Clinical and Experimental Obstetrics & Gynecology

Clin. Exp. Obstet. Gynecol. **2026**; 53(1): 45511
https://doi.org/10.31083/CEOG45511

Original Research

Predictive Risk Model for Severe Preeclampsia Associated With Placental Abruption

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Academic Editor: Michael H. Dahan
Submitted: 3 August 2025 Revised: 11 October 2025 Accepted: 21 October 2025 Published: 26 January 2026

Abstract

Background: To evaluate the feasibility and clinical utility of developing a risk prediction model for placental abruption among patients with severe preeclampsia, incorporating maternal age, baseline systolic blood pressure (SBP), baseline diastolic blood pressure (DBP), retroplacental hematoma width, placental growth factor (PlGF), and the soluble fms-like tyrosine kinase-1/PlGF factor (sFlt-1/PlGF) ratio. **Methods**: This retrospective study enrolled 260 patients with severe preeclampsia who were admitted to the hospital from January 2022 to October 2024. The cases were randomly divided into a training set (n = 182) and a validation set (n = 78) in a 7:3 ratio. The primary outcome was placental abruption. Clinical data, imaging parameters, and biomarker levels were collected. Univariate and multivariate logistic regression analyses were performed to identify independent risk factors. A nomogram was subsequently developed, and its predictive performance was evaluated and validated. **Results**: The incidence of placental abruption was 35.16% (64/182) in the training set and 33.33% (26/78) in the validation set. Multivariate logistic regression analysis identified age, baseline SBP, baseline DBP, retroplacental hematoma width, PlGF, and the sFlt-1/PlGF ratio as independent risk factors (all $p < 0.05$). The nomogram demonstrated good discriminative ability, with C-index values of 0.890 and 0.848 in the training and validation sets, respectively. The area under the curve (AUC) was 0.890 (95% confidence interval [CI]: 0.827–0.953) and 0.848 (95% CI: 0.733–0.963), respectively. Sensitivity was 0.766 in the training set and 0.588 in the validation set, whereas specificity was 0.890 and 0.944, respectively. Calibration curves showed excellent agreement between predicted and observed outcomes. The Hosmer-Lemeshow test yielded p-values of 0.583 and 0.290, respectively, suggesting good model fit. **Conclusions**: The nomogram model, incorporating age, baseline SBP, baseline DBP, retroplacental hematoma width, PlGF, and sFlt-1/PlGF ratio effectively predicted the risk of placental abruption in patients with severe preeclampsia. This model may support early clinical intervention. However, the use of single-center data and lack of external validation limit its generalizability, highlighting the need for further verification through multicenter studies.

Keywords: severe preeclampsia; placental abruption; prediction model; imaging; biomarkers; PlGF; sFlt-1/PlGF ratio

1. Introduction

Severe preeclampsia is a pregnancy-specific complication characterized by hypertension, proteinuria, and multi-organ dysfunction. Its global incidence ranges from approximately 2% to 8%, significantly contributing to adverse maternal and fetal outcomes [1–3]. Placental abruption, a severe complication of preeclampsia, occurs in 0.4%–2.3% of cases. It can result in fetal distress, preterm birth, stillbirth, maternal hemorrhage, and a three- to five-fold increase in perinatal mortality [4,5]. A study indicates that placental abruption in severe preeclampsia is closely related to placental ischemia-hypoxia and vascular endothelial injury [6]. However, the pathogenesis remains complex, making clinical prediction challenging [7,8].

Currently, clinical prediction of placental abruption mainly depends on single indicators, such as ultrasound detection of retroplacental hematoma or biomarkers like placental growth factor (PlGF) and soluble fms-like tyrosine kinase-1 (sFlt-1). However, the predictive performance of these indicators alone remains limited [9]. For instance, decreased PlGF levels indicate placental insufficiency, but their sensitivity alone ranges from 60% to 70%. Similarly, ultrasound detection of retroplacental hematoma depends greatly on examination timing and operator experience [10]. Although clinical factors (e.g., blood pressure and maternal age) have demonstrated associations with placental abruption, existing studies have not integrated imaging markers, clinical biomarkers, and baseline characteristics into a unified predictive model. This gap limits the accuracy of early risk assessment in clinical practice. For example, one previous study developed a predictive model for placental abruption in preeclampsia patients [11] but included only clinical parameters and partial biomarkers, excluding imaging indicators such as retroplacental hematoma width [4]. In contrast, our study integrates multiple indicators, including age, baseline blood pressure (clinical indicators), retroplacental hematoma width (imaging indicator), and PlGF and sFlt-1/PlGF ratio (molecular biomarkers). This approach comprehensively covers the key aspects of placental abruption pathogenesis and enhances predictive accuracy [12]. A study has also emphasized the importance of integrating multiple indicators

to improve prediction performance, further supporting our model construction strategy [13].

With advances in precision obstetrics, the development of multidimensional predictive models has become essential to improving maternal and neonatal outcomes. This study aims to establish a nomogram-based predictive model by analyzing interactions among maternal age, blood pressure indices, placental imaging features, and biomarkers. This model provides a reliable tool for the early identification and intervention of placental abruption in patients with severe preeclampsia, ultimately reducing the risk of adverse pregnancy outcomes.

2. Materials and Methods

2.1 Study Population

This retrospective study enrolled 260 patients with severe preeclampsia between January 2022 and October 2024 in our hospital. The inclusion criteria were: (1) diagnosis of severe preeclampsia, defined as systolic blood pressure (SBP) $\geq$160 mmHg and/or diastolic blood pressure (DBP) $\geq$110 mmHg after 20 weeks of gestation, accompanied by proteinuria $\geq$5 g/24 h or complications such as thrombocytopenia, hepatic or renal dysfunction, pulmonary edema, new-onset central nervous system abnormalities, or visual disturbances; (2) singleton pregnancy; (3) complete clinical and imaging data. Exclusion criteria included: (1) pre-existing chronic hypertension, renal diseases, or autoimmune disorders; (2) fetal anomalies or chromosomal abnormalities; (3) history of placental abruption; (4) placental abruption upon admission. This study focused exclusively on predicting placental abruption in patients with severe preeclampsia. Diagnostic methods for ovarian diseases or extra-ovarian disorders, such as ovarian imaging or diagnostic hysteroscopy, were therefore not employed.

