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ORIGINAL RESEARCH

External validation of the fullPIERS model for predicting adverse maternal outcomes among Swedish women with preeclampsia

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Abstract

Introduction: Preeclampsia affects 3%–4% of all pregnancies in the Nordic countries. It is a leading cause of maternal morbidity and mortality. Using risk stratification for women diagnosed with preeclampsia could potentially reduce adverse maternal outcomes. The fullPIERS model is a clinical prediction tool that has not been validated in the Nordic countries. We therefore aimed to externally validate the fullPIERS model among Swedish women with preeclampsia.

Material and Methods: Data were obtained from two Swedish prospective cohort studies. Predictor variables were assessed within 24 h of inclusion. Outcomes were assessed at 48 h and 7 days after inclusion. The performance of the fullPIERS model was evaluated by discrimination using the area under the receiver operating characteristic curve (AUC) and by risk stratification at prespecified risk cutoffs. Analyses were conducted with and without hematological outcomes (blood transfusion or severe thrombocytopenia).

Results: In total, 617 women were enrolled between May 2019 and March 2025. Adverse maternal outcomes occurred in 34 (5.5%) women within 48 h and 68 (11.0%) within 7 days. The fullPIERS model demonstrated modest discrimination for predicting adverse maternal outcomes within 48 h (AUC 0.64; 95% confidence interval [CI] 0.54, 0.74) and within 7 days (AUC 0.64; 95% CI: 0.57, 0.71). When analyses were restricted to nonhematological outcomes ($n = 14$ for 48 h and 19 for 7 days), the AUC was 0.79 (95% CI: 0.68, 0.89) within 48 h and 0.78 (95% CI: 0.69, 0.87) within 7 days. At a low-risk threshold of <0.01, 297 (48.1%) women were classified as low risk, of

Abbreviations: ALT, alanine transaminase concentration; AST, aspartate transaminase concentration; AUC, area under the curve; CI, confidence Interval; fullPIERS, preeclampsia integrated estimate of RiSk; GoPROVE, Gothenburg Preeclampsia Obstetric Adverse Events; HELLP, hemolysis, elevated liver enzymes, and low platelet count; NPV, negative predictive value; PPV, positive predictive value; ROC, receiver operating characteristic; UPMOST, Uppsala Pregnancy Complication Study.

Linnea Dahlberg and Tove Sandberg contributed equally to this work.

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whom one developed a nonhematological adverse outcome within 48 h, corresponding to a sensitivity of 0.93 (95% CI: 0.69–0.99) and a negative predictive value of 1.00 (95% CI: 0.98–1.00).

Conclusions: Overall, the fullPIERS model demonstrated poor clinical utility in identifying low-risk and high-risk individuals for adverse maternal outcomes among Swedish women with preeclampsia. When hematological outcomes were excluded, the model improved its ability to identify women without adverse maternal outcomes. The model may have clinical utility facilitating physicians in identifying those who could be managed with a lower level of surveillance.

KEYWORDS
adverse maternal events, fullPIERS model, hypertension, prediction, preeclampsia, pregnancy complications

1 | INTRODUCTION

Preeclampsia is a progressive disorder of pregnancy characterized by hypertension and end organ dysfunction. It affects 3%–4% of all pregnancies in Nordic countries.[1,2] Complications of preeclampsia are among the leading causes of maternal and perinatal morbidity and mortality.[1] These include maternal death, eclampsia, pulmonary edema, heart failure, renal failure, liver hematoma, and disseminated intravascular coagulation.[3] Besides delivery, there is currently no curative treatment for preeclampsia.[1] In high-income countries, such as Sweden, most women diagnosed with preeclampsia are well monitored and experience fewer complications than in many other countries.[1,4] However, complications that do occur are challenging to predict.

Earlier detection and risk stratification could potentially reduce adverse maternal outcomes in preeclampsia. The only multivariable prediction model for adverse maternal outcomes in preeclampsia that has been externally validated with moderate to good discrimination across different populations is the Preeclampsia Integrated Estimate of RiSk (fullPIERS) model.[5] The fullPIERS model was developed by von Dadelszen et al.[6] to identify women with preeclampsia at highest risk of developing adverse maternal outcomes. The model is based on simple clinical variables used in routine care.[7] While the fullPIERS model has been externally validated in both high- and low- and middle-income countries, it has not yet been validated in a Nordic setting.[8–22] In addition, the performance has varied, requiring population-specific validation.[5] Therefore, we set out to externally validate the fullPIERS model in a Swedish population of women with a confirmed diagnosis of preeclampsia.

Key message

There is a clinical need for risk stratification of adverse maternal outcomes in preeclampsia. In this Swedish validation study, the fullPIERS model showed poor clinical utility, but performance improved, in particular as a rule-out test, when hematological outcomes were excluded.

2 | MATERIAL AND METHODS

2.1 | Study design and population

Data were retrieved from two Swedish prospective cohort studies: the Gothenburg Preeclampsia Obstetric Adverse Events (GoPROVE) study and the Uppsala Pregnancy Complication Study (UPMOST).[23] These studies included women ≥ 18 years of age with a confirmed diagnosis of preeclampsia who received care at Sahlgrenska University Hospital in Gothenburg, Uppsala University Hospital in Uppsala, or Södra Älvsborgs Hospital in Borås.

The inclusion criteria were based on the protocols for GoPROVE and UPMOST. Preeclampsia was defined as new onset hypertension (≥ 140/90 mm Hg) after 20 weeks of gestation, together with significant proteinuria (defined by albumin/creatinine ratio ≥ 8 mg/mmol or protein/creatinine ratio ≥ 30 mg/mmol) or superimposed preeclampsia (chronic hypertension together with significant proteinuria ≥ 20 weeks of gestation).[4,23] This definition is consistent with the definition used in the development cohort for the fullPIERS model. The development cohort for the fullPIERS model also included women with HELLP syndrome without hypertension or proteinuria.[7]

Excluding criteria were preexisting renal or cerebral disease or inability to provide informed consent.[23] For the present analysis, women with at least one adverse maternal outcome at inclusion, or missing predictor data for chest pain and/or dyspnea, were excluded.

Outcome and predictor data were collected through medical charts and questionnaires. All data collection was aligned between sites. Detailed information on data collection is described in Supporting Information Table S1.

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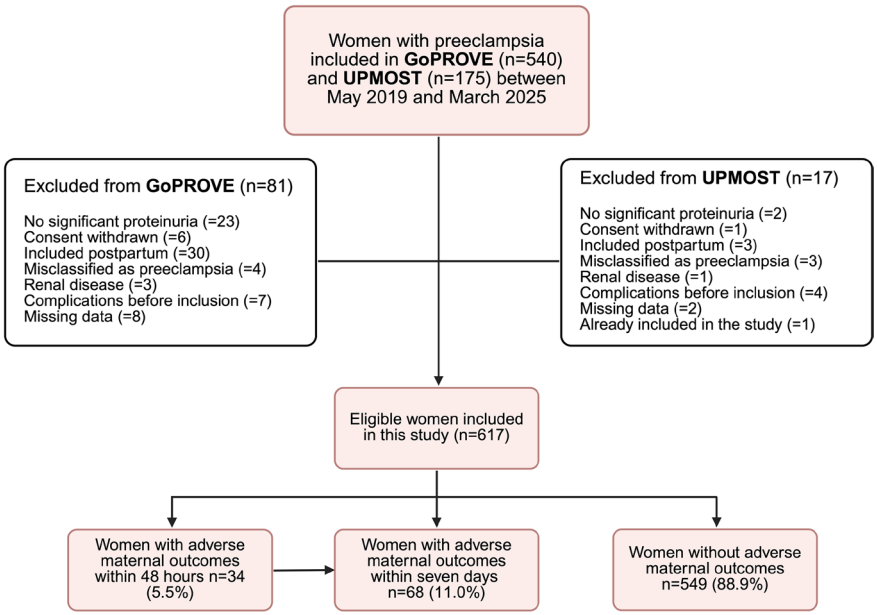

FIGURE 1 Flowchart of the study population selection process. Significant proteinuria is defined by albumin/creatinine ratio ≥ 8 mg/mmol or protein/creatinine-ratio ≥ 30 mg/mmol. Missing data reflect for no blood samples, missing outcome data, or missing chest pain and/or dyspnea. GoPROVE had missing data on blood samples ($n = 3$), outcomes ($n = 4$), and chest pain and/or dyspnea ($n = 1$). UPMOST had missing data on outcomes ($n = 1$) and chest pain and/or dyspnea ($n = 1$). Created in BioRender. Herrock, O. (2026) https://BioRender.com/2m974zm.

2.2 | Outcomes

The primary outcome was a binary composite of adverse maternal outcomes defined according to the original study on the fullPIERS model.[7] Ascites was the only outcome not included as it was not routinely recorded in the GoPROVE and UPMOST databases. Adverse maternal outcomes were further categorized into the following groups: maternal mortality, central nervous system, cardiorespiratory, hematological, hepatic, renal, and other adverse events. Outcomes were evaluated at two timepoints: within 48 h and within 7 days of inclusion. Events occurring within 48 h were also included in the 7-day assessment. Considering that blood transfusion is not a specific complication to preeclampsia, we also evaluated a secondary composite outcome, without hematological events (transfusion of any blood product and platelet count $< 50 \times 10^9$/L with no transfusion). Complete definitions of individual outcomes are provided in Supporting Information Table S1.

2.3 | Predictors

Predictor variables were primarily defined according to the original publication describing the fullPIERS model.[7] Predictors included gestational age, oxygen saturation, chest pain and/or dyspnea, platelet count, creatinine, and aspartate transaminase concentration (AST). Due to clinical practice and the structure of the GoPROVE and UPMOST definitions, some predictors were adapted accordingly, representing predefined deviations from the original fullPIERS model.[23] In this study, alanine transaminase (ALT) was predominantly collected in accordance with Swedish clinical practice and converted to AST. All samples were collected within 24 h of inclusion. However, if multiple values were recorded, the value closest to the time of inclusion was used in the analyses. Complete definitions of predictors are provided in Supporting Information Table S1.

2.4 | Sample size calculations

A total sample size of 600 women was targeted based on primary requirement of a ±0.10 margin of error to ensure adequate precision of the discriminative performance of the fullPIERS model for adverse maternal outcomes within 48 h and within 7 days after inclusion. For the 48-h outcome, assuming an event rate of 5% and an anticipated area under the receiver operating characteristic curve (AUC) of 0.70 or higher, a minimum of 547 participants was required to achieve a margin of error of ±0.10 around the estimated AUC. For the 7-day outcome, assuming an event rate of 10% and the same anticipated AUC, 589 participants were required to achieve a margin of error of ±0.07.[24] Sample size calculations were performed using the pmvalsampsize package (version 0.1.0) in R (R Foundation for Statistical Computing, Vienna, Austria).

2.5 | Statistical methods

Descriptive statistics were summarized as mean (SD) or median (IQR) for continuous variables and as counts (n) and percentages for categorical variables. Univariable logistic regression analyses were performed to evaluate associations between individual predictors and adverse maternal outcomes within 48 h and 7 days of study inclusion. Right-skewed continuous variables (platelet count, creatinine, ALT, and AST) were log-transformed, and odds ratios (ORs) were expressed per 33% decrease in platelet count and per 50% increase in creatinine and liver transaminases. In these analyses, participants with missing data were excluded (available-case analysis).

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TABLE 1 Baseline characteristics of the study cohort, overall and stratified by development of adverse maternal outcomes.

Characteristic	Total study population ($n=617$)	Any adverse maternal outcome	
		Yes ($n=68$)	No ($n=549$)
Demographics			
Age at diagnosis (years), mean (SD)	31.5 (4.9)	31.0 (5.2)	31.6 (4.8)
BMI prior to pregnancy (kg/m^2), mean (SD)	27.2 (5.9)	26.5 (6.0)	27.2 (5.9)
Ethnicity			
Black African	26 (4.2%)	4 (5.9%)	22 (4.0%)
East/central Asian	14 (2.3%)	0 (0.0%)	14 (2.6%)
Hispanic	3 (0.5%)	0 (0.0%)	3 (0.5%)
South Asian	19 (3.1%)	2 (2.9%)	17 (3.1%)
White	512 (83.0%)	55 (80.9%)	457 (83.2%)
Not specified	12 (1.9%)	2 (2.9%)	10 (1.8%)
Other	31 (5.0%)	5 (7.4%)	26 (4.7%)
Total years of full-time education, median (IQR)	15.0 (13.0–17.0)	15.0 (12.0–16.8)	15.0 (13.0–17.0)
Gestational age at inclusion < 34 weeks	139 (22.5%)	12 (17.6%)	127 (23.1%)
Obstetric history			
Nulliparous	473 (76.7%)	55 (80.9%)	418 (76.1%)
Previous pregnancy with preeclampsia	47 (7.6%)	2 (2.9%)	45 (8.2%)
Plurality			
Singleton pregnancy	596 (96.6%)	66 (97.1%)	530 (96.5%)
Twin pregnancy	21 (3.4%)	2 (2.9%)	19 (3.5%)
Medical history			
Hypertension	24 (3.9%)	2 (2.9%)	22 (4.0%)
Diabetes mellitus type 1	10 (1.6%)	0 (0.0%)	10 (1.8%)
Diabetes mellitus type 2	3 (0.5%)	0 (0.0%)	3 (0.5%)
Smoking in this pregnancy	29 (4.7%)	2 (2.9%)	27 (4.9%)
fullPIERS predictors			
Gestational age at inclusion (weeks), median (IQR)	37.1 (34.4–38.9)	38.2 (35.1–39.9)	37.1 (34.3–38.7)
Chest pain or dyspnea	122 (19.8%)	21 (30.9%)	101 (18.4%)
Oxygen saturation < 97%	43 (7.0%)	9 (13.2%)	34 (6.2%)
Platelet count (×10^9/L), median (IQR)	203 (165–242)	178 (145–221)	206 (168–245)
Creatinine (μmol/L), median (IQR)	56 (49–67)	62 (51–68)	56 (49–66)
ALT (μkat/L), median (IQR)	0.24 (0.16–0.46)	0.25 (0.16–0.68)	0.24 (0.16–0.45)
AST (μkat/L), median (IQR)	0.45 (0.36–0.68)	0.53 (0.33–0.85)	0.45 (0.36–0.65)
Pregnancy outcomes			
HELLP syndrome	11 (1.8%)	3 (4.4%)	8 (1.5%)
Gestational age at delivery (weeks), median (IQR)	37.6 (35.3–39.1)	38.4 (36.0–40.0)	37.4 (35.3–38.9)
Route of delivery[a]			
Vaginal delivery	290 (45.5%)	36 (51.4%)	254 (44.7%)
Operative vaginal delivery	38 (6.0%)	7 (10.0%)	31 (5.5%)
Cesarean section	304 (47.6%)	27 (38.6%)	277 (48.8%)
Birthweight (g)[a]	2770 (930)	2979 (1051)	2842 (976)
Stillbirth[a]	2 (0.3%)	0 (0.0%)	2 (0.4%)

Note: Data are presented as mean (SD) or median (IQR) for continuous variables and as *n* (%) for categorical variables. Missing data were present for age ($n=1$), BMI ($n=13$), total years of education ($n=51$), oxygen saturation ($n=432$; values set to 97% when not recorded), platelet count ($n=6$), creatinine ($n=7$), ALT ($n=29$), AST ($n=231$), gestational age at delivery ($n=4$), birthweight ($n=5$), stillbirth ($n=5$), and route of delivery ($n=6$).

Abbreviations: ALT, alanine transaminase; AST, aspartate transaminase; BMI, body mass index; HELLP, hemolysis, elevated liver enzymes, and low platelet count; IQR, interquartile range; SD, standard deviation.

[a]Data on route of delivery, birthweight, and stillbirth are based on the number of newborns ($n=638$) due to twin pregnancy.

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TABLE 2 Occurrence of adverse maternal outcomes within 48 h and within 7 days of study inclusion.

	Within 48 h	Within 7 days
Any adverse maternal outcome	34 (5.5%)	68 (11.0%)
Central nervous system	< 3 (< 0.5%)	< 3 (< 0.5%)
Eclampsia	< 3 (< 0.5%)	< 3 (< 0.5%)
Cardiorespiratory	4 (0.6%)	7 (1.1%)
Positive inotropic support	< 3 (< 0.5%)	< 3 (< 0.5%)
Infusion of a third parenteral antihypertensive drug	0 (0.0%)	< 3 (< 0.5%)
$SpO_2 < 90\%$	3 (0.5%)	4 (0.6%)
≥ 50% FiO_2 for > 1 h	< 3 (< 0.5%)	< 3 (< 0.5%)
Intubation (other than for cesarean section)	< 3 (< 0.5%)	< 3 (< 0.5%)
Pulmonary edema	< 3 (< 0.5%)	4 (0.6%)
Hematological	26 (4.2%)	57 (9.2%)
Transfusion of any blood product	26 (4.2%)	57 (9.2%)
Platelet count $< 50 \times 10^9$/L with no transfusion	0 (0.0%)	< 3 (< 0.5%)
Hepatic	3 (0.5%)	3 (0.5%)
Hepatic dysfunction	3 (0.5%)	3 (0.5%)
Renal	5 (0.8%)	6 (1.0%)
Acute renal insufficiency	5 (0.8%)	6 (1.0%)
Other	3 (0.5%)	4 (0.6%)
Placental abruption	3 (0.5%)	4 (0.6%)

Note: Data are presented as *n* (%), based on the total study population ($n = 617$). Participants may have experienced multiple adverse events. Cells with < 3 events are suppressed to maintain confidentiality. No events were observed for maternal mortality, coma, stroke or reversible ischemic neurologic deficit, transient ischemic attack, cortical blindness or retinal detachment, posterior reversible encephalopathy, myocardial ischemia or infarction, hepatic hematoma or rupture, acute renal failure, dialysis, or Bell's palsy.

Abbreviations: FiO_2, fraction of inspired oxygen; SpO_2, oxygen saturation (pulse oximetry).

The performance of the fullPIERS model was evaluated in terms of discrimination (AUC), calibration (calibration-in-the-large, calibration slope, and graphical calibration plots), and risk stratification by dividing predicted risk into predefined categories (low risk defined by < 0.025 and high risk defined by ≥ 0.30). Missing oxygen saturation values were imputed as 97%, consistent with the original fullPIERS model.[7] As AST was not routinely measured in all participants, ALT values were used to estimate AST when missing. This was based on a log-linear relationship ($\log(AST) = 0.58 \times \log(ALT)$) derived among participants with both measurements available, to retain alignment with the original model specification. Other missing data in the fullPIERS validation were handled using single imputation.

To evaluate potential model improvements, model updating was performed via recalibration and refitting. Recalibration was conducted using logistic regression with the original model's linear predictor as the sole explanatory variable, whereas refitting was performed using multivariable logistic regression in the study cohort to re-estimate model coefficients. Updated models were evaluated in the full dataset, and internal validation was conducted using leave-one-out cross-validation.

All statistical analyses were conducted using R version 4.5.1 (R Foundation for Statistical Computing, Vienna, Austria) and IBM SPSS Statistics version 29.0 (IBM Corp., Armonk, NY, USA).

3 | RESULTS

Between May 2019 and March 2025, 715 women with preeclampsia were enrolled in the GoPROVE and UPMOST studies. Among them, 617 were eligible for inclusion in this study. In total, 34 (5.5%) women experienced an adverse maternal outcome within 48 h and 68 (11.0%) women within 7 days of inclusion (Figure 1).

The participants had a mean age of 31.5 years (SD 4.9) and 512 (83.0%) of the women were of white ethnicity. The mean body mass index was 27.2 kg/m^2 (SD 5.9). A few had pregestational conditions such as hypertension ($n = 24$, 3.9%) or diabetes mellitus type 1 or 2 ($n = 13$, 2.1%). Most were singleton pregnancies ($n = 596$, 96.6%) and 473 (76.7%) were nulliparous. There were two (2.9%) women with any adverse maternal outcome and 19 (3.5%) women in the group without any adverse maternal outcome that had a twin pregnancy. Of the multiparous women, 47 (7.6%) women had a history of previous preeclampsia. Eleven (1.8%) were diagnosed with Hemolysis, Elevated Liver enzymes and Low Platelet count (HELLP) syndrome. Most were term pregnancies (median gestational age at inclusion of 37.3 weeks, IQR: 34.4–38.9), 43 (7.0%) had oxygen saturation < 97%, and 122 (19.8%) reported chest pain and/or dyspnea. The median platelet count was 203×10^9/L (IQR: 165–242), the median creatinine was 56 (IQR: 49–67) μmol/L, and median ALT and AST levels were 0.24 (IQR: 0.16–0.46) and 0.45 (IQR: 0.36–0.68) μkat/L,

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TABLE 3 Univariable associations between fullPIERS predictors and adverse maternal outcomes within 48 h and 7 days of study inclusion.

Predictor	Within 48 h, OR (95% CI)	*P*	Within 7 days, OR (95% CI)	*P*
Any adverse maternal outcome	*n* = 34		*n* = 68	
Gestational age at inclusion, per week decrease	0.91 (0.81, 1.01)	0.085	0.94 (0.87, 1.01)	0.091
Chest pain or dyspnea	2.03 (0.96, 4.30)	0.063	1.98 (1.13, 3.46)	0.016
Oxygen saturation, per percentage point decrease	0.75 (0.49, 1.13)	0.17	1.08 (0.77, 1.51)	0.67
Platelet count, per 33% decrease	1.91 (1.23, 2.97)	0.004	1.72 (1.23, 2.39)	0.001
Creatinine, per 50% increase	2.00 (1.08, 3.69)	0.027	1.62 (1.02, 2.56)	0.040
AST, per 50% increase	1.10 (0.84, 1.44)	0.48	1.08 (0.88, 1.32)	0.45
ALT, per 50% increase	1.10 (0.94, 1.28)	0.23	1.06 (0.94, 1.20)	0.32
Nonhematological adverse outcome	*n* = 14		*n* = 19	
Gestational age at inclusion, per week decrease	1.01 (0.89, 1.16)	0.84	1.09 (0.98, 1.20)	0.097
Chest pain or dyspnea	1.64 (0.51, 5.33)	0.41	1.92 (0.71, 5.15)	0.20
Oxygen saturation, per percentage point decrease	1.10 (0.54, 2.26)	0.79	1.15 (0.62, 2.12)	0.67
Platelet count, per 33% decrease	3.11 (1.59, 6.06)	<0.001	2.49 (1.40, 4.42)	0.002
Creatinine, per 50% increase	6.66 (2.66, 16.66)	<0.001	4.79 (2.16, 10.60)	<0.001
AST, per 50% increase	1.34 (0.98, 1.84)	0.067	1.31 (0.99, 1.73)	0.057
ALT, per 50% increase	1.20 (0.98, 1.48)	0.083	1.21 (1.01, 1.46)	0.041

Note: Missing data were present for platelet count (*n* = 6), creatinine (*n* = 7), oxygen saturation (*n* = 432; values set to 97% when not recorded), ALT (*n* = 29), and AST (*n* = 231). Analyses were performed using available-case data. Statistical analyses were conducted using univariable logistic regression with one predictor analyzed at a time. Results are presented as odds ratios (ORs) with 95% confidence intervals (CIs). Platelet count, creatinine, AST and ALT were log-transformed prior to analysis. Predictors were scaled as follows: gestational age (per week decrease), oxygen saturation (per percentage point decrease), platelet count (per 33% decrease), and creatinine/AST/ALT (per 50% increase).

Abbreviations: ALT, alanine transaminase; AST, aspartate transaminase.

respectively. Overall, there were few missing data points in this dataset, with the exception of oxygen saturation (*n* = 432 [70.0%]) and AST (*n* = 231 [37.4%]) (Table 1).

The prevalence of adverse maternal outcomes within 48 h and 7 days is presented in Table 2. The most common complication was transfusion of any blood product, occurring in 26 cases (4.2%) within 48 h and 57 (9.2%) within 7 days. Eclampsia occurred in fewer than three (< 0.5%) women. Four (0.6%) women had any cardiorespiratory event within 48 h and seven (1.1%) women within 7 days of inclusion. Three (0.5%) women had hepatic dysfunction within 48 h of inclusion. Five (0.8%) women had acute renal insufficiency within 48 h and six (1.0%) women within 7 days of inclusion. Placental abruption occurred in three (0.5%) women within 48 h and four (0.6%) within 7 days of inclusion.

Univariable analyses of predictors for adverse maternal outcomes within 48 h and 7 days are presented in Table 3. The strongest associations with adverse maternal outcomes were observed for decrease in platelet count and increase in creatinine levels. For observations within 48 h, each 33% decrease in platelet count was associated with 1.9-fold higher odds of any event (95% CI: 1.23, 2.97; $p = 0.004$). Each 50% increase in creatinine was associated with 2-fold higher odds for any event (95% CI: 1.08, 3.69; $p = 0.027$). Both platelet count and creatinine remained statistically significant predictors of adverse maternal outcomes within 7 days of inclusion.

In analyses excluding hematological outcomes the associations remained. Each 33% decrease in platelet count was associated with 3.1-fold higher odds of non-hematological adverse outcome within 48 h (95% CI: 1.59, 6.06; $p < 0.001$) and 2.5-fold higher odds within 7 days (95% CI: 1.40, 4.42; $p = 0.002$). Similarly, each 50% increase in creatinine was associated with 6.7-fold higher odds of a non-hematological adverse outcome within 48 h (95% CI: 2.66, 16.66; $p < 0.001$) and 4.8-fold higher odds within 7 days (95% CI: 2.16, 10.60; $p < 0.001$).

The fullPIERS model demonstrated modest discriminatory performance for predicting adverse maternal outcomes within 48 h (AUC 0.64; 95% CI: 0.54, 0.74) and within 7 days (AUC 0.64; 95% CI: 0.57, 0.71). When analyses were restricted to nonhematological outcomes, discrimination improved, with AUCs of 0.79 (95% CI: 0.68, 0.89) at 48 h and 0.78 (95% CI: 0.69, 0.87) at 7 days (Figure 2). Refitting the model in the current cohort yielded higher apparent

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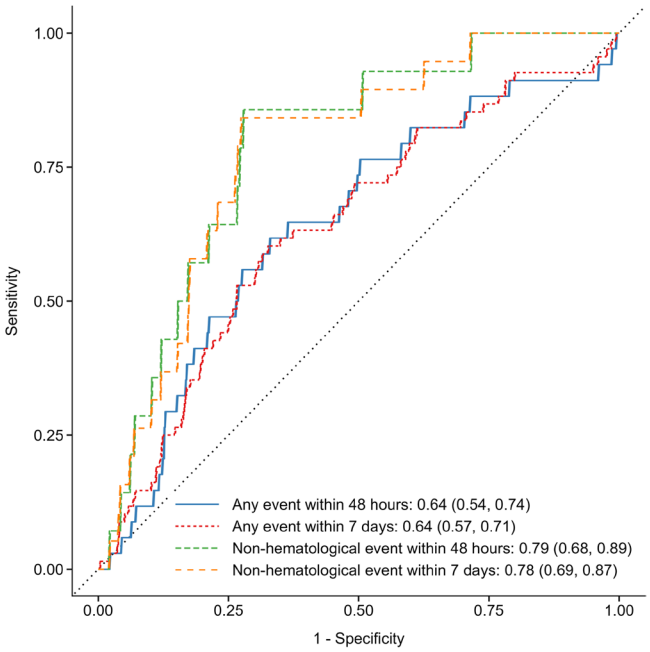

FIGURE 2 Receiver operating characteristic (ROC) curves for prediction of adverse maternal outcomes according to the fullPIERS model. Solid blue line: Any event within 48 h; dotted red line: Any event within 7 days; short-dashed green line: nonhematological event within 48 h; and long-dashed orange line: nonhematological event within 7 days of study inclusion. Values represent the area under the ROC curve (AUC) with 95% confidence intervals. The diagonal line indicates no discrimination (AUC = 0.5).

AUCs; however, these improvements were attenuated after correction for optimism using cross-validation and did not result in improved performance compared with the original model (Supporting Information Figure S1).

For the composite outcome of any adverse maternal outcome, the model demonstrated clear miscalibration at both 48 h and 7 days. At 48 h, risk was underestimated among lower risk individuals and overestimated among those at higher predicted risk. At 7 days, the model showed overall underestimation of risk across the range of predicted probabilities. When analyses were restricted to nonhematological outcomes, calibration improved substantially, with no statistically significant evidence of miscalibration (calibration intercept confidence intervals including 0 and calibration slope confidence intervals including 1) (Figure 3).

Risk stratification according to the fullPIERS model is presented in Table 4. A total of 489 (79.3%) women were classified as low risk (< 0.025), of whom 20 (4.1%) developed an adverse maternal outcome within 48 h, with a corresponding sensitivity of 0.41 (95% CI: 0.26, 0.58), specificity of 0.80 (95% CI: 0.77, 0.83). The positive predictive value (PPV) was 0.11 (95% CI: 0.07, 0.18), and the negative predictive value (NPV) was 0.96 (95% CI: 0.94, 0.97). At the upper end of the risk spectrum, 17 (2.8%) women had a predicted risk ≥ 0.10; however, only one (5.9%) of these experienced an adverse maternal outcome within 48 h. Within 7 days, 44 (9.0%) women classified as low risk had an adverse maternal outcome, with a corresponding sensitivity of 0.35 (95% CI: 0.25, 0.47), specificity of 0.81 (95% CI: 0.78, 0.84), PPV of 0.19 (95% CI: 0.13, 0.26), and NPV of 0.91 (95% CI: 0.88, 0.93).

For nonhematological adverse outcomes, risk stratification showed improved rule-out performance. Among the 489 women classified as low risk (< 0.025), six (1.2%) developed a nonhematological event within 48 h, with a sensitivity of 0.57 (95% CI: 0.33, 0.79) and a specificity of 0.80 (95% CI: 0.77, 0.83). The PPV was 0.06 (95% CI: 0.03, 0.12), and the NPV was 0.99 (95% CI: 0.97, 0.99). Within 7 days, eight (1.6%) developed a nonhematological event, with a sensitivity of 0.58 (95% CI: 0.36, 0.77), specificity of 0.80 (95% CI: 0.77, 0.83), PPV of 0.09 (95% CI: 0.05, 0.15), and NPV of 0.98 (95% CI: 0.97, 0.99).

At a risk threshold of < 0.01, 297 (48.1%) women were classified as low risk, of whom one (0.3%) developed a nonhematological event within 48 h, corresponding to a sensitivity of 0.93 (95% CI: 0.69, 0.99) and specificity of 0.49 (95% CI: 0.45, 0.53). The PPV was 0.04 (95% CI: 0.02, 0.07), and the NPV was 1.00 (95% CI: 0.98, 1.00). Within 7 days, two (0.6%) women classified as low risk developed a nonhematological event, corresponding to a sensitivity of 0.89 (95% CI: 0.69, 0.97), specificity of 0.49 (95% CI: 0.45, 0.53), PPV of 0.05 (95% CI: 0.03, 0.08), and NPV of 0.99 (95% CI: 0.98, 1.00).

4 | DISCUSSION

In this external validation study, the fullPIERS model showed modest discriminatory performance for predicting adverse maternal outcomes in preeclampsia. Using the predefined low-risk threshold of < 0.025, the model showed limited sensitivity and underestimated risk among women classified as low risk. Excluding hematological outcomes improved performance, reducing the number of events in the low-risk group from 20 to six and increased sensitivity. Defining the risk cutoff at < 0.01, nearly half of the cohort was identified as low risk. While one adverse nonhematological outcome occurred below this threshold, overall sensitivity and negative predictive value remained high. This suggests that the model may still support risk-adapted clinical decision-making, although it may not be independently sufficient as a strict rule-out test for future complications.

Adverse maternal outcomes were rare in our population, and a higher proportion of women were classified as low risk compared to the original fullPIERS study.[7] The fullPIERS model appeared to overestimate the risk of adverse maternal outcomes and demonstrated poor performance in identifying high-risk individuals in our cohort. This may be explained by the close monitoring of pregnant women in Sweden, leading to a treatment paradox. This includes well-established antenatal care, aspirin use in high-risk pregnancies, administration of magnesium sulfate to women with neurological symptoms and timing of birth (induction of labor or cesarean section at the latest at 37 weeks or at diagnosis at term).[1,4]

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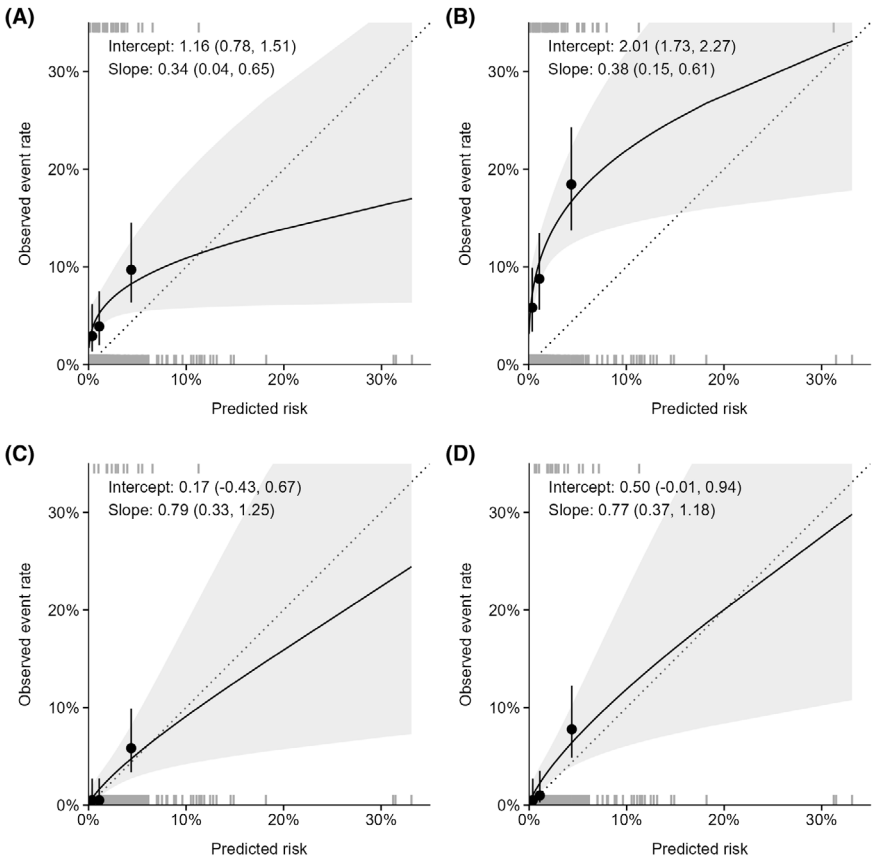

FIGURE 3 Calibration plots of the fullPIERS model for prediction of adverse maternal outcomes. (A) Any event within 48 h, (B) any event within 7 days, (C) nonhematological event within 48 h, and (D) nonhematological event within 7 days of study inclusion. The solid line represents observed versus predicted risk estimated using a logistic regression model with natural cubic splines, with shaded areas indicating 95% confidence intervals (CIs). Points with error bars represent observed event rates with 95% CIs across risk groups comprising 40%, 30%, 20%, and 10% of the cohort, ordered by predicted risk. The diagonal line represents perfect calibration. Calibration intercept (calibration-in-the-large) and calibration slope estimates, with 95% CIs, are shown within each panel. Rug marks along the top and bottom of the *x*-axis indicate the distribution of predicted risks among women with and without events, respectively.

When including hematological events, the AUCs were lower than those of the original fullPIERS model development cohort (AUC 0.88, 95% CI 0.84, 0.92).[7] Previous external validations have been conducted across diverse geographic and clinical settings. A few of these studies have demonstrated good discrimination; however, they are collectively characterized by their small sample sizes.[13,14,17,19] Other studies have demonstrated more moderate discrimination.[8,11,15,18,20,21,25]

Mirkovic et al., Ukah et al., and Gomes et al.[16,22,26] reported results comparable to ours, with AUC 0.67 within 48 h and AUCs ranging from 0.63 to 0.67 within 7 days. Our study included both early-onset and late-onset preeclampsia, as did Gomes et al.[22] In contrast, Mirkovic et al.[16,26] included only women with early-onset preeclampsia, and the cohort study by Ukah et al. also predominantly comprised women with early-onset preeclampsia. Akkermans et al.[14] also examined early-onset alone; however, they presented a higher AUC of 0.97 (95% CI 0.87, 0.99) within 48 h and AUC of 0.80 (95% CI 0.70, 0.87) within 7 days.

Some studies validating the fullPIERS model used different adverse outcomes than those in the original fullPIERS model development, including HELLP syndrome.[11,13,17,21] These modifications did not appear to consistently affect the AUC. Furthermore, Mirkovic et al.[16] included fetal growth restriction (FGR) as a complication, also not described in the original fullPIERS model. In their study, FGR was present in 68.9% of women, which could have impacted the discrimination of the model. Previous validation studies have also highlighted concerns regarding the inclusion of blood transfusion as an outcome, since it is not specific to preeclampsia.[10] In the cohort reported by Mirkovic et al.,[16] 60.7% of all the women received blood transfusion within 7 days, which may also have impacted the model discrimination. In our cohort, 9.2% received any blood product, making transfusion the most common complication. We conducted analyses with and without the hematological outcomes, focusing on organ complications more related to the pathophysiology of preeclampsia alone. This improved model performance.

Ukah et al.[26] incorporated an angiogenic biomarker to the fullPIERS model, although this did not improve predictive accuracy of the model. Mirkovic et al.[16] compared the predictive value for adverse maternal outcomes between angiogenic biomarkers and the fullPIERS model, showing a better predictive value for the model using only angiogenic biomarkers. However, Carlberg et al.[27] showed that angiogenic factors have little to modest value in predicting adverse maternal outcomes in preeclampsia in a Swedish population. Future studies could focus on further evaluating the predictive value of biomarkers with the fullPIERS model.

In the univariable analyses, lower platelet count, higher creatinine levels, and the presence of chest pain or dyspnea were associated with an increased risk of the composite adverse maternal outcome. No other predictors showed statistically significant associations. When analyses were restricted to nonhematological outcomes, lower platelet count and higher creatinine levels were significantly

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TABLE 4 Risk stratification performance of the fullPIERS model for prediction of adverse maternal outcomes within 48 h and 7 days of inclusion, overall and excluding hematological events.

Predicted risk category[a]	Event rate, *n/N* (%)	Sensitivity (95% CI)	Specificity (95% CI)	Positive predictive value (95% CI)	Negative predictive value (95% CI)
Any adverse maternal outcome within 48 h					
0–0.009	8/297 (2.7%)	1.00 (0.90, 1.00)	0.00 (0.00, 0.01)	0.06 (0.04, 0.08)	NA
0.01–0.024	12/192 (6.2%)	0.76 (0.60, 0.88)	0.50 (0.46, 0.54)	0.08 (0.06, 0.12)	0.97 (0.95, 0.99)
0.025–0.049	10/81 (12.3%)	0.41 (0.26, 0.58)	0.80 (0.77, 0.83)	0.11 (0.07, 0.18)	0.96 (0.94, 0.97)
0.05–0.09	3/30 (10.0%)	0.12 (0.05, 0.27)	0.93 (0.90, 0.94)	0.09 (0.03, 0.20)	0.95 (0.93, 0.96)
0.10–0.29	1/14 (7.1%)	0.03 (0.01, 0.15)	0.97 (0.96, 0.98)	0.06 (0.01, 0.27)	0.94 (0.92, 0.96)
≥ 0.30	0/3 (0.0%)	0.00 (0.00, 0.10)	0.99 (0.98, 1.00)	0.00 (0.00, 0.56)	0.94 (0.92, 0.96)
Total	34/617				
Any adverse maternal outcome within 7 days					
0–0.009	19/297 (6.4%)	1.00 (0.95, 1.00)	0.00 (0.00, 0.01)	0.11 (0.09, 0.14)	NA
0.01–0.024	25/192 (13.0%)	0.72 (0.60, 0.81)	0.51 (0.46, 0.55)	0.15 (0.12, 0.20)	0.94 (0.90, 0.96)
0.025–0.049	15/81 (18.5%)	0.35 (0.25, 0.47)	0.81 (0.78, 0.84)	0.19 (0.13, 0.26)	0.91 (0.88, 0.93)
0.05–0.09	7/30 (23.3%)	0.13 (0.07, 0.23)	0.93 (0.91, 0.95)	0.19 (0.10, 0.33)	0.90 (0.87, 0.92)
0.10–0.29	1/14 (7.1%)	0.03 (0.01, 0.10)	0.97 (0.96, 0.98)	0.12 (0.03, 0.34)	0.89 (0.86, 0.91)
≥ 0.30	1/3 (33.3%)	0.01 (0.00, 0.08)	1.00 (0.99, 1.00)	0.33 (0.06, 0.79)	0.89 (0.86, 0.91)
Total	68/617				
Nonhematological adverse outcome within 48 h					
0–0.009	1/297 (0.3%)	1.00 (0.78, 1.00)	0.00 (0.00, 0.01)	0.02 (0.01, 0.04)	NA
0.01–0.024	5/192 (2.6%)	0.93 (0.69, 0.99)	0.49 (0.45, 0.53)	0.04 (0.02, 0.07)	1.00 (0.98, 1.00)
0.025–0.049	4/81 (4.9%)	0.57 (0.33, 0.79)	0.80 (0.77, 0.83)	0.06 (0.03, 0.12)	0.99 (0.97, 0.99)
0.05–0.09	3/30 (10.0%)	0.29 (0.12, 0.55)	0.93 (0.91, 0.95)	0.09 (0.03, 0.20)	0.98 (0.97, 0.99)
0.10–0.29	1/14 (7.1%)	0.07 (0.01, 0.31)	0.97 (0.96, 0.98)	0.06 (0.01, 0.27)	0.98 (0.96, 0.99)
≥ 0.30	0/3 (0.0%)	0.00 (0.00, 0.22)	1.00 (0.99, 1.00)	0.00 (0.00, 0.56)	0.98 (0.96, 0.99)
Total	14/617				
Nonhematological adverse outcome within 7 days					
0–0.009	2/297 (0.7%)	1.00 (0.83, 1.00)	0.00 (0.00, 0.01)	0.03 (0.02, 0.05)	NA
0.01–0.024	6/192 (3.1%)	0.89 (0.69, 0.97)	0.49 (0.45, 0.53)	0.05 (0.03, 0.08)	0.99 (0.98, 1.00)
0.025–0.049	6/81 (7.4%)	0.58 (0.36, 0.77)	0.80 (0.77, 0.83)	0.09 (0.05, 0.15)	0.98 (0.97, 0.99)
0.05–0.09	4/30 (13.3%)	0.26 (0.12, 0.49)	0.93 (0.91, 0.95)	0.11 (0.05, 0.23)	0.98 (0.96, 0.99)
0.10–0.29	1/14 (7.1%)	0.05 (0.01, 0.25)	0.97 (0.96, 0.98)	0.06 (0.01, 0.27)	0.97 (0.95, 0.98)
≥ 0.30	0/3 (0.0%)	0.00 (0.00, 0.17)	0.99 (0.99, 1.00)	0.00 (0.00, 0.56)	0.97 (0.95, 0.98)
Total	19/617				

Note: Confidence intervals (CIs) for true positive rate (sensitivity), false positive rate (1 − specificity), positive predictive value, and negative predictive value were calculated using the Wilson score method.

Abbreviation: NA, not applicable (negative predictive value undefined).

[a]Event rates are presented within each predicted-risk interval. Sensitivity, specificity, positive predictive value, and negative predictive value were calculated using the lower boundary of each interval as the classification threshold, with women having a predicted risk greater than or equal to the threshold classified as test-positive.

associated with adverse maternal outcomes. These predictors in the fullPIERS model may reflect early or subclinical manifestations of later maternal complications. For example, chest pain or dyspnea may indicate emerging respiratory compromise. A model that would be more clinically useful should include signs and symptoms that come before clinical deterioration. Similar interpretations have been proposed in previous studies.[10] Furthermore, the fullPIERS model is based on a wide variety of clinical predictors that are used to predict several different adverse maternal outcomes. This might hamper the model's predictive performance. When hematological outcomes, one of the most common groups of outcomes and arguably the least directly related to preeclampsia pathophysiology, were excluded from the composite outcome, the model's performance improved. These aspects can be explored further in future studies, focusing

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on one or a few outcomes sharing a common pathophysiological pathway.

A strength of this study is its generalizability to the broader Swedish preeclampsia population, with similar characteristics in terms of age, BMI, and gestational age at delivery compared to national register data.[28] Another strength is that this study has the same inclusion criteria as the development cohort of the fullPIERS model except for not including women with HELLP syndrome without hypertension and proteinuria.[7]

One limitation was that there was a substantial amount of missing data for oxygen saturation and AST. Though, missing data for oxygen saturation has been common also in the development cohort and other validation studies. Imputation for oxygen saturation was conducted in accordance with the original fullPIERS development study.[7] Since AST was not routinely measured in all participants, AST was estimated from ALT when missing. Previous validation studies have reported similar or improved performance when using ALT.[15] However, to preserve consistency with the original model specification, we chose to estimate AST from ALT rather than replacing AST directly with ALT. All outcome variables except for severe ascites were available in our cohort. In the original cohort, severe ascites was present in two out of 2023 women.[7]

Another limitation is the moderate number of observations in the higher-risk strata, particularly after excluding hematological outcomes. This reduces the precision of risk stratification and contributes to wide confidence intervals in calibration. Nevertheless, the overall study sample met the prespecified sample size requirements and exceeded that of many previous external validation studies.[11,13,14,16–20,22,26]

5 | CONCLUSION

In this Swedish external validation study, the fullPIERS model showed poor clinical utility for identifying both low-risk and high-risk individuals for ruling in or ruling out adverse maternal outcomes. Excluding hematological outcomes improved performance. At a risk threshold of <0.01, the model performed well in identifying women at low risk for nonhematological outcomes. This suggests potential clinical utility in supporting risk-adapted clinical decision-making. Future studies should focus on validating the fullPIERS model without hematological outcomes, evaluate the performance as rule out at <0.01 threshold, and combine the model with preeclampsia specific biomarkers. If the model would demonstrate good predictive performance, examining the outcomes among high- and low-risk patients could be of interest.

AUTHOR CONTRIBUTIONS

Linnea Dahlberg and Tove Sandberg: Coordinated the project, conducted most of the validation and manuscript preparation, and performed parts of the statistical analyses. Henrik Imberg: Oversaw and planned statistical analyses and performed statistical analyses. Revised manuscript. Julia Andersson: Planned the project. Performed initial analyses and validation of data. Revised manuscript. Anna Pontoppidan: Planned the project. Performed initial analyses and validation of data. Revised manuscript. Niclas Carlberg: Validated data. Revised manuscript. Valentina Bucher: Structured the manuscript. Revised manuscript. Katja Junus: Validated data. Revised manuscript. Erica Wesslén: Validated data. Revised manuscript. Catherine Cluver: Revised manuscript. Anna-Karin Wikström: Planned the project. Revised manuscript. Nicole Wallin: Planned the project. Validated data. Structured the manuscript. Revised manuscript. Provided supervision. Lina Bergman: Planned the project and provided overall supervision. Revised manuscript. Secured funding for the project.

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CONFLICT OF INTEREST STATEMENT

CC provides consulting services to Diamedica, Avilar, and Beech Biotech. HI provides consulting services to Diamedica and Beech Biotech. LB provides consulting services to Diamedica and Arraka. LB has received donated PlGF and sFlt-1 reagents from Thermo Fisher, Roche, and Revvity. CC and LB have received donated drug and placebo from Merck Damstadt. AK-W has received donated PlGF reagents from Thermo Fisher, Roche, and Revvity. None of these are related to this study.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

ETHICS STATEMENT

The the Swedish Ethical Review Authority approved the GoPROVE (reference number 2018-955-18, approved on December 28, 2018) and UPMOST studies (reference number 2019-00309, approved February 27, 2019). Diary numbers for amendments to GoPROVE are 2019-03734, 2020-02291, 2022-00574-02, 2023-02271-02, 2024-00677-02, and 2025-03365-02. Diary numbers for amendments to UPMOST are 2020-01997, 2020-05651, 2022-05383-02 and 2025-00201-02. The GoPROVE and UPMOST Database and Biobank are registered as one study at ISRCTN (13060768) and published as one protocol.[23] All participants provided informed consent before data collection. The study was conducted in accordance with the Declaration of Helsinki.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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