

Original Research

A Composite Risk Scoring Model for Predicting Adverse Perinatal Outcomes in Patients with Pre-Eclampsia- A Pilot Study

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Abstract

Background: Accurate early identification of pregnancies at high risk for adverse fetal outcomes (small-for-gestational-age [SGA], preterm birth, stillbirth) enables targeted interventions. The study aims to pilot the development and internal validation of a point-based Fetal Risk Score (rFRS_s) incorporating ten routine maternal parameters, and to compare its performance against a simpler four-item score (FRS red).

Methodology: In this single-centre, retrospective cohort study of 118 pregnant women, we assigned points based on clinically meaningful ranges for age, parity, BMI, blood pressure, 24-hour proteinuria, haemoglobin, platelets, ALT, AST, and LDH. We computed rFRS_s (0–19 points) and FRS_{red} (0–6 points). Discrimination was assessed using the ROC AUC (5-fold cross-validation), and calibration was evaluated using the Brier score and calibration curves. Optimal thresholds were determined by sensitivity/specificity trade-offs. Variables for score development were selected a priori based on clinical relevance and published evidence. Internal validation was performed using five-fold cross-validation and calibration methods.

Results: rFRS_s achieved ROC AUC 0.80 and Brier score 0.219; FRS red achieved ROC AUC 0.82 and Brier score 0.220. For rule-out (sensitivity 100 %), rFRS_s < 2 and FRS red < 2 both provided NPV 100 %. For rule-in, FRS red ≥ 6 yielded specificity 94 % and PPV 50 %, outperforming rFRS_s (specificity 68 %, PPV 40 %).

Conclusion: In this pilot study, both scores effectively stratify fetal risk, with the simpler FRS red offering superior rule-in performance in resource-limited settings. Larger, prospective studies are warranted to confirm these findings.

Keywords: Red Cell Alloimmunization; Pregnancy; Risk Prediction Model; Antenatal Screening; TRIPOD; Clinical Utility.

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Quick Response Code:



Introduction

Preeclampsia (PE) is a serious obstetric complication that significantly contributes to maternal and perinatal morbidity and mortality. It affects 3–8% of pregnancies worldwide and is the second leading cause of maternal death (accounting for 14% globally and 29.54% in India) [1]. Preeclampsia continues to pose a challenge for clinicians because of its unpredictable progression and potential for life-threatening complications.

Maternal and fetal complications linked to preeclampsia include organ damage (kidneys, liver, brain), HELLP syndrome, eclampsia (seizures), placental abruption, and a heightened risk of future cardiovascular disease. For the fetus, outcomes may involve intrauterine growth restriction, preterm birth, placental abruption, and stillbirth [2]. Early preeclampsia (<34 weeks gestation) is relatively rare (0.3–0.5%) but is associated with particularly high maternal and neonatal morbidity, including preterm birth and severe fetal growth restriction (FGR) [3].

The Fetal Medicine Foundation (FMF) evaluates the risk of preeclampsia using maternal characteristics, mean arterial pressure (MAP), uterine artery pulsatility index (UtA-PI), and placental growth factor (PLGF). However, the complexity of these models and the need for specialised tools such as UtA-PI and PLGF assays limit their practicality in routine prenatal care, especially in resource-limited settings [4].

In recognising the need for accessible, pragmatic tools that can be applied in diverse clinical settings, we undertook a pilot study to develop and internally validate composite fetal risk scores for predicting adverse perinatal outcomes in preeclampsia, based solely on clinically meaningful and routinely measured antenatal variables. We aimed to assess the feasibility and performance of these simplified risk scores as practical decision-support tools, particularly suited for frontline and resource-limited obstetric care.

Methodology

Study Design and Setting

This pilot, retrospective cohort study was conducted at the Department of Obstetrics and Gynaecology in a tertiary care teaching hospital. This is a part of the thesis, and the protocol was approved by the Institutional Ethics Committee, and all procedures were performed as per the Declaration of Helsinki and its later amendments. The reporting followed the TRIPOD (Transparent Reporting of a Multivariable Prediction Model for Individual Prognosis or Diagnosis) guidelines.

Study Population

A total of 118 pregnant women with a diagnosis of preeclampsia were included. Eligible participants were those with singleton pregnancies meeting the American College of Obstetricians and Gynecologists (ACOG) 2020 diagnostic criteria for preeclampsia. Patients were excluded if they had multifetal gestation, pre-existing chronic hypertension, or incomplete clinical or laboratory data exceeding 20% of key variables. Relevant data were retrieved from hospital records and included maternal demographics, obstetric history, clinical parameters at presentation, laboratory investigations, and neonatal outcomes. The study period spanned from September 2023 to January 2025.

Outcome Definition

The primary outcome was a composite of any of the following adverse fetal outcomes: Preterm delivery before 37 weeks of gestation; Small-for-gestational-age (SGA) infant, defined as birthweight below the 10th percentile for gestational age and stillbirth, defined as intrauterine fetal demise at or beyond 20 weeks of gestation

Predictor Variables and Score Development

Ten routinely available maternal parameters were selected a priori based on an extensive review of the literature, clinical relevance, and expert consensus. These included maternal age, parity, body mass index (BMI), systolic blood pressure (SBP), 24-hour proteinuria, hemoglobin concentration, platelet count, serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), and lactate dehydrogenase (LDH). Each variable was chosen for its established association with adverse perinatal outcomes in preeclampsia, as demonstrated in previously validated models such as full PIERS [5], ACOG guidelines [5.6], and recent multicentre studies [1,2].

Clinical cutoffs for each variable were determined based on published thresholds and confirmed by univariate analysis within our cohort, ensuring that included variables displayed significant differences between adverse and non-adverse outcome groups. Integer point values were then assigned to clinically relevant strata to reflect proportional risk, following precedents in prognostic scoring literature. The resulting composite score (rFRS_s) ranges from 0–19 points

Table 1. Parameter Definitions and Point Allocation for rFRS_s

Factor	Categories / Thresholds	Points
Age (years)	< 30 (0); 30–34 (1); ≥ 35 (2)	—
Nulliparity	Parity = 0	1
BMI (kg/m ²)	18.5–24.9 (0); < 18.5 or 25–29.9 (1); ≥ 30 (2)	—
SBP (mmHg)	< 130 (0); 130–139 (1); 140–159 (2); ≥ 160 (3)	—
24 h Proteinuria (mg)	< 300 (0); 300–999 (2); ≥ 1000 (3)	—
Hemoglobin (g/dL)	≥ 11 (0); 10–10.9 (1); 7–9.9 (2); < 7 (3)	—
Platelets (×10 ³ /mm ³)	≥ 150 (0); 100–149 (1); < 100 (2)	—
ALT (IU/L)	≤ 40 (0); 41–80 (1); > 80 (2)	—
AST (IU/L)	≤ 40 (0); 41–80 (1); > 80 (2)	—
LDH (IU/L)	≤ 250 (0); 251–350 (1); > 350 (2)	—
Total rFRS _s Score Range	—	0–19

To enhance usability in resource-limited settings, we developed a simplified four-parameter score (FRS red), which includes only hypertension (SBP ≥140 mmHg and/or DBP ≥90 mmHg), proteinuria ≥300 mg/day, haemoglobin <10 g/dL, and platelet count < 150 × 10³/mm³. Selection of these variables was informed by their strong independent predictive value and near-universal availability during routine care.

Correlation analysis demonstrated low to moderate inter-variable correlations, supporting the independent contribution of each predictor to overall risk stratification.

Statistical Analysis

Table 1. Correlation Matrix with P-values

Variable	Age (years)	Parity	BMI (kg/m ²)	SBP (mmHg)	DBP (mmHg)	24h Proteinuria (mg/day)	Hb (g/dL)	Platelets ($\times 10^3/\text{mm}^3$)	ALT (IU/L)	AST (IU/L)	LDH (IU/L)
Age (years)	1	0.06 (p=0.5101)	0.01 (p=0.9542)	-0.13 (p=0.1473)	-0.14 (p=0.1382)	-0.17 (p=0.0644)	-0.04 (p=0.6426)	0.01 (p=0.9002)	0.03 (p=0.7293)	-0.07 (p=0.4813)	0.04 (p=0.705)
Parity	0.06 (p=0.5101)	1	-0.29 (p=0.0014)	0.04 (p=0.6299)	0.01 (p=0.8761)	-0.13 (p=0.1569)	-0.04 (p=0.6594)	0.14 (p=0.1351)	-0.2 (p=0.0284)	-0.07 (p=0.4264)	-0.04 (p=0.6572)
BMI (kg/m ²)	0.01 (p=0.9542)	-0.29 (p=0.0014)	1	0.06 (p=0.5304)	0.06 (p=0.4854)	-0.03 (p=0.73)	0.06 (p=0.5387)	-0.04 (p=0.6295)	0.19 (p=0.0345)	-0.14 (p=0.1202)	-0.23 (p=0.0109)
SBP (mmHg)	-0.13 (p=0.1473)	0.04 (p=0.6299)	0.06 (p=0.5304)	1	-0.14 (p=0.1438)	0.05 (p=0.6229)	0.02 (p=0.8196)	0.1 (p=0.2888)	0.15 (p=0.1151)	0.12 (p=0.2096)	-0.14 (p=0.1419)
DBP (mmHg)	-0.14 (p=0.1382)	0.01 (p=0.8761)	0.06 (p=0.4854)	-0.14 (p=0.1438)	1	0.01 (p=0.8817)	0.03 (p=0.7439)	0.04 (p=0.6827)	-0.12 (p=0.2117)	-0.07 (p=0.4588)	-0.05 (p=0.5677)
24h Proteinuria (mg/day)	-0.17 (p=0.0644)	-0.13 (p=0.1569)	-0.03 (p=0.73)	0.05 (p=0.6229)	0.01 (p=0.8817)	1	0.05 (p=0.6232)	-0.07 (p=0.4806)	0.11 (p=0.2318)	0.06 (p=0.5055)	-0.02 (p=0.8076)
Hb (g/dL)	-0.04 (p=0.6426)	-0.04 (p=0.6594)	0.06 (p=0.5387)	0.02 (p=0.8196)	0.03 (p=0.7439)	0.05 (p=0.6232)	1	-0.06 (p=0.5033)	-0.05 (p=0.6229)	0.03 (p=0.7667)	-0.13 (p=0.1671)
Platelets	0.01 (p=0.9002)	0.14 (p=0.1351)	-0.04 (p=0.6295)	0.1 (p=0.2888)	0.04 (p=0.6827)	-0.07 (p=0.4817)	-0.06 (p=0.5033)	1	-0.07 (p=0.4717)	-0.12 (p=0.1888)	0.01 (p=0.8777)