Patients were allocated randomly using a complete randomization method. Specifically, SPSS (version 26.0, IBM Corp., Armonk, NY, USA), R (version 4.2.3, R Foundation for Statistical Computing, Vienna, Austria) were employed to generate random numbers (ranging from 0 to 1) for each eligible patient. Patients were then sorted in ascending order. The first 70% (n = 182) were assigned to the training set, and the remaining 30% (n = 78) formed the validation set [4]. This method ensured comparability of baseline characteristics (age, gestational age, blood pressure) between groups, thereby minimizing selection bias. The hospital ethics committee approved the study, and informed consent was obtained from all participants.

2.2 Sample Size Estimation

Sample size calculation was conducted before starting the study, based on the number of independent predictors and the expected event rate. A general rule for logistic regression is to have at least 10 events (cases of placental abruption) per independent variable. In this study, six independent predictors were included. Considering a significance level ($\alpha = 0.05$), statistical power ($1-\beta = 0.80$), and an expected incidence of placental abruption of approximately 30% among patients with severe preeclampsia, the minimum required sample size was estimated at 200 cases. This ensured sufficient power to detect associations between predictors and outcomes. The final enrolled sample size of 260 cases exceeded this minimum, providing robust statistical reliability for model development and validation.

2.3 Diagnostic Criteria of Placental Abruption

Placental abruption was diagnosed based on: (1) Clinical symptoms of sudden, persistent abdominal or back pain, with or without vaginal bleeding, possibly progressing to shock; (2) Ultrasound findings of retroplacental hematoma (hypoechoic area between placenta and uterine wall), placental thickening, or marginal elevation; (3) Postpartum placental examination revealing maternal-side clots or indentations. Diagnosis required either criteria (1) and (2) or (1) and (3) [14].

2.4 Data Collection

Demographic and clinical data collected included age, gestational age, gravidity, parity, body mass index (BMI), family history of hypertension, and diabetes. Baseline SBP and baseline DBP were measured upon admission. Ultrasound assessments included placental thickness and retroplacental hematoma width, if present. Biomarker analyses included: (1) PlGF and sFlt-1 measured by enzyme-linked immunosorbent assay (ELISA) using kits (PlGF kit lot number: PLG-202205; sFlt-1 kit lot number: sFLT-202206; R&D Systems, Minneapolis, MN, USA), with calculation of the sFlt-1/PlGF ratio; (2) D-dimer levels measured via clotting assays using a D-dimer clotting assay kit (lot number: DD-202207; Siemens Healthineers, Erlangen, Germany) (3) Platelet count measured using an automated hematology analyzer (Model: XN-9000; Sysmex Corporation, Kobe, Japan); (4) Serum creatinine and serum alanine aminotransferase (ALT) measured by biochemical assays. All procedures were strictly followed by the manufacturer's protocols. Due to the limited scope of data collection in this single-center study, uterine artery pulsatility index (PI) and coagulation function markers (e.g., prothrombin time) were not included. These indicators will be included in future expanded studies to enhance the comprehensiveness of the model.

2.5 Statistical Analysis

Data were analyzed using SPSS (version 26.0, IBM Corp., Armonk, NY, USA), R (version 4.2.3, R Foundation for Statistical Computing, Vienna, Austria). Continuous variables were expressed as mean $\pm$ standard deviation (SD) for normally distributed data (tested by the Shapiro-Wilk test) or as median (interquartile range, Q1–Q3) for non-normally distributed data. Group comparisons were performed using independent *t*-tests or Mann-Whitney U

Table 1. Comparison of baseline characteristics between training and validation sets in patients with severe preeclampsia.

Indicators	Training set (n = 182)	Validation set (n = 78)	t/χ^2	p
Age (years)	28.69 ± 4.42	29.52 ± 4.45	1.385	0.167
Gestational age (weeks)	33.05 ± 2.14	33.21 ± 2.45	0.529	0.598
Gravidity (times)	2.00 (1.00, 3.00)	2.00 (1.00, 3.00)	0.752	0.452
Parity (times)	1.00 (0.00, 2.00)	1.00 (0.00, 2.00)	0.489	0.625
BMI (kg/m^2)	25.56 ± 3.15	25.85 ± 3.24	0.675	0.501
Baseline SBP (mmHg)	167.05 ± 10.36	164.32 ± 11.21	1.899	0.059
Baseline DBP (mmHg)	105.41 ± 8.51	107.52 ± 9.21	1.787	0.075
Family history of hypertension (yes/no)	42/140 (23.08/76.92)	25/53 (32.05/67.95)	2.299	0.129
Diabetes history (yes/no)	28/154 (15.38/84.62)	18/60 (23.08/76.92)	2.219	0.136
Placental thickness (mm)	35.71 ± 5.01	36.52 ± 4.89	1.203	0.231
Retroplacental hematoma width (mm)	14.73 ± 2.96	13.98 ± 2.89	1.886	0.061
PlGF (pg/mL)	107.51 ± 29.23	109.52 ± 29.53	0.507	0.613
sFlt-1/PlGF ratio	67.13 ± 8.23	68.32 ± 8.56	1.056	0.292
D-dimer (mg/L)	2.98 ± 0.65	3.01 ± 0.56	0.355	0.723
Platelet count (×10^9/L)	118.53 ± 40.09	115.65 ± 41.56	0.525	0.601
Serum creatinine (μmol/L)	86.66 ± 13.43	88.56 ± 12.89	1.058	0.291
Serum ALT (U/L)	57.71 ± 17.78	58.65 ± 16.65	0.398	0.691

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; PlGF, placental growth factor; sFlt-1/PlGF, soluble fms-like tyrosine kinase-1/placental growth factor; ALT, alanine aminotransferase.

tests, respectively. Categorical variables were compared using chi-square or Fisher's exact tests, and results were reported as number (percentage). Univariate logistic regression was first used to identify potential predictors ($p < 0.05$). Significant variables were then entered into a multivariate logistic regression to determine independent risk factors. Multicollinearity was assessed using the variance inflation factor (VIF); variables with VIF <5 indicated no significant multicollinearity and were included in the analysis. A nomogram was constructed based on the identified predictors. Model performance was evaluated using receiver operating characteristic (ROC) curves, with area under the curve (AUC) and 95% confidence intervals (CI). Calibration was assessed using calibration curves and the Hosmer-Lemeshow test. Statistical significance was set at $p < 0.05$. To evaluate model overfitting, bootstrap validation with 1000 resamples was performed on the training set. The C-index after bootstrap validation was 0.872, closely matching the original C-index (0.890), indicating good model stability and low overfitting risk.

3. Results

3.1 Comparison of Baseline Characteristics Between Training and Validation Sets

A total of 260 patients with severe preeclampsia were enrolled and divided into a training set (n = 182) and a validation set (n = 78). No significant differences (all $p > 0.05$) were found between the two sets in age, gestational age, gravidity, parity, BMI, baseline blood pressure, hypertension or diabetes history, placental thickness, retroplacental hematoma width, PlGF, sFlt-1/PlGF ratio, D-dimer, platelet count, serum creatinine, or ALT levels, confirming comparability (Table 1).

Statistically, this table verifies the absence of significant baseline differences (all $p > 0.05$) between groups. Achieving baseline balance ensures that the model built on the training set is not biased by group differences during validation. This is a critical prerequisite for evaluating a model's external generalizability, consistent with predictive modeling research standards.

3.2 Analysis of Risk Factors for Placental Abruption in the Training Cohort

In the training cohort, placental abruption occurred in 64 patients (35.16%), while 118 patients (64.84%) did not experience this complication. In the validation set, placental abruption occurred in 26 patients (33.33%). Univariate analysis indicated statistically significant differences ($p < 0.05$) between the abruption and non-abruption groups in maternal age, baseline SBP, baseline DBP, retroplacental hematoma width, PlGF, and sFlt-1/PlGF ratio. However, no significant differences ($p > 0.05$) were observed in gestational age, gravidity, parity, BMI, family history of hypertension, diabetes mellitus, placental thickness, D-dimer levels, platelet count, serum creatinine, or serum ALT (Table 2).

The statistical purpose of Table 2 is to screen potential risk factors for placental abruption (variables with $p < 0.05$ are included in subsequent multivariate regression analysis). Variables overlapping with Table 1 (e.g., age, baseline blood pressure) represent key clinical indicators serving dual roles in baseline balance verification (Table 1) and risk factor identification (Table 2).

Table 2. Analysis of risk factors for placental abruption in the training cohort.

Indicators	Placental abruption group (n = 64)	Non-abruption group (n = 118)	t/χ^2	p
Age (years)	30.25 ± 4.56	27.85 ± 4.12	3.613	0.001
Gestational age (weeks)	32.85 ± 2.32	33.16 ± 2.05	0.929	0.354
Gravidity (times)	2.00 (1.00, 3.00)	2.00 (1.00, 3.00)	0.915	0.360
Parity (times)	1.00 (0.00, 2.00)	1.00 (0.00, 2.00)	1.183	0.237
BMI (kg/m^2)	26.12 ± 3.25	25.25 ± 3.08	1.785	0.076
Baseline SBP (mmHg)	170.36 ± 10.56	165.25 ± 9.85	3.258	0.001
Baseline DBP (mmHg)	108.25 ± 8.56	103.85 ± 8.12	3.425	0.001
Family history of hypertension (yes/no)	15/49 (23.44/76.56)	27/91 (22.88/77.12)	0.007	0.932
Diabetes history (yes/no)	10/54 (15.63/84.37)	18/100 (15.25/84.75)	0.004	0.947
Placental thickness (mm)	36.05 ± 5.32	35.52 ± 4.85	0.681	0.497
Retroplacental hematoma width (mm)	16.36 ± 3.56	13.85 ± 2.12	5.961	0.001
PlGF (pg/mL)	98.56 ± 25.32	112.36 ± 30.15	3.113	0.002
sFlt-1/PlGF ratio	79.65 ± 12.56	68.52 ± 8.32	7.162	0.001
D-dimer (mg/L)	3.12 ± 0.95	2.88 ± 0.72	1.913	0.057
Platelet count (×10^9/L)	115.36 ± 35.62	120.25 ± 42.36	0.785	0.434
Serum creatinine (μmol/L)	89.15 ± 15.32	85.32 ± 12.15	1.849	0.066
Serum ALT (U/L)	61.16 ± 20.56	55.85 ± 15.85	1.939	0.054

Table 3. Multivariate logistic regression analysis of placental abruption in severe preeclampsia.

Indicators	β	SE	Wald	p	OR	95% CI
Age	0.074	0.046	2.516	0.013	1.076	1.008–1.150
PlGF	–0.018	0.007	6.149	0.013	0.982	0.969–0.996
Baseline SBP	0.051	0.021	5.789	0.016	1.052	1.009–1.096
Baseline DBP	0.075	0.028	7.323	0.007	1.078	1.021–1.138
Retroplacental hematoma width	0.278	0.077	13.150	0.001	1.320	1.136–1.534
sFlt-1/PlGF ratio	0.096	0.022	18.442	0.001	1.100	1.053–1.149

SE, standard error; OR, odds ratio; CI, confidence interval.

3.3 Multivariate Logistic Regression Analysis of Placental Abruption in Severe Preeclampsia

Placental abruption (yes = 1, no = 0) was defined as the dependent variable. Variables with $p < 0.05$ in the univariate analysis were included as covariates in the multivariate logistic regression model. The results showed that maternal age, baseline SBP, baseline DBP, retroplacental hematoma width, and sFlt-1/PlGF ratio significantly increased the risk of placental abruption (all $p < 0.05$). PlGF was significantly associated with a reduced risk ($p < 0.05$) (Table 3).

3.4 Construction of the Nomogram Prediction Model

A nomogram was constructed based on the independent risk factors identified by multivariate logistic regression analysis. Each variable was assigned a score corresponding to its regression coefficient. The total score predicts the probability of placental abruption (Fig. 1). A higher total score indicates a higher predicted risk. Variable importance, determined by the absolute value of regression coefficients, ranked as follows: retroplacental hematoma width ($\beta = 0.278$) > sFlt-1/PlGF ratio ($\beta = 0.096$) > baseline DBP ($\beta = 0.075$) > age ($\beta = 0.074$) > baseline SBP ($\beta = 0.051$) > PlGF ($\beta = 0.018$). Thus, retroplacental hematoma width had the highest relative contribution, whereas PlGF had the lowest.

3.5 Evaluation and Validation of the Nomogram Model

In the training set, the nomogram demonstrated a C-index of 0.890, and the Hosmer-Lemeshow test yielded a p-value of 0.583, indicating good model fit. Bootstrap validation with 1000 resamples resulted in a C-index of 0.872, suggesting good model stability and no significant overfitting. The ROC curve revealed an AUC of 0.890 (95% CI: 0.827–0.953), with a sensitivity of 0.766 and specificity of 0.890. In the validation set, the C-index was 0.848, with a Hosmer-Lemeshow test p-value of 0.290. The AUC was 0.848 (95% CI: 0.733–0.963), with a sensitivity of 0.588 and specificity of 0.944. Calibration and ROC curves are shown in Figs. 2,3.

3.6 Decision Curve Analysis

Decision curve analysis indicated that applying the nomogram model to predict placental abruption provided optimal net benefit when the threshold probability ranged between 0.10 and 0.95 (Fig. 4).

4. Discussion

In recent years, severe preeclampsia, a high-risk pregnancy-specific complication, has received considerable attention due to its association with placental abruption

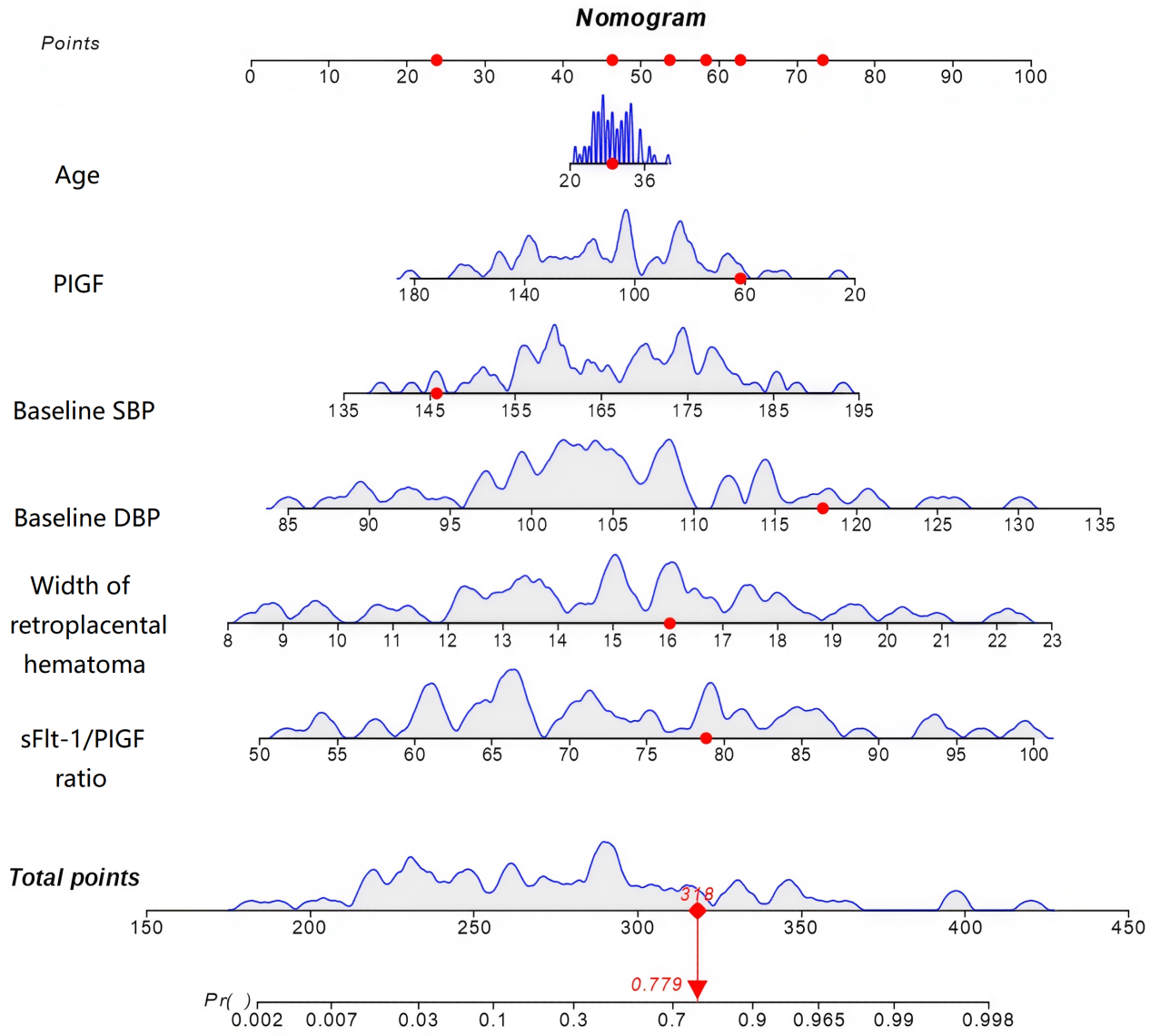

Fig. 1. Nomogram prediction model for placental abruption in patients with severe preeclampsia. Note: Each axis represents different variable values. To predict a patient's risk, identify each variable's specific value on its axis, sum the points, and locate the corresponding probability of placental abruption on the "Risk Axis" based on the total score. The arrows in the nomogram serve as a visual bridge, linking patient indicators to their corresponding scores, and then converting the total score into a predicted probability of placental abruption, enabling rapid risk assessment without complex calculations.

[15]. The global incidence of severe preeclampsia ranges from 2% to 8%. Placental abruption, one of its most severe complications, occurs in 0.4%–2.3% of pregnancies, increasing perinatal mortality by three- to five-fold. It can result in severe consequences, such as fetal distress and significant maternal hemorrhage [16–18].

Currently, clinical prediction of placental abruption mainly relies on single indicators, such as ultrasound detection of retroplacental hematoma or biomarkers like PlGF and sFlt-1. However, these methods have significant limitations. For instance, the sensitivity of PlGF alone is only 60%–70%, limiting its effectiveness in early risk identification. Additionally, ultrasound examinations are subjective, influenced by operator experience and timing. Although clinical factors such as blood pressure are related to placental abruption, there remains a lack of quantitative risk prediction models [19]. This single-dimensional approach hinders precise early intervention, potentially causing clinicians to miss the critical period for improving maternal and fetal outcomes.

To address this issue, our study integrated multiple indicators, including age, baseline blood pressure, retroplacental hematoma width, and sFlt-1/PlGF ratio, to construct a nomogram prediction model. The results demonstrated

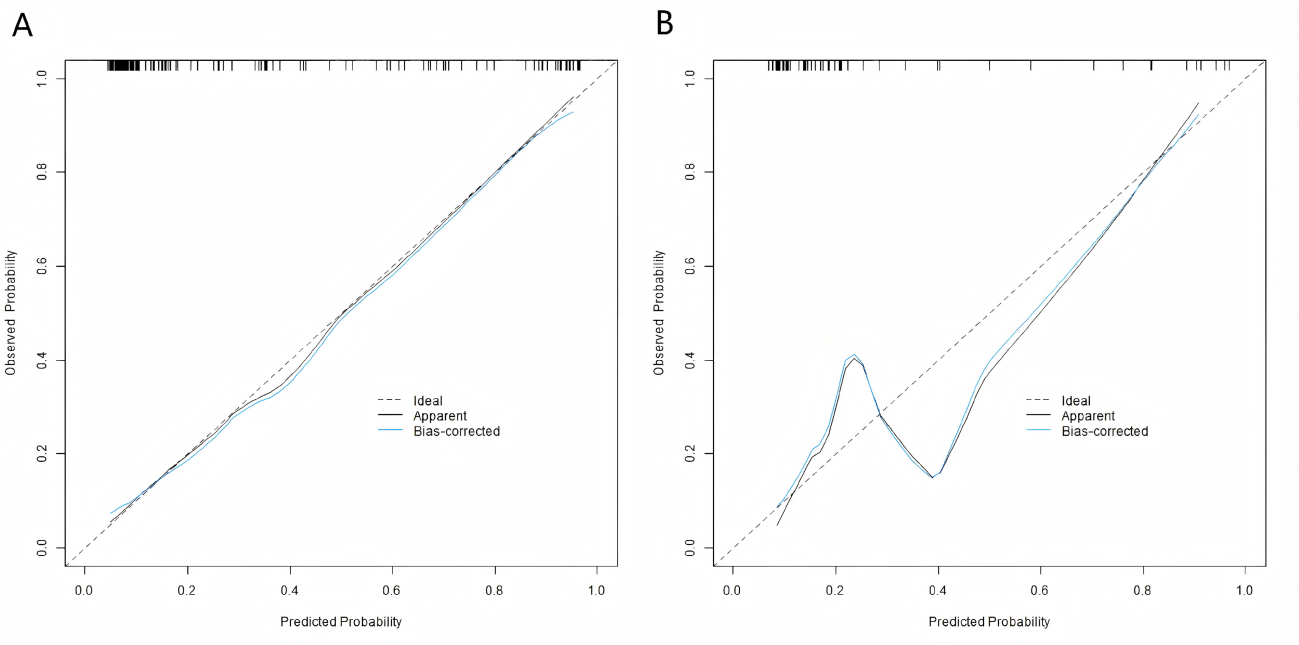

Fig. 2. Calibration curves in the training set (A) and validation set (B).

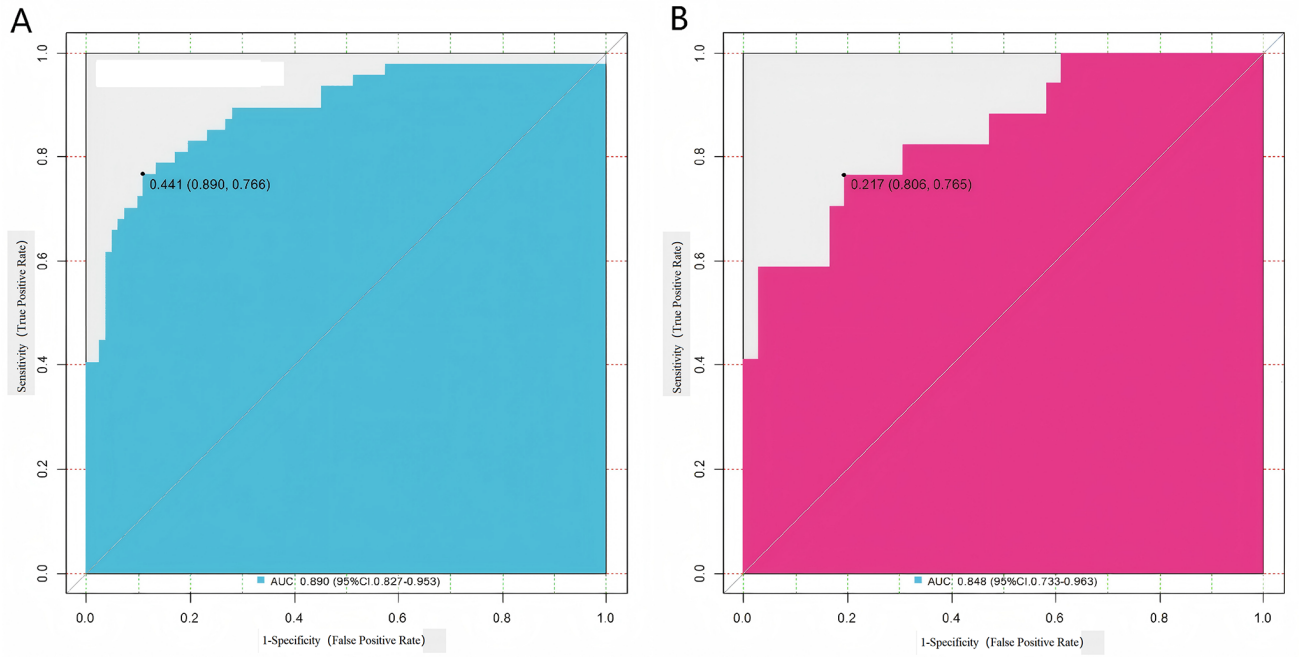

Fig. 3. Receiver operating characteristic (ROC) curves in the training set (A) and validation set (B). AUC, area under the curve.

that the model achieved a C-index of 0.890 in the training set and 0.848 in the validation set, with an AUC greater than 0.84 in both sets. The model also showed excellent sensitivity and specificity, confirming the feasibility and superiority of multi-indicator integration. This provides clinicians with a new quantitative tool for early intervention and risk stratification of placental abruption in patients with severe preeclampsia.

The comprehensive predictive model developed in this study, integrating imaging and clinical biomarkers, overcomes the limitations of traditional single-indicator models. It highlights the advantages of a multi-dimensional approach in the context of precision obstetrics. From a methodological standpoint, six independent risk factors were identified through univariate and multivariate logistic regression, encompassing demographic factors (age), clinical signs (blood pressure), imaging features (retroplacental hematoma width), and molecular markers (PlGF, sFlt-1/PlGF ratio). Thus, the model effectively integrates macro-level clinical characteristics and micro-level patho-

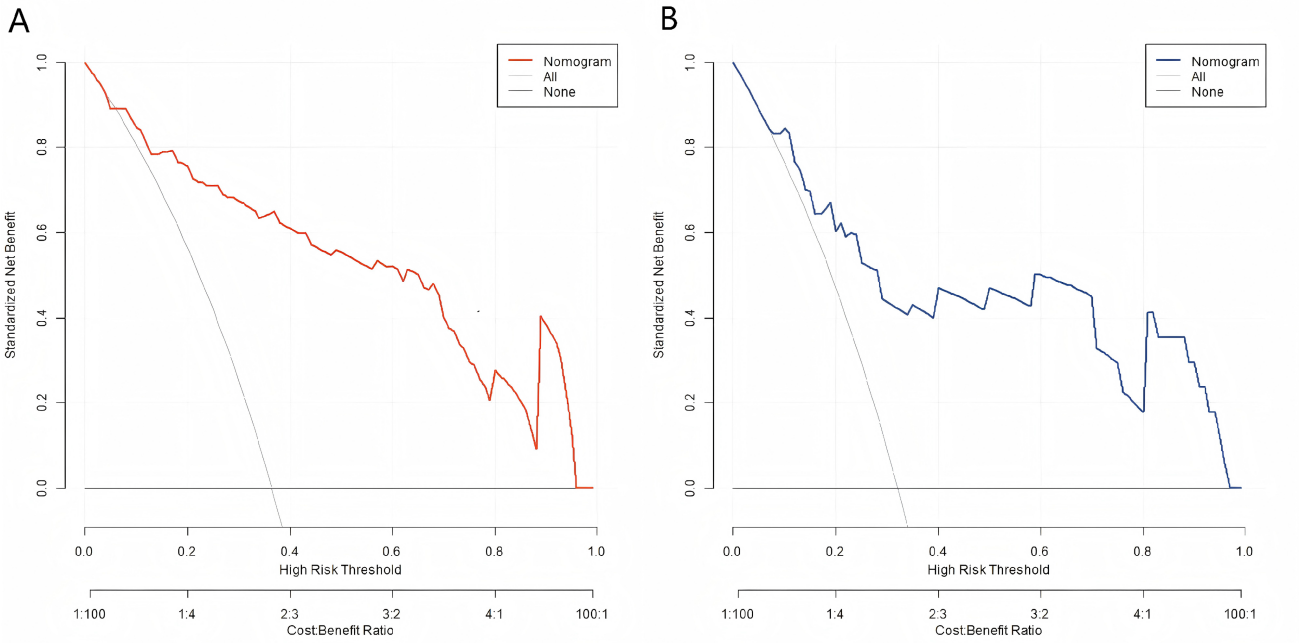

Fig. 4. Decision curves in the training set (A) and validation set (B).

logical indicators. This integrative model is scientifically robust because placental abruption involves multiple pathological mechanisms, including vascular endothelial injury, placental ischemia-hypoxia, and abnormal coagulation. Single indicators reflect only one aspect of the pathological process, whereas multiple combined indicators provide comprehensive coverage of disease progression. A study focused only on decreased PlGF levels, indicating placental insufficiency, and neglected the effects of increased blood pressure on placental hemodynamics [20]. By simultaneously including baseline systolic and DBP and PlGF, this model captures the cascade of "abnormal vascular pressure-insufficient placental perfusion-vascular endothelial injury".

Regarding model performance, the nomogram achieved C-indexes of 0.890 and 0.848 in the training and validation sets, respectively. The AUC exceeded 0.84 in both datasets, outperforming existing models based on single or limited combined indicators. This finding aligns with the decision curve analysis, which showed optimal net benefit when the threshold probability ranged between 0.10 and 0.95. Thus, the model effectively identifies high-risk patients and reduces both overtreatment and missed diagnoses, regardless of conservative or active intervention thresholds. The visual presentation of the nomogram simplifies risk assessment, enabling clinicians to directly obtain probabilities by summing patient-specific indicator scores. This straightforward approach avoids complex statistical calculations, making it particularly suitable for primary hospitals or emergency settings. The "quantitative-visual" transformation addresses the challenge of implementing traditional multi-factor models clinically. Compared with a recent predictive model that incorporated only 4 predictors and achieved an AUC of 0.81 in validation, our model integrates six multidimensional predictors, achieving a superior AUC of 0.848 [21]. In recent years, artificial intelligence (AI) and machine learning methods have increasingly been applied in placental abruption prediction. For instance, a machine learning model based on multiple clinical and imaging indicators achieved an AUC of 0.91 in training, but its complexity limited its widespread use in primary hospitals [22]. In contrast, our nomogram offers both strong predictive performance and clinical practicality, enhancing its suitability for broader implementation. Clinically, this model was validated among patients with severe preeclampsia at approximately 33 weeks' gestation (training set: 33.05 $\pm$ 2.14 weeks; validation set: 33.21 $\pm$ 2.45 weeks). Subgroup analysis revealed that the model maintained good predictive performance, with an AUC of 0.821 in patients $<$32 weeks' gestation and an AUC of 0.865 in patients $\geq$32 weeks' gestation. In addition, the AUC values were 0.852 in primiparas and 0.843 in multiparas, demonstrating stable predictive capability across parity groups. These findings support the model's applicability across diverse clinical scenarios.

Age, as an independent risk factor, can be explained by vascular aging. In this study, the mean age of patients with placental abruption was significantly higher than those without (30.25 vs. 27.85 years), with an odds ratio (OR) of 1.076, suggesting that each additional year of age increases the risk by 7.6%. This association may result from reduced vascular elasticity and degenerative changes in placental vessels in older pregnant women. Such changes im-

pair the ability of the placenta to regulate blood flow, increasing susceptibility to abruption under stress conditions like hypertension. Additionally, older pregnant women are more likely to have chronic conditions. Although this study excluded patients with preexisting chronic diseases, pregnancy itself may heighten vascular endothelial sensitivity to damage, indirectly increasing the risk of placental abruption [23].

An increase in baseline systolic and DBP significantly predicts placental abruption (OR = 1.052 and OR = 1.078, respectively). This relationship arises from their direct impact on placental hemodynamics. Severe preeclampsia involves systemic small-vessel spasms, already reducing placental perfusion. Elevated baseline blood pressure further exacerbates ischemia in the placental intervillous space, facilitating the formation of a retroplacental hematoma. Additionally, sudden increases in blood pressure may directly rupture vessels at the placental attachment, triggering abruption [24]. Notably, the OR of DBP exceeds that of SBP. This indicates that increased peripheral vascular resistance (reflected by diastolic pressure) exerts a greater influence on placental microcirculation, explaining the priority placed on controlling DBP in preeclampsia management [12].

The width of retroplacental hematoma, the sole imaging indicator, had an OR of 1.320, underscoring the essential role of imaging in predicting placental abruption. Retroplacental hematoma detected by ultrasound provides direct imaging evidence of placental abruption. A larger width indicates a broader abruption area and an increased risk of fresh bleeding [25]. In this study, hematoma width in the abruption group was significantly greater than in the non-abruption group (16.36 mm vs. 13.85 mm), confirming a quantitative relationship between hematoma size and abruption risk. Compared to other imaging measures (such as placental thickness), retroplacental hematoma width shows more dynamic changes, facilitating continuous ultrasound monitoring of disease progression and aiding the timely selection of interventions [26].

