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Research Article

EFFICACY OF LOW-DOSE ASPIRIN IN HIGH-RISK PREGNANT WOMEN IDENTIFIED BY FIRST-TRIMESTER DOPPLER AND BIOCHEMICAL MARKERS

Dr. Sonalica Suresh[1], Dr. Kalpana G[2], Dr Rejeesh S Ravi[3], Dr. Laxmi Menon

[1]Specialist Obstetrics and Gynecology, NMC Specialty Hospital, Dubai.
[2]Associate Professor, Department of OBG, P.K. Das Institute of Medical sciences, Vaniamkulam, Ottapalam, Palakkad, Kerala – 679522, India.
[3]Assistant Professor, Department of OBG, Malabar Medical College, Ulliyeri, Kozhikode
[4]Assistant Professor, Department of Obstetrics and Gynaecology, Sri Muthukumaran Medical College and Research Institute, (SMMMCH&RI), Chikkarayapuram, Kundrathur Road, Mangadu, Chennai – 600069, Tamil Nadu, India.

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Corresponding Author: Dr. Laxmi Menon, Assistant Professor, Department of obstetrics and Gynaecology, Sri Muthukumaran medical College and research institute, (SMMMCH&RI), Chikkarayapuram, Kundrathur Road, Mangadu, Chennai – 600069, Tamil Nadu, India.
Email: laxmimenon5599@gmail.com

ABSTRACT

Background: Preeclampsia remains a major cause of maternal and perinatal morbidity and mortality. Early identification of high-risk women enables preventive strategies, but reliable predictive markers are limited in routine use. This study evaluated the predictive role of uterine artery Doppler, serum PAPP-A, serum β-hCG, and mean arterial pressure in the early detection of preeclampsia. **Methods:** A prospective observational study was conducted among 400 singleton pregnancies enrolled at 11-14 weeks of gestation. Maternal mean arterial pressure was measured, uterine artery Doppler velocimetry was performed, and maternal serum PAPP-A and β-hCG levels were estimated. Participants were followed until delivery, and development of preeclampsia was documented. Diagnostic performance was assessed using sensitivity, specificity, predictive values, and area under the ROC curve (AUC). **Results:** Preeclampsia developed in 28 women (7.0%). Those who developed the disorder had significantly higher mean arterial pressure (87.9 vs 84.2 mmHg, $p = 0.0002$) and uterine artery PI in both first (1.94 vs 1.44, $p < 0.001$) and second trimesters (1.51 vs 1.18, $p < 0.001$). Serum PAPP-A and β-hCG levels were significantly lower in affected women (1.01 vs 1.59 MoM and 1.66 vs 2.58 MoM, respectively; $p < 0.001$). Predictive accuracy was highest for first-trimester uterine artery PI (AUC 0.941) and PAPP-A (AUC 0.902), while MAP and β-hCG showed moderate accuracy (AUC 0.72 and 0.78, respectively). A combined model of MAP, uterine artery PI, and PAPP-A improved detection with sensitivity 93.3% and specificity 96.4%. Women receiving low-dose aspirin prophylaxis showed a trend toward reduced early-onset preeclampsia. **Conclusion:** Uterine artery Doppler indices, serum PAPP-A, β-hCG, and MAP measured in the first trimester provide valuable predictive information for preeclampsia, with multiparametric models offering the greatest accuracy. Early identification and prophylaxis may mitigate disease severity and improve outcomes.

Keywords: Preeclampsia prediction. Uterine artery Doppler. PAPP-A.

INTRODUCTION

Hypertensive disorders of pregnancy (HDP), particularly preeclampsia, continue to be among the leading causes of maternal and perinatal morbidity and mortality worldwide, despite decades of advances in obstetric care. Preeclampsia is a multisystem, pregnancy-specific disorder typically characterized by new-onset hypertension and proteinuria after 20 weeks of gestation, although atypical presentations without proteinuria are increasingly recognized. Globally, preeclampsia complicates approximately 2–8% of all pregnancies, and its burden is disproportionately higher in low- and middle-income countries where access to early screening and adequate antenatal care may be limited. The consequences extend beyond maternal morbidity and mortality-perinatal outcomes such as intrauterine growth restriction (IUGR), preterm birth, stillbirth, and neonatal intensive care admissions are frequently observed. Against this backdrop, the development of effective preventive strategies is paramount in contemporary obstetrics.[1][2]

The pathogenesis of preeclampsia is complex and multifactorial, involving abnormal placentation, impaired trophoblastic invasion, defective remodeling of the spiral arteries, and exaggerated maternal systemic inflammatory response. Central to its origin is the inadequate transformation of spiral arteries into low-resistance vessels during early pregnancy, leading to reduced uteroplacental perfusion. This state triggers the release of antiangiogenic factors, oxidative stress, and endothelial dysfunction, which together culminate in the clinical syndrome of preeclampsia. Because the pathophysiological cascade begins in the first trimester, well before clinical symptoms manifest, attention has shifted towards early identification of women at risk and timely preventive interventions. The recognition that prophylaxis must begin before the clinical onset of disease, ideally in early pregnancy, has driven significant interest in first-trimester screening strategies.[3]