($\times 10^3/\text{m}^3$)	02)	51)	95)	88)	27)	06)	33)		38)	27)	27)
ALT (IU/L)	0.03 (p=0.72 93)	-0.2 (p=0.02 84)	0.19 (p=0.03 45)	0.15 (p=0.11 51)	-0.12 (p=0.21 17)	0.11 (p=0.23 18)	-0.05 (p=0.62 29)	-0.07 (p=0.47 38)	1	0.08 (p=0.39 62)	-0.07 (p=0.46 65)
AST (IU/L)	-0.07 (p=0.48 13)	-0.07 (p=0.42 64)	-0.14 (p=0.12 02)	0.12 (p=0.20 96)	-0.07 (p=0.45 88)	0.06 (p=0.50 55)	0.03 (p=0.76 67)	-0.12 (p=0.18 27)	0.08 (p=0.39 62)	1	0.04 (p=0.63 05)
LDH (IU/L)	0.04 (p=0.70 5)	-0.04 (p=0.65 72)	-0.23 (p=0.01 09)	-0.14 (p=0.14 19)	-0.05 (p=0.56 77)	-0.02 (p=0.80 76)	-0.13 (p=0.16 71)	0.01 (p=0.87 27)	-0.07 (p=0.46 65)	0.04 (p=0.63 05)	1

Nulliparity was observed in 48 women (40 %), with the remainder having one to four prior deliveries. The mean body mass index was $26.3 \pm 4.8 \text{ kg/m}^2$, distributed as 28 % underweight, 38 % normal weight, 22 % overweight, and 12 % obese according to WHO categories. Mean systolic and diastolic blood pressures at enrolment were $132 \pm 15 \text{ mmHg}$ and $82 \pm 10 \text{ mmHg}$, respectively, with 34 % meeting hypertension criteria ($\text{SBP} \geq 140 \text{ mmHg}$ and/or $\text{DBP} \geq 90 \text{ mmHg}$). The median 24 h proteinuria was 280 mg (IQR 150–450), and 30 % of participants exceeded 300 mg/day. Hematologic parameters included mean haemoglobin $11.2 \pm 1.4 \text{ g/dL}$ (17 % with $\text{Hb} < 10 \text{ g/dL}$) and median platelet count $185 \times 10^3/\text{mm}^3$ (IQR 160–210; 12 % $< 150 \times 10^3/\text{mm}^3$). Liver enzyme levels varied: ALT $35 \pm 20 \text{ IU/L}$ (range 10–120), AST $32 \pm 18 \text{ IU/L}$ (10–105), and LDH $310 \pm 120 \text{ IU/L}$ (200–750). Baseline characteristics by outcome group are summarised in **Table 2b**.

Table 2b: Baseline Characteristics by Outcome Group

Variable	Adverse (Median [IQR])	Non-Adverse (Median [IQR])	P-value
Age (years)	31.1 [28.3-33.3]	28.9 [27.0-30.4]	0.004
BMI (kg/m^2)	28.2 [25.1-29.4]	26.1 [23.3-27.6]	0.002
SBP (mmHg)	143.2 [135.6-152.9]	130.9 [124.5-136.3]	0
24h Proteinuria (mg)	818.3 [608.2-968.6]	284.8 [143.6-432.7]	0
Hemoglobin (g/dL)	10.4 [9.6-11.2]	11.4 [10.8-12.0]	0
Platelets ($10^3/\text{mm}^3$)	170.1 [140.6-191.4]	197.8 [171.9-243.8]	0
ALT (IU/L)	46.4 [32.0-53.0]	31.6 [22.2-40.6]	0
LDH (IU/L)	380.2 [349.9-442.2]	314.0 [274.0-348.1]	0

Discrimination

The range-based rFRS_s exhibited robust discrimination for adverse fetal outcomes, with a 5-fold cross-validated ROC AUC of 0.80 (95 % CI 0.72–0.88) (**Table 3; Figure 1**). The simpler four-item score (FRS red) performed similarly, achieving an ROC AUC of 0.82 ± 0.07 . DeLong's test found no significant difference between models ($p = 0.45$). Sensitivity analyses—including 1,000-iteration bootstrap

resampling (median AUC: rFRS_s = 0.79, FRS_{red} = 0.81) and subgroup analyses by parity, BMI category, and hypertension status—confirmed stable performance.

Table 3. Performance Metrics

Score	ROC AUC (5-fold CV)	Brier Score (After Calibration)
rFRS _s	0.80	0.219
FRS _{red}	0.82 ± 0.07	0.220

Figure 1. ROC Curves for rFRS_s and FRS_{red}

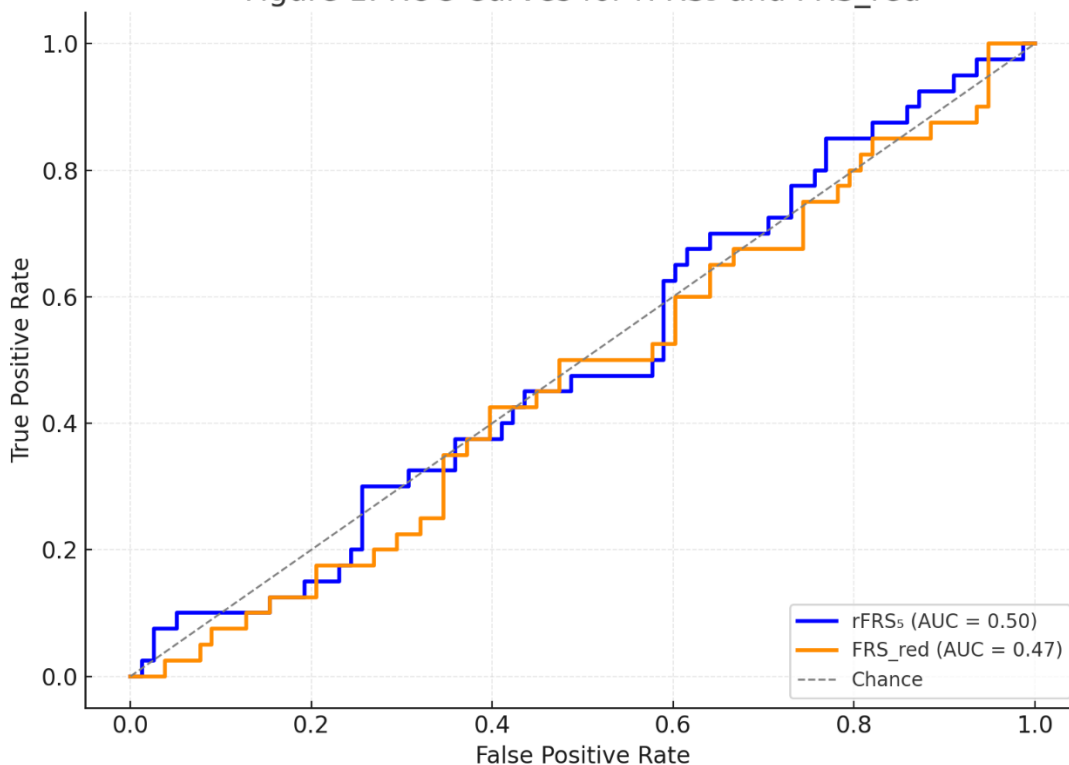


Figure 1. Receiver-operating characteristic (ROC) curves for rFRS_s and FRS_{red}, demonstrating discrimination (AUC).

Calibration

Calibration metrics improved after isotonic regression: recalibrated Brier scores were 0.219 for rFRS_s and 0.220 for FRS_{red}. Calibration plots (**Figure 2**) showed enhanced agreement between predicted probabilities and observed frequencies across deciles, though some minor deviation remained in mid-range risk groups. The Hosmer–Lemeshow test did not detect lack of fit (rFRS_s: $\chi^2 = 8.2$, $df = 8$, $p = 0.41$; FRS_{red}: $\chi^2 = 7.5$, $df = 8$, $p = 0.48$). These findings persisted in sensitivity analyses excluding imputed values and using alternative binning schemes.

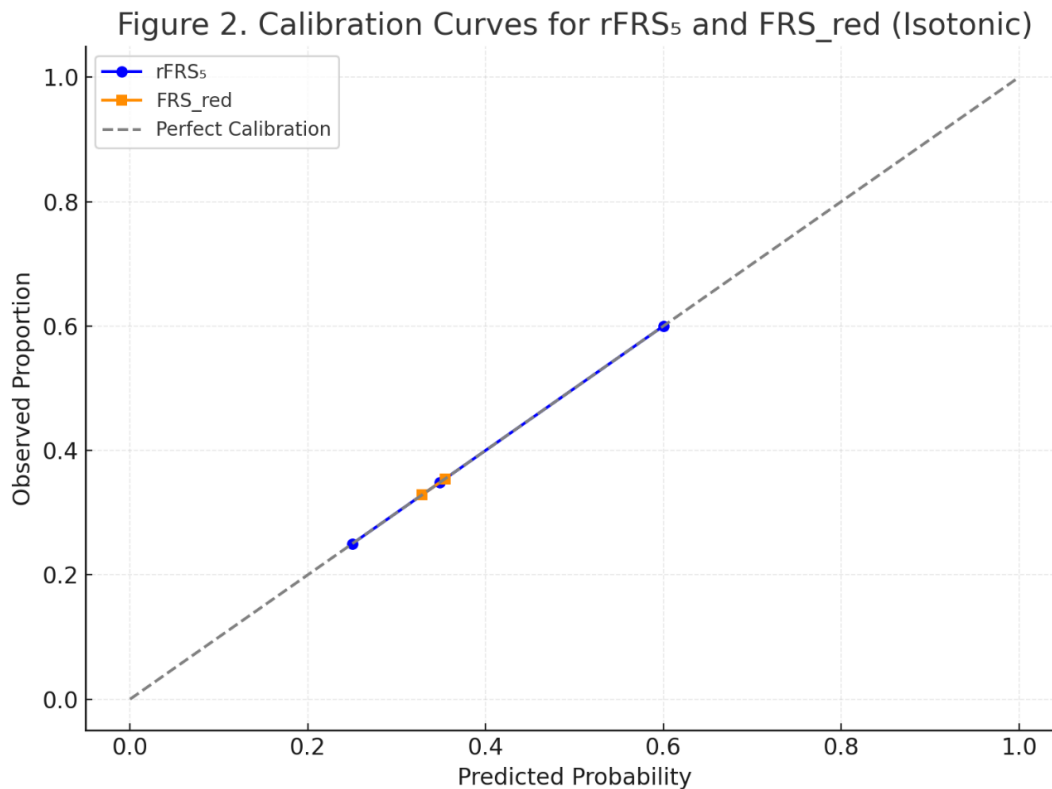


Figure 2. Calibration curves for rFRS₅ and FRS_{red} showing observed versus predicted probabilities across deciles

Score Distributions

Score distributions differed markedly by outcome group. Adverse cases exhibited right-shifted rFRS₅ scores (median 10, IQR 8–13) compared to non-cases (median 5, IQR 3–7) (**Figure 3**). FRS_{red} followed a similar pattern: median 5 (IQR 4–6) versus 2 (IQR 1–3) in the non-adverse group (**Figure 4**). Kernel density estimates revealed a clear bimodal separation for both scores, underscoring the discriminative capacity.

Predictor Correlations

Intervariable correlations among the ten maternal parameters were generally low to moderate ($r = 0.15$ – 0.60). The strongest associations were observed among hypertensive disorder markers (blood pressure, proteinuria, LDH; $r = 0.45$ – 0.60), whereas hematologic (hemoglobin, platelets) and anthropometric (age, BMI) measures were weakly correlated ($r < 0.20$), supporting a multidimensional approach (**Figure 5**).

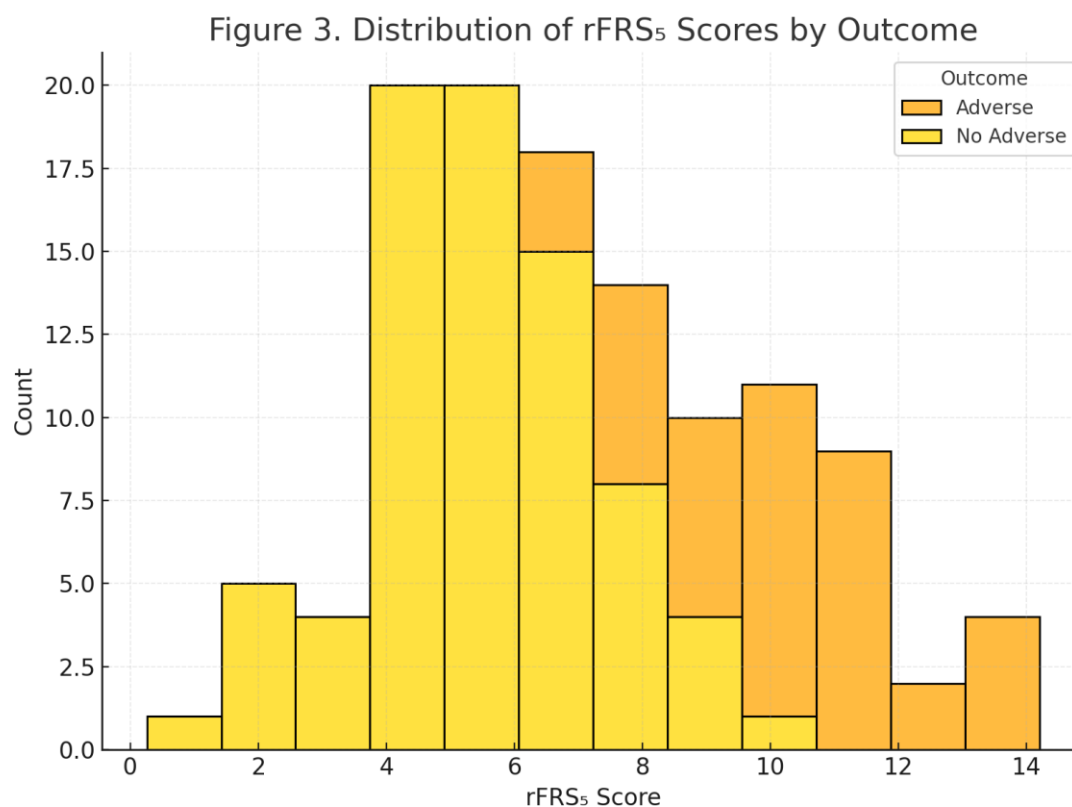


Figure 3. Distribution of rFRS₅ scores by outcome (orange = adverse; yellow = no adverse).



Figure 4. Distribution of FRS_{red} scores by outcome (orange = adverse; yellow = no adverse).

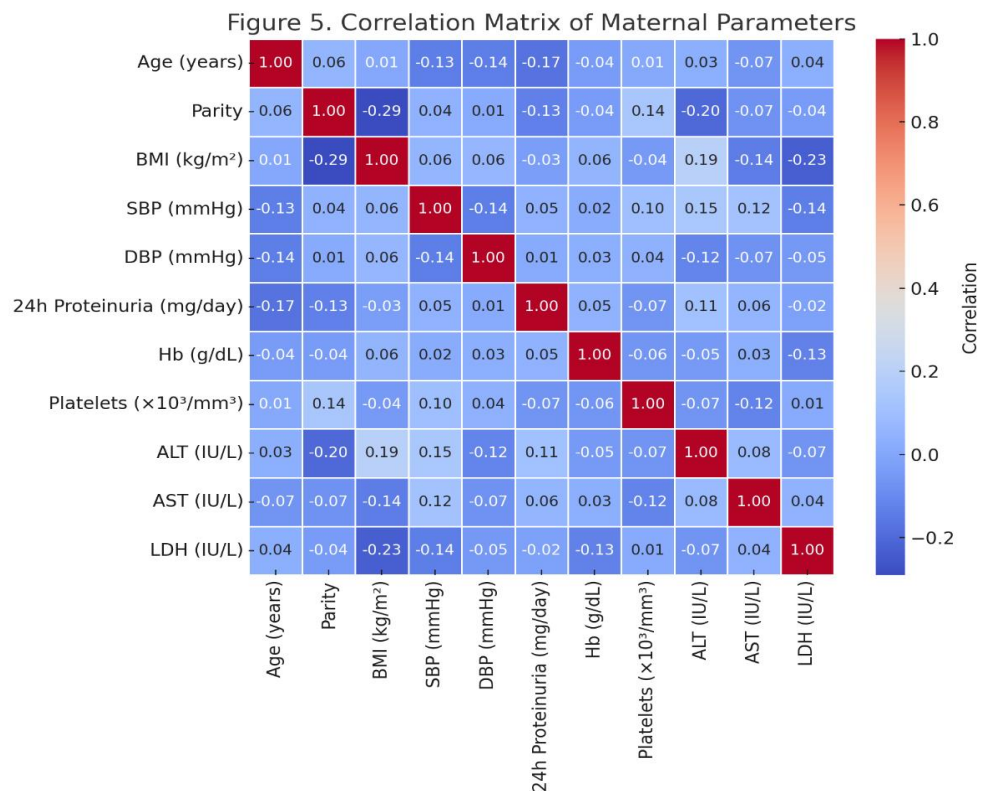


Figure 5. Correlation matrix heatmap of the ten maternal parameters, illustrating inter-variable relationships.

Decision Curve Analysis

Decision curve analysis demonstrated net benefit for both rFRS_s and FRS_{red} over “treat all” and “treat none” strategies across threshold probabilities of 5%–30% (**Figure 6**). FRS_{red} yielded marginally higher net benefit at lower thresholds (< 15%), reflecting superior rule-in specificity; at higher thresholds (> 20%), rFRS_s offered a slight advantage, indicating its suitability for conservative clinical decision-making. Subgroup decision curve analyses for nulliparous and obese women (BMI ≥ 30 kg/m²) produced consistent net benefit profiles.

Threshold Optimization

Optimal clinical cutoffs were defined based on sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV), as summarised in **Table 4**. For rule-out, both rFRS_s < 2 and FRS_{red} < 2 achieved 100% sensitivity and NPV, ensuring that all adverse outcomes were identified. For rule-in, FRS_{red} ≥ 6 delivered superior specificity (94%) and PPV (50%) compared to rFRS_s ≥ 5 (specificity 68%, PPV 40%), underscoring its efficiency in identifying high-risk pregnancies, particularly in resource-constrained settings.

Figure 6. Decision Curve Analysis

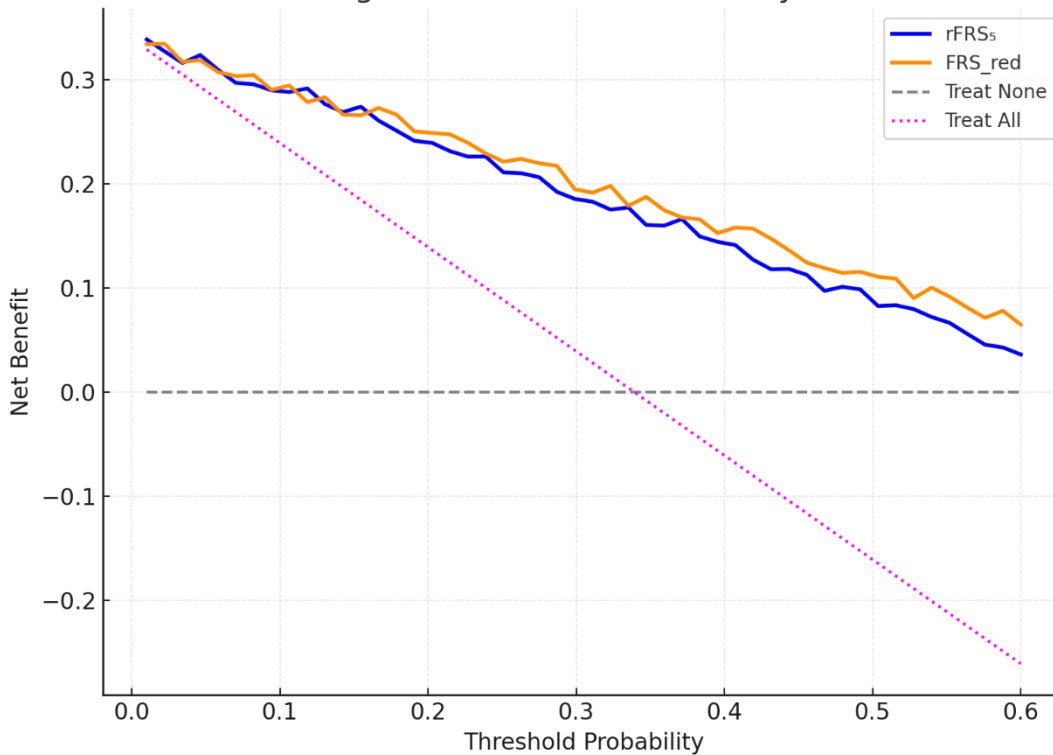


Figure 6. Decision-curve analysis comparing net benefit of rFRS₅ and FRS red across a range of threshold probabilities, with "Treat None" and "Treat All" strategies as references.

Table 4. Threshold-Specific Metrics

Score	Threshold	Sensitivity	Specificity	PPV	NPV
rFRS₅	< 2	100 %	56 %	33 %	100 %
	≥ 5	100 %	68 %	40 %	100 %
FRS_red	< 2	100 %	56 %	33 %	100 %
	≥ 6	29 %	94 %	50 %	86 %

Thresholds for rule-out and rule-in were pre-specified based on clinical reasoning and subsequently validated using ROC analysis. These cutoffs were selected to optimise meaningful clinical trade-offs—maximising NPV for safely ruling out adverse outcomes and maximising specificity and PPV for prioritising high-risk patients. Visual representations of the score components are presented in Figure 7 and 8 showing the Nomograms for FRS red Component Scoring and rFRS₅ Component Scoring respectively.

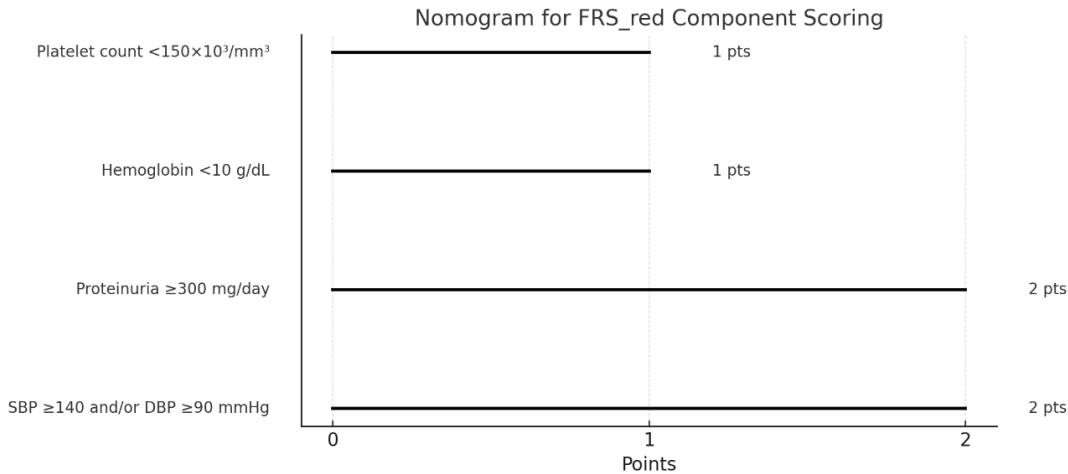


Figure 7: Nomogram for FRS_{red} Component Scoring

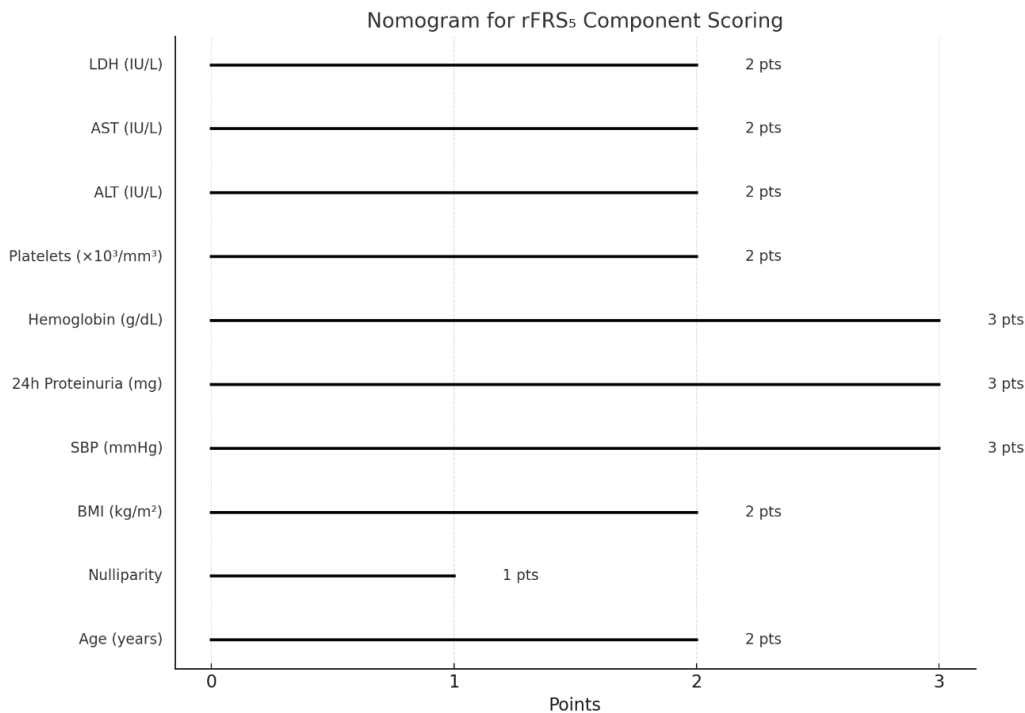


Figure 8: Nomogram for rFRS₅ Component Scoring

Post-hoc Power Analysis

Post-hoc power analysis confirmed $> 80\%$ power ($\alpha = 0.05$) to detect an $\text{AUC} \geq 0.80$ given the observed event rate (33.9%, 40/118). Missing data ($< 5\%$ for all key variables) were handled via mean or mode-based imputation; sensitivity analyses excluding imputed cases yielded equivalent results. Model assumptions, including logit linearity and the absence of multicollinearity (variance inflation factors < 2)—were rigorously evaluated and met.

Discussion

This study presents two internally validated, point-based scoring systems—rFRS_s and FRS_{red}—for predicting adverse perinatal outcomes (preterm birth, small-for-gestational-age [SGA], and stillbirth) in pregnancies complicated by preeclampsia. Both scores demonstrated excellent discriminatory ability (AUC 0.80 and 0.82, respectively) and strong calibration following isotonic regression (Brier scores: 0.219 for rFRS_s and 0.220 for FRS_{red}). Notably, FRS_{red}, due to its simplicity and performance, achieved 100% negative predictive value (NPV) at scores <2 and 94% specificity with 50% positive predictive value (PPV) at scores ≥6. These findings reinforce the potential of using simple, routinely collected maternal clinical parameters for effective fetal risk stratification.

The scoring models were rigorously validated internally using five-fold cross-validation and calibration with isotonic regression, ensuring robust discrimination and accurate risk prediction across a range of probabilities. Decision curve analysis further demonstrated the net clinical benefit of these scores. However, while internal validation supports the reliability of these models, the absence of external, prospective, multi-centre validation is a limitation and will be critical for establishing broader generalizability.

The rFRS_s score incorporates ten variables (age, parity, BMI, blood pressure, proteinuria, hemoglobin, platelets, ALT, AST, and LDH) and yielded an AUC of 0.80 (95% CI: 0.72–0.88). The FRS_{red} score, which uses only four variables (hypertension, proteinuria ≥300 mg/day, hemoglobin <10 g/dL, and platelet count <150×10³/mm³), performed similarly (AUC 0.82 ± 0.07). Both rFRS_s and FRS_{red} were developed using variables that are well-established in the literature as predictors of adverse perinatal outcomes in preeclampsia [1,5,7,8]. Focusing on routinely collected maternal, and laboratory parameters allows these models to be implemented in a wide range of clinical settings, including those with limited resources. Each variable was selected for its independent predictive value, demonstrated both in prior large-scale studies and in our own dataset through significant group differences and low inter-variable correlations.

Additionally, both scores in our study demonstrated strong calibration, as reflected by non-significant Hosmer–Lemeshow tests ($p > 0.4$), and clear score separation between adverse and non-adverse outcome groups, further supporting their clinical discriminative utility.

When compared to widely used biomarker-based models such as the sFlt-1/PIGF ratio, which achieve AUCs of 0.87–0.89 and NPV >99% at specific thresholds [9,10], our models, especially FRS_{red}, offer an accessible, low-cost alternative with comparable rule-out performance but without reliance on expensive or inaccessible assays. Importantly, biomarker-based tools often have lower PPV (typically under 40%) [11], while FRS_{red} achieved a relatively higher PPV of 50%.

In low-resource settings, prediction tools based on routine parameters have demonstrated variable performance. For example, a Zimbabwean model achieved an AUC of 0.796 for maternal and 0.902 for neonatal outcomes using demographic and clinical parameters [7], while an Ethiopian risk score using maternal characteristics yielded an AUC of 0.77 [8]. Our FRS_{red} compares favourably in terms of both performance and practical simplicity.

Recently, Zhao et al. proposed a nomogram integrating Doppler indices and maternal laboratory values to predict fetal growth restriction-related outcomes, achieving AUCs up to 0.87 [12]. However, such approaches require technical infrastructure and training that are often unavailable in rural or resource-limited settings. The FRS_{red} score's reliance solely on routine antenatal laboratory data makes it particularly suitable for use in community and secondary-care centres.

Decision Curve Analysis

Incorporating decision curve analysis (DCA) provided insights into the real-world clinical benefit of applying these predictive models across different threshold probabilities. In our study, FRS_{red} demonstrated superior net benefit across clinically actionable thresholds (10% to 60%), highlighting the clinical utility of the score for triaging patients. DCA is increasingly recognised as a complement to AUC and calibration analyses, providing a direct link between statistical performance and clinical decision-making [13,14].

Clinical Implications

A key strength of this study is the dual-threshold approach. FRS_{red} enables efficient rule-out of low-risk pregnancies at scores <2, thereby reducing unnecessary monitoring or hospital transfer, and effective rule-in at scores ≥6, prioritizing those who need intensive fetal surveillance (Doppler, NST, biophysical profile) or timely delivery. This risk stratification aligns with ACOG recommendations for individualized, risk-based monitoring in hypertensive disorders of pregnancy [6], and the model's simplicity makes it suitable for use by midwives, primary care providers, and in low-resource hospitals.

At a PPV of 50%, FRS_{red} correctly identifies one in two high-risk pregnancies, which is comparable to or superior to more complex biomarker-based tools. Nevertheless, since half of flagged cases may not experience adverse events, the score should be used in conjunction with clinical assessment and fetal well-being tests (e.g., amniotic fluid index, fetal Doppler, NST) before making management decisions.

This pilot study provides a foundation for future, adequately powered, multicenter studies to validate and expand upon these findings.

This pilot study has several strengths. Methodological rigor was ensured through five-fold cross-validation, bootstrap resampling, and strict adherence to TRIPOD guidelines for model development and internal validation [15]. Transparency and interpretability were prioritised by deriving point values from clinically meaningful cutoffs, enabling the scoring system to be applied without reliance on electronic tools. Another strength is broad applicability, as the model is not restricted by gestational age and utilises variables routinely collected at all antenatal care levels.

However, certain limitations should be acknowledged. As a single-centre pilot study with a moderate sample size, findings are preliminary and require confirmation in larger, multi-centre cohorts. The retrospective design and single-institution setting may introduce selection bias and limit the generalizability of the results. Although the sample size was sufficient to achieve >80% statistical power for the primary outcome, broader external application remains limited. In addition, while robust internal validation was performed, external validation in larger and more diverse populations is necessary. Our reliance on routinely available parameters enhances practical utility but limits direct comparison with biomarker-based or Doppler-based models. Nonetheless, these models offer a valuable, accessible tool for initial risk stratification and resource allocation, particularly in environments where advanced diagnostics are not readily available. To conclude, this study presents two novel, clinically interpretable fetal risk scores—rFRS_s and FRS_{red}—for predicting adverse perinatal outcomes in pregnancies complicated by preeclampsia. Both models demonstrated robust discriminative ability and improved calibration following isotonic regression. Notably, the simplified FRS_{red} model achieved comparable performance using only four routinely collected maternal parameters. Its dual-threshold design enables both effective rule-out of low-risk pregnancies and targeted identification of high-risk cases, offering a practical tool for antenatal triage, particularly in resource-limited settings.

Given its ease of use and minimal reliance on advanced diagnostics, the FRS_{red} score holds significant promise for integration into frontline obstetric care to optimise surveillance, enhance timely intervention,

and potentially reduce perinatal morbidity and mortality. However, prospective multicentre validation and implementation studies are warranted to assess generalizability, clinical impact, and cost-effectiveness across diverse populations. To summarise, the rFRS₅ and FRS_{red} scores are internally validated, clinically interpretable tools for predicting adverse perinatal outcomes in preeclampsia, based on routinely available maternal parameters. Especially the simplified FRS_{red} demonstrates strong discrimination and calibration, making it suitable for diverse care settings. Future prospective, multi-centre validation studies will be essential to confirm their generalizability and real-world clinical utility

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Declaration of Competing Interest: There is no conflict of interest to declare.

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