The combined use of PlGF and the sFlt-1/PlGF ratio, as molecular markers reflecting placental function and vascular endothelial status, represents the core innovation of this model. PlGF, an angiogenic factor secreted by placental trophoblast cells, directly indicates impaired trophoblast invasion and insufficient angiogenesis when its levels decline (OR = 0.982), leading to placental ischemia and hypoxia. Although PlGF is an independent risk factor, its relatively small effect size (OR = 0.982) suggests that each unit change has limited clinical impact on abruption risk and must be assessed in combination with other predictors. sFlt-1, a soluble receptor of PlGF, competitively inhibits the vascular protective effects of PlGF. An elevated sFlt-1/PlGF ratio (OR = 1.100) amplifies endothelial injury effects [27]. A study indicate this ratio better captures the imbalance between "angiogenesis and anti-angiogenesis" than individual indicators alone. In preeclampsia patients, this ratio may increase 4–6 weeks before clinical symptoms emerge, highlighting its early warning potential for placental abruption [11]. In addition, the ratio is less influenced by placental size and gestational age, providing more stable prediction performance than PlGF alone.

The core advantages of this study are evident in two main aspects. Firstly, it precisely integrates multidimensional indicators. Unlike previous studies focusing solely on biomarkers or imaging, this model systematically includes demographic characteristics, clinical signs, imaging indicators, and molecular markers. Thus, it establishes a comprehensive "clinical-imaging-molecular" framework, covering the entire pathological progression of placental abruption. Secondly, the model demonstrates substantial clinical translation value. The visually intuitive design and strong predictive performance (high C-index and AUC) enhance its practicality and scientific validity. Consequently, it provides clinicians with a directly applicable risk assessment tool, shifting placental abruption prediction from "empirical judgment" toward "data-driven decision-making".

Limitations

The limitations of this study were as follows: (1) Single-center sampling. All 260 patients in this study were from the same hospital. Although random grouping ensured homogeneity between the training and validation sets, regional population biases may still exist. For instance, the age distribution and incidence of underlying diseases among pregnant women in this region may differ from those in other areas. Therefore, the model may be more suitable for local populations and less applicable to regions with different demographic structures and lifestyle habits. Future multicenter studies should collect samples from various regions and hospital levels to validate and optimize the model. (2) Lack of external validation. Due to the short study period (January 2022 to October 2024), establishing data-sharing mechanisms with other centers was not feasible. Additionally, external validation requires strict adherence to inclusion and exclusion criteria, making it difficult to gather sufficient comparable data quickly. This significantly limits the generalizability of the model across different clinical settings. In future research, we plan to collaborate with multiple hospitals, collect more diverse samples, and conduct external validation to enhance the model's applicability. (3) Omission of potential indicators. This study did not include factors such as placental blood flow resistance indices (e.g., uterine artery PI) or coagulation indicators (e.g., prothrombin time), which may influence placental abruption. Expanding the indicator dimensions in future studies could improve the robustness of the model.

5. Conclusions

In conclusion, the nomogram developed in this study accurately predicts placental abruption risk in severe

preeclampsia by integrating multi-dimensional indicators. Its high predictive performance and clinical usability offer new perspectives for managing critical obstetric conditions. Future multicenter research should increase sample size, perform external validation, and investigate dynamic monitoring indicators (such as the rate of change in retroplacental hematoma width and the trend of sFlt-1/PlGF ratio) to further enhance the model's application across varied clinical scenarios.

Availability of Data and Materials

The data used or analyzed during the current study are available from the corresponding author on reasonable request.

Author Contributions

LS and YW conceived and designed the study; GH collected the data; LS and YW performed formal analysis and statistical analysis; YW provided resources and supervised the study; LS constructed the nomogram and visualized the data. All authors contributed to the critical revision of the manuscript for important intellectual content. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was approved by the Ethics Committee of Ezhou Central Hospital (No. EZCH0141), and informed consent was obtained from all patients. This study was conducted in accordance with the Declaration of Helsinki.

Acknowledgment

The authors wish to thank Dr. Lihuan Zhu for his valuable technical assistance in statistical analysis and data visualization.

Funding

This research received no external funding.

Conflict of Interest

The authors declare no conflict of interest.

References

[1] Allard M, Grosch S, Jouret F, Masson V, Surinder T, Masset C. Prevention of preeclampsia and its complications. Revue Medicale De Liege. 2024; 79: 448–454.

[2] Capriglione S, Gulino FA, Latella S, De Felice G, Filippini M, Farinelli M, *et al*. Results of a Five-Year Experience in First Trimester Preeclampsia Screening. Journal of Clinical Medicine. 2022; 11: 4555. https://doi.org/10.3390/jcm11154555.

[3] Duvekot JJ, Duijnhoven RG, van Horen E, Bax CJ, Bloemenkamp KW, Brussé IA, *et al*. Temporizing management vs immediate delivery in early-onset severe preeclampsia between 28 and 34 weeks of gestation (TOTEM study): An open-label randomized controlled trial. Acta Obstetricia et Gynecologica Scandinavica. 2021; 100: 109–118. https://doi.org/10.1111/aogs.13976.

[4] Fishel Bartal M, Sibai BM. Eclampsia in the 21st century. American Journal of Obstetrics and Gynecology. 2022; 226: S1237–S1253. https://doi.org/10.1016/j.ajog.2020.09.037.

[5] Gonen N, Levy M, Kovo M, Schreiber L, Noy LK, Volpert E, *et al*. Placental Histopathology and Pregnancy Outcomes in "Early" vs. "Late" Placental Abruption. Reproductive Sciences (Thousand Oaks, Calif.). 2021; 28: 351–360. https://doi.org/10.1007/s43032-020-00287-3.

[6] Mikuscheva A, Strassding F, MacKenzie E. Three Cases of Severe Placental Abruption as a First Symptom of Preeclampsia. Case Reports in Obstetrics and Gynecology. 2021; 2021: 3863607. https://doi.org/10.1155/2021/3863607.

[7] Larosa M, Le Guern V, Morel N, Belhocine M, Ruffatti A, Silva NM, *et al*. Evaluation of the severe preeclampsia classification criterion for antiphospholipid syndrome in a study of 40 patients. Arthritis Research & Therapy. 2021; 23: 134. https://doi.org/10.1186/s13075-021-02518-7.

[8] Li J, Li Y, Duan Y, Xiao X, Luo J, Luo M. Dose-response associations of maternal age with pregnancy complications and multimorbidity among nulliparas and multiparas: A multicentric retrospective cohort study in southern China. Journal of Global Health. 2023; 13: 04117. https://doi.org/10.7189/jogh.13.04117.

[9] Álvarez SC, Zurita AR, Del Carmen De Ganzo Suárez T, De Luis Escudero JF, Medina NS, Pérez CC, *et al*. Is a sFlt-1/PlGF cutoff of 38 suitable to predict adverse outcomes in pregnancies with abnormal uterine artery Doppler velocimetry in the second trimester? Pregnancy Hypertension. 2023; 34: 13–18. https://doi.org/10.1016/j.preghy.2023.09.006.

[10] Chen Y, Huang L, Zhang J, Guo N. Serum sFlt-1/PlGF ratio combined with ANXA2 correlates with adverse pregnancy outcomes in women with suspected preeclampsia. The Journal of Maternal-fetal & Neonatal Medicine: the Official Journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, the International Society of Perinatal Obstetricians. 2025; 38: 2506008. https://doi.org/10.1080/14767058.2025.2506008.

[11] Reddy M, Palmer K, Rolnik DL, Wallace EM, Mol BW, Da Silva Costa F. Role of placental, fetal and maternal cardiovascular markers in predicting adverse outcome in women with suspected or confirmed pre-eclampsia. Ultrasound in Obstetrics & Gynecology: the Official Journal of the International Society of Ultrasound in Obstetrics and Gynecology. 2022; 59: 596–605. https://doi.org/10.1002/uog.24851.

[12] Sanusi AA, Leach J, Boggess K, Dugoff L, Sibai B, Lawrence K, *et al*. Pregnancy Outcomes of Nifedipine Compared With Labetalol for Oral Treatment of Mild Chronic Hypertension. Obstetrics and Gynecology. 2024; 144: 126–134. https://doi.org/10.1097/AOG.0000000000005613.

[13] Espinoza J, Calsavara VF, Kilpatrick S, Rana S, Costantine MM, Boggess K, *et al*. Plasma soluble fms-like tyrosine kinase 1 to placental growth factor ratio of 11.5 multiples of median predicts preeclampsia with severe features within 2 weeks of testing. American Journal of Obstetrics and Gynecology. 2024; 231: 363.e1–363.e11. https://doi.org/10.1016/j.ajog.2024.05.050.

[14] Roberts DJ, Baergen RN, Boyd TK, Carreon CK, Duncan VE, Ernst LM, *et al*. Criteria for placental examination for obstetrical and neonatal providers. American Journal of Obstetrics and Gynecology. 2023; 228: 497–508.e4. https://doi.org/10.1016/j.ajog.2022.12.017.

[15] Overton E, Tobes D, Lee A. Preeclampsia diagnosis and management. Best Practice & Research. Clinical Anaesthesiology. 2022; 36: 107–121. https://doi.org/10.1016/j.bpa.2022.02.003.

[16] Reddy Eleti M, Agrawal M, Dewani D, Goyal N. Serum LDH Levels in Normotensive and Preeclamptic-Eclamptic Pregnant Women and Its Correlation With Fetomaternal Outcome. Cureus. 2023; 15: e37220. https://doi.org/10.7759/cureus.37220.

[17] Tita AT, Szychowski JM, Boggess K, Dugoff L, Sibai B, Lawrence K, *et al*. Treatment for Mild Chronic Hypertension during Pregnancy. The New England Journal of Medicine. 2022; 386: 1781–1792. https://doi.org/10.1056/NEJMoa2201295.

[18] Williams S, Tran C, Forseter S. Coinciding Placental Abruption and Pulmonary Edema in a Patient With Preeclampsia. Cureus. 2024; 16: e66065. https://doi.org/10.7759/cureus.66065.

[19] Kumar N, Das V, Agarwal A, Agrawal S. Correlation of sFlt/PlGF ratio with severity of preeclampsia in an Indian population. AJOG Global Reports. 2023; 3: 100177. https://doi.org/10.1016/j.xagr.2023.100177.

[20] Yang M, Wang M, Zhu Q, Li N. Development and validation of a risk prediction model for placental abruption in patients with preeclampsia. Placenta. 2025; 164: 1–9. https://doi.org/10.1016/j.placenta.2025.03.005.

[21] Amar S, Potter BJ, Paradis G, Lewin A, Maniraho A, Brousseau É, *et al*. Outcomes of Postpartum Preeclampsia: A Retrospective Cohort Study of 1.3 Million Pregnancies. BJOG: an International Journal of Obstetrics and Gynaecology. 2025; 132: 752–759. https://doi.org/10.1111/1471-0528.18030.

[22] Tzur Y, Rimon E, Geva G, Herzlich J, Kupferminc MJ. Progression from isolated gestational proteinuria to preeclampsia with severe features. Acta Obstetricia et Gynecologica Scandinavica. 2021; 100: 1620–1626. https://doi.org/10.1111/aogs.14198.

[23] Chen D, Gao X, Yang T, Xin X, Wang G, Wang H, *et al*. Independent risk factors for placental abruption: a systematic review and meta-analysis. BMC Pregnancy and Childbirth. 2025; 25: 351. https://doi.org/10.1186/s12884-025-07482-7.

[24] Harper LM, Kuo HC, Boggess K, Dugoff L, Sibai B, Lawrence K, *et al*. Blood pressure control in pregnant patients with chronic hypertension and diabetes: should <130/80 be the target? American Journal of Obstetrics and Gynecology. 2025; 232: 482.e1–482.e8. https://doi.org/10.1016/j.ajog.2024.09.006.

[25] Aplin JD, Myers JE, Timms K, Westwood M. Tracking placental development in health and disease. Nature Reviews. Endocrinology. 2020; 16: 479–494. https://doi.org/10.1038/s41574-020-0372-6.

[26] Vaupel P, Multhoff G. Blood Flow and Respiratory Gas Exchange in the Human Placenta at Term: A Data Update. Advances in Experimental Medicine and Biology. 2022; 1395: 379–384. https://doi.org/10.1007/978-3-031-14190-4_62.

[27] Leaños-Miranda A, Graciela Nolasco-Leaños A, Ismael Carrillo-Juárez R, José Molina-Pérez C, Janet Sillas-Pardo L, Manuel Jiménez-Trejo L, *et al*. Usefulness of the sFlt-1/PlGF (Soluble fms-Like Tyrosine Kinase-1/Placental Growth Factor) Ratio in Diagnosis or Misdiagnosis in Women With Clinical Diagnosis of Preeclampsia. Hypertension (Dallas, Tex.: 1979). 2020; 76: 892–900. https://doi.org/10.1161/HYPERTENSIONAHA.120.15552.