Traditionally, risk stratification for preeclampsia relied on maternal demographic and clinical characteristics such as chronic hypertension, diabetes, obesity, advanced maternal age, and history of preeclampsia. While helpful, these risk factors alone have poor predictive accuracy. Over the last two decades, integration of biophysical markers-most notably uterine artery Doppler velocimetry-with biochemical markers such as pregnancy-associated plasma protein A (PAPP-A), placental growth factor (PlGF), and soluble fms-like tyrosine kinase-1 (sFlt-1), has significantly improved predictive models. First-trimester uterine artery Doppler assesses the resistance to blood flow in the uteroplacental circulation, with abnormal indices (elevated pulsatility index, presence of notching) reflecting impaired trophoblastic invasion. Likewise, low levels of PlGF or PAPP-A correlate with poor placentation and heightened preeclampsia risk. Together, these modalities allow earlier and more accurate identification of high-risk women, offering a window of opportunity for preventive intervention.[4][5]

Among available preventive options, low-dose aspirin (acetylsalicylic acid, ASA) has emerged as the most widely studied and promising pharmacological agent. The rationale for aspirin use lies in its ability to irreversibly inhibit platelet cyclooxygenase-1, thereby reducing thromboxane A2 synthesis and shifting the prostacyclin–thromboxane balance towards vasodilation and reduced platelet aggregation. Aspirin also exerts anti-inflammatory and antioxidative effects, which may mitigate the endothelial dysfunction central to preeclampsia. Importantly, when administered before 16 weeks of gestation-during the critical window of spiral artery remodeling-low-dose aspirin has been shown to significantly reduce the incidence of preeclampsia, fetal growth restriction, and preterm birth in high-risk women. Multiple randomized controlled trials (RCTs) and meta-analyses support its efficacy, with some

evidence suggesting greater benefit in women at the highest risk identified by combined screening models.[6]

Aim

To evaluate the role of uterine artery Doppler, maternal serum PAPP-A, β-hCG, and mean arterial pressure in predicting preeclampsia.

Objectives

1. To assess the predictive accuracy of uterine artery Doppler, PAPP-A, β-hCG, and mean arterial pressure for preeclampsia.
2. To evaluate the effectiveness of initiating preventive measures (e.g., low-dose aspirin) in high-risk cases identified through early screening.
3. To determine whether timely management strategies could reduce progression and complications of preeclampsia.

MATERIAL AND METHODOLOGY

The study population consisted of all pregnant women attending the antenatal clinic.

A prospective observational study was carried out.

A total of 400 pregnant women were enrolled. Sample size was calculated using the formula:

$$n = \frac{(1.96)^2 \times (S_n)(1 - S_n)}{L^2 P}$$

With Z = 1.96 (5% alpha error), anticipated sensitivity = 90%, precision = 0.1, and incidence of PE = 9%, the calculated sample size was 385. Allowing for attrition, 400 women were included.

Inclusion Criteria

- Pregnant women in their first trimester (up to 14 weeks gestation).
- Singleton pregnancies.
- Women who provided informed consent for participation.

Exclusion Criteria

- Women attending antenatal clinic beyond 14 weeks gestation.
- Chronic hypertension.
- Multiple gestation.
- Pre-existing renal disease, cardiovascular disease, or autoimmune disorders.

Procedure and Methodology

At recruitment, a detailed history, systemic examination, and obstetric assessment were conducted. Maternal risk factors were recorded.

Mean Arterial Pressure (MAP): Blood pressure was measured with a mercury sphygmomanometer using the auscultatory method. Women were seated comfortably with the cuff at heart level. Korotkoff's phase V was taken as diastolic pressure. MAP was calculated as:

$$MAP = \frac{SBP + 2(DBP)}{3}$$

A cut-off of >86.5 mmHg (derived by ROC analysis) was considered predictive for PE.

Serum Markers: Blood samples were collected at 11+0 to 13+6 weeks gestation. PAPP-A and β-hCG levels were measured by standardized commercial assays and expressed as multiples of the median (MoM), adjusted for gestational age, maternal weight, ethnicity, and diabetes status. Cut-offs: PAPP-A <1.085 MoM and β-hCG <1.975 MoM were taken as predictive.

Uterine Artery Doppler: Performed by an experienced sonologist using a 3.5-5 MHz transabdominal probe. The uterine artery was identified at the level of the internal cervical os

with color Doppler. Pulsatility Index (PI) was measured from three consecutive waveforms with an insonation angle <30°. The mean of left and right uterine artery PI was recorded. PI >1.745 in the first trimester and >1.265 in the second trimester were considered predictive. Early diastolic notching was also noted.

Follow-up: Women with abnormal screening parameters were started on low-dose aspirin (75 mg once daily) before 16 weeks. Uterine artery Doppler was repeated at 18-20 weeks. Participants were followed until delivery, and maternal and perinatal outcomes were documented.

Sample Processing

Serum samples were processed in the hospital laboratory under strict quality control. Results were reported in MoM for standardization.

Statistical Methods

Data were analyzed using SPