters, and perinatal outcomes of the study groups are presented in Table 1.

Upon grouping severe PE cases according to the level of proteinuria in spot urine, there was no significant difference

between the groups in terms of C-section rates ( $p = 0.721$ ). Hospitalization systolic and diastolic BP were found to be higher in the group with proteinuria of +2 and more ( $p < 0.05$ , all). In cases of severe PE with spot urine protein levels of +2 or more, creatinine and albumin levels were found to be significantly higher ( $p = 0.034$  and  $p = 0.001$ , respectively). Although PAR and TACPR values were higher in cases of severe PE with proteinuria of +2 or higher in spot urine, this difference was not statistically significant between proteinuria groups ( $p > 0.05$ , all). While there was no significant difference in NICU admission rates between proteinuria groups in cases of severe PE ( $p = 0.066$ ), the rates of preterm birth and LBW were higher in the group with proteinuria of +2 or greater ( $p = 0.042$  and  $p < 0.001$ , respectively). Detailed data on the clinical characteristics, laboratory parameters, and perinatal outcomes of severe PE cases according to urine spot protein levels are presented in Table 2.

The summary of the ROC analysis showing the optimal cut-off value of TACPR for predicting preterm birth in cases of severe PE is presented in Table 3. According to this analysis, the optimal cut-off value for TACPR was determined to be 0.86 (63.6 % sensitivity, 63.2 % specificity, area under the curve [AUC] 0.701,  $p < 0.001$ ). The ROC curve for this analysis is shown in Figure 2.



**Figure 2.** Receiver operating characteristic (ROC) curve of TACPR in predicting preterm birth in cases of severe PE. Discussion



**Table 1.** Comparison of clinical-demographic characteristics, laboratory parameters, and perinatal outcomes of study groups.

			Severe PE (n=60)	Controls (n=120)	p-value*
Age			28.48±5.21	28.52±5.22	0.968 <sup>a</sup>
BMI			28.58(3.32)	28.62±3.48	0.210 <sup>a</sup>
Gravidity			2(2)	2(2)	0.465 <sup>b</sup>
Parity			0(1)	0(1)	0.556 <sup>b</sup>
Systolic BP (mmHg)			152.6±12.12	113.2±9.44	<0.001 <sup>a</sup>
Diastolic BP (mmHg)			92.7±9.92	69.3±8.58	<0.001 <sup>a</sup>
C-section rate			51(85%)	64(53.3%)	<0.001 <sup>c</sup>
Urine protein analysis	Dipstick test	< +2	28(46.7%)	-	-
		> +2	32(53.3%)		
	24-hour urine test	None	24(40%)	-	-
		<300 mg	10(16.7%)		
		300 mg-2g	23(38.3%)		
		2-5 g	2(3.3%)		
		> 5g	1(1.7%)		
Headache			55(91.7%)	-	-
Visual symptoms			14(23.3%)		
Epigastric pain			4(6.7%)		
Laboratory parameters		Albumin (mg/dL)	35.6±3.76	36±1.81	0.487 <sup>a</sup>
		Creatinine (g/dL)	0.56±0.12	0.49±0.10	<0.001 <sup>a</sup>
		ALT (IU/L)	14(10)	12(5)	0.017 <sup>b</sup>
		AST (IU/L)	16.5(9)	14(6)	0.003 <sup>b</sup>
		PLT (109/L)	253.3±69.3	246.4±65.8	0.515 <sup>a</sup>
		PAR	7.11±1.87	6.86±1.85	0.380 <sup>a</sup>
		TACPR	0.91(1.36)	0.65(0.77)	0.016 <sup>b</sup>
Perinatal outcomes	Birth week	37(3)	38(2)	<0.001 <sup>b</sup>	
	Birth weight (g)	2822.1±591.6	3195.1±401.3	<0.001 <sup>a</sup>	
	APGAR score (1st min.)	7(1)	7(1)	0.055 <sup>b</sup>	
	APGAR score (5th min.)	9(1)	9(0)	0.057 <sup>b</sup>	
	Umbilical cord arterial pH	7.37(0.06)	7.4(0.01)	0.501 <sup>b</sup>	
	LBW (<2500g)	16(26.7%)	4(3.3%)	<0.001 <sup>c</sup>	
	Preterm birth	28(46.7%)	16(13.3%)	<0.001 <sup>c</sup>	
	NICU admission	13(21.7%)	1(0.8%)	<0.001 <sup>c</sup>	

Abbrev.: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); BP, blood pressure; LBW, low birth weight; NICU, neonatal intensive care unit; PAR, platelet-albumin ratio; PE, preeclampsia; PLT, platelet; TACPR, transaminase complex-platelet ratio. Values are presented as mean±standard deviation and median (IQR), or as number (percentage). \* P-values calculated using: a Independent T test, b Mann–Whitney U test, c Fisher's Exact Test. p<0.05 was considered statistically significant. Statistically significant data are indicated in bold.



**Table 2.** Comparison of clinical characteristics, laboratory parameters, and perinatal outcomes of severe PE cases according to urine spot protein levels.

		Urine spot proteinuria <+2 (n=28)	Urine spot proteinuria >+2 (n=32)	p-value*
Systolic BP (mmHg)		149.17±12.80	155.62±10.82	0.039 <sup>a</sup>
Diastolic BP (mmHg)		89.10±8.38	95.90±10.19	0.007 <sup>a</sup>
C-section rate		23(82.1%)	28(87.5%)	0.721 <sup>c</sup>
Laboratory parameters	Albumin (mg/dL)	37.36±2.62	34.13±4	0.001 <sup>a</sup>
	Creatinine (g/dL)	0.52±0.12	0.59±0.12	0.034 <sup>a</sup>
	ALT (IU/L)	14(13)	14(10)	0.894 <sup>b</sup>
	AST (IU/L)	16.7±8.12	18(10)	0.177 <sup>b</sup>
	PLT (109/L)	258.6±66.2	248.6±72.7	0.584 <sup>a</sup>
	PAR	6.92±1.73	7.28±2.00	0.456 <sup>a</sup>
	TACPR	0.84(1.64)	0.98(1.25)	0.351 <sup>b</sup>
Perinatal outcomes	Birth week	38(3)	36(3)	0.005 <sup>b</sup>
	Birth weight (g)	3159.5±453.6	2526.8±542.3	<0.001 <sup>a</sup>
	APGAR score (1st min.)	7(1)	7(1)	0.020 <sup>b</sup>
	APGAR score (5th min.)	9(1)	9(1)	0.933 <sup>b</sup>
	Umbilical cord arterial pH	7.4(0.04)	7.39(0.07)	0.198 <sup>b</sup>
	LBW (<2500g)	1(3.6%)	15(46.9%)	<0.001 <sup>c</sup>
	Preterm birth	9(32.1%)	19(59.4%)	0.042 <sup>c</sup>
	NICU admission	3(10.7%)	10(31.3%)	0.066 <sup>c</sup>

Abbrev.: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BP, blood pressure; LBW, low birth weight; NICU, neonatal intensive care unit; PAR, platelet-albumin ratio; PE, preeclampsia; PLT, platelet; TACPR, transaminase complex-platelet ratio. Values are presented as mean±standard deviation and median (IQR), or as number (percentage). \* P-values calculated using: a Independent T test, b Mann–Whitney U test, c Fisher's Exact Test. p<0.05 was considered statistically significant. Statistically significant data are indicated in bold.

**Table 3.** ROC analysis table evaluating the predictive performance of TACPR for preterm birth in cases of severe PE.

Variable	Outcome	AUC	Std. error	Sensitivity	Specificity	Asymp. Sig*	95%CI		Cut-off value
							Lower	Upper	
TACPR	Preterm birth	0.701	0.048	63.6%	63.2%	<0.001	0.608	0.795	0.86

Abbrev.: AUC, area under curve; CI, confidence interval; PE, preeclampsia; TACPR, transaminase complex-platelet ratio.

This study investigated the clinical utility of two novel composite biomarkers, TACPR and PAR, in predicting adverse perinatal outcomes among women diagnosed with severe PE. The results demonstrated a significant elevation in TACPR values in the severe PE group, supporting its potential as a marker of hepatic and hematologic dysfunction. However, PAR did not show a statistically significant difference between groups. Our findings showed that TACPR levels were significantly higher in severe PE cases and correlated with preterm delivery, LBW, and NICU admission. The ROC analysis yielded an AUC of 0.701, with an optimal cut-off value of 0.86, suggesting moderate discriminative power in predicting preterm birth. These results align with growing evidence emphasizing the value of liver enzymes, particularly AST and ALT, as indicators of disease severity in PE.

Elevated AST and ALT levels have been associated with liver dysfunction due to endothelial damage and microvascular compromise, which are hallmarks of severe PE (16). Combining transaminases with PLT count, another parameter that declines with disease progression, may provide an integrated reflection of hepatic inflammation and consumptive coagulopathy, two critical pathways in PE pathophysiology. Danielli [17] has advocated for a similar integrated biomarker strategy, emphasizing the importance of perivascular and systemic biomarker panels in reflecting fetal-maternal interface stress in hypertensive disorders of pregnancy.

Despite theoretical justifications, the PAR index did not significantly differentiate between the severe PE and control groups. While albumin levels are known to decline in inflammatory states and vascular leakage, this marker may lack sensitivity when used in isolation or in combination with PLT

count. Prior research by Shi et al [18] emphasized that declining serum albumin levels can serve as an early warning biomarker for the onset of PE, suggesting that hypoalbuminemia reflects increased vascular permeability and endothelial dysfunction. However, in our study, PAR did not reach statistical significance, possibly due to the timing of blood collection near delivery, when albumin changes may plateau. Klajnbard et al [19] established comprehensive laboratory reference intervals for pregnancy, showing that albumin levels progressively decrease during gestation and stabilize near term, largely due to hemodilution and plasma volume expansion. This natural decline may mask pathological changes, particularly when relying on single-time-point measurements rather than dynamic trends across pregnancy stages. Nonetheless, AlSheeha et al [7] reported that altered PLT indices, including MPV, in conjunction with serum albumin, may enhance PE prediction when used dynamically rather than at a single time point. This suggests that temporal dynamics of albumin decline, rather than a single time-point value, may hold greater diagnostic promise.

The significance of AST-derived ratios in PE risk assessment is further supported by the study of Ipek et al [20], who demonstrated that the Aspartate Aminotransferase to Platelet Ratio Index (APRI) measured in the first trimester can serve as an early predictor of superimposed PE. This research aligns with our findings, as both APRI and TACPR incorporate AST and PLT count to reflect hepatic stress and thrombocytopenia. However, while APRI is well-established for staging chronic liver disease, TACPR incorporates ALT into the equation, which could offer improved specificity for acute hepatic involvement in PE. This similarity suggests that hepatic-hematologic composite indices could be useful for stratifying PE risk early in pregnancy and during critical perinatal stages.

An interesting secondary finding of this study was the stratification of PE patients based on urine protein levels. Cases with  $\geq +2$  proteinuria had significantly higher systolic/diastolic BPs and increased risk of preterm birth and LBW. These findings are consistent with previous work suggesting that higher proteinuria reflects more severe glomerular injury and correlates with worse fetal outcomes [21].

This study has several limitations that should be considered when interpreting the findings. First, its retrospective design may introduce bias due to reliance on medical records and limited control over confounding variables. Second, as a single-center study conducted in a tertiary referral hospital, the results may not be generalizable to broader populations, particularly in primary care or rural settings. Third, although the

sample size was statistically adequate, it was modest, limiting the robustness of subgroup analyses, especially in proteinuria stratification. Fourth, biomarker measurements were taken only at the time of delivery, preventing evaluation of their dynamic changes or predictive value earlier in pregnancy. Lastly, TACPR and PAR were derived and validated within the same cohort without external or prospective validation, limiting the clinical applicability of the proposed cut-off values.

A key strength of this study is the development and evaluation of TACPR, a novel and easily calculable biomarker derived from routine laboratory tests, which showed significant association with adverse perinatal outcomes in severe PE. The inclusion of well-matched control group and the use of real clinical data obtained from a tertiary center increase the practical importance of this study, especially in settings with limited resources where access to advanced biomarkers may be restricted.

In conclusion, this study demonstrates that the TACPR is a promising, easily accessible biomarker that reflects both hepatic and hematologic dysfunction in severe PE. Elevated TACPR levels were significantly associated with adverse perinatal outcomes, including preterm birth and LBW, and showed moderate predictive ability with a defined cut-off value. In contrast, the PAR did not exhibit a statistically significant difference between groups, suggesting limited utility in the near-delivery period. These findings support the integration of TACPR into clinical assessment models, particularly in settings where advanced diagnostic tools may not be readily available. Future prospective, multicenter studies with longitudinal follow-up are warranted to validate TACPR as a routine tool for risk stratification and to explore its role in the early prediction of disease progression and fetal outcomes in preeclamptic pregnancies.

#### **Ethics Committee Approval**

Institutional review board approval was obtained from the Ethics Committee of the Republic of Turkey Ministry of Health Ankara City Hospital (Approval number: TABED 2-25-1117).

#### **Conflicts of Interest**

The authors declare they have no conflicts of interest.

#### **Financial Disclosure**

The authors declared that this study has received no financial support.

#### **Acknowledgments**

Special thanks to all the health care staff of our hospital who work devotedly for the health of our community.

## Authors Contributions

Writing original draft: G.O., Concept and Design: D.S., Data Collection or Processing: G.O., Analysis or Interpretation: G.O., Literature Search: G.O., D.S., Writing-editing: G.O., D.S., Supervision: D.S.

## References

1. Poon LC, Shennan A, Hyett JA, Kapur A, Hadar E, Divakar H et al. The International Federation of Gynecology and Obstetrics (FIGO) initiative on pre-eclampsia: A pragmatic guide for first-trimester screening and prevention. *Int J Gynaecol Obstet* 2019; 145(S1): 1-33.
2. Turpin CA, Sakyi SA, Owiredun WKBA, Ephraim RKD, Anto EO. Association between adverse pregnancy outcome and imbalance in angiogenic regulators and oxidative stress biomarkers in gestational hypertension and preeclampsia. *BMC Pregnancy Childbirth* 2015; 15: 189.
3. Emeruwa UN, Gyamfi-Bannerman C, Laurent LC. Biomarkers and the Risk of Preeclampsia. *JAMA*. 2023; 329: 539-41.
4. Giannini EG, Testa R, Savarino V. Liver enzyme alteration: a guide for clinicians. *CMAJ* 2005; 172: 367.
5. Pratt DS, Kaplan MM. Evaluation of abnormal liver-enzyme results in asymptomatic patients. *N Engl J Med* 2000; 342: 1266-71.
6. Woldeamanuel GG, Tlaye KG, Wu L, Poon LC, Wang CC. Platelet count in preeclampsia: a systematic review and meta-analysis. *Am J Obstet Gynecol MFM* 2023; 5: 100979.
7. AlSheeha MA, Alaboudi RS, Alghasham MA, Iqbal J, Adam I. Platelet count and platelet indices in women with preeclampsia. *Vasc Health Risk Manag* 2016; 12: 477-80.
8. Al Ghazali B, Al-Taie AA-H, Hameed RJ. Study of the clinical significance of serum albumin level in preeclampsia and in the detection of its severity. *Am J BioMed* 2014; 2: 964-74.
9. Cao S-L, Guo-Qing Z, Jing L, Li B, Xiao-Mei L, Quan-Peng J et al. Platelet-to-Albumin Ratio is a Potential Biomarker for Predicting Diabetic Nephropathy in Patients with Type 2 Diabetes. *Biomarkers in Medi* 2023; 17: 841-8.
10. Hao P, Feng S, Suo M, Wang S, Wu X. Platelet to albumin ratio: A risk factor related to prognosis in patients with non-ST-segment elevation acute coronary syndrome undergoing percutaneous coronary intervention. *Int J Cardiol* 2024; 395: 131588.
11. Tan J, Song G, Wang S, Dong L, Liu X, Jiang Z et al. Platelet-to-Albumin Ratio: A Novel IgA Nephropathy Prognosis Predictor. *Front Immunol* 2022; 13: 842362.
12. Gestational Hypertension and Preeclampsia: ACOG Practice Bulletin, Number 222. *Obstet Gynecol* 2020; 135: e237-e60.
13. Magee LA, Brown MA, Hall DR, Gupte S, Hennessy A, Karumanchi SA et al. The 2021 International Society for the Study of Hypertension in Pregnancy classification, diagnosis & management recommendations for international practice. *Pregnancy Hypertens* 2022; 27: 148-69.
14. Goldenberg RL, Culhane JF, Iams JD, Romero R. Epidemiology and causes of preterm birth. *Lancet* 2008; 371: 75-84.
15. Cutland CL, Lackritz EM, Mallett-Moore T, Bardaji A, Chandrasekaran R, Lahariya C, et al. Low birth weight: Case definition & guidelines for data collection, analysis, and presentation of maternal immunization safety data. *Vaccine* 2017; 35: 6492-500.
16. Al-Jameil N, Aziz Khan F, Fareed Khan M, Tabassum H. A brief overview of preeclampsia. *J Clin Med Res* 2014; 6: 1-7.
17. Danielli M. Role of perivascular biomarkers at the fetal-maternal interface in hypertensive disorders of pregnancy: University of Leicester; Thesis submitted for the Degree of Doctor of Philosophy 2022.
18. Shi JM, Yang Z, Li FQ, Wang GJ. Preliminary study of human serum albumin level in early warning onset of preeclampsia]. *Zhonghua Fu Chan Ke Za Zhi* 2020; 55: 29-35.
19. Klajnbard A, Szecsi PB, Colov NP, Andersen MR, Jørgensen M, Bjørngaard B et al. Laboratory reference intervals during pregnancy, delivery and the early postpartum period. *Clin Chem Lab Med* 2010; 48: 237-48.
20. İpek G, Tanaçan A, Ağaoğlu Z, Gülçin Baştemur A, Gülen Yıldız E, Şahin D. The role of aspartate aminotransferase to platelet ratio index (APRI) in the first trimester for the prediction of superimposed preeclampsia: A case-control study from a tertiary center. *Pregnancy Hypertens* 2024; 37: 101132.
21. Xiao J, Fan W, Zhu Q, Shi Z. Diagnosis of proteinuria using a random urine protein-creatinine ratio and its correlation with adverse outcomes in pregnancy with preeclampsia characterized by renal damage. *J Clin Hypertens (Greenwich)* 2022; 24: 652-9.

This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<http://creativecommons.org/licenses/by/4.0/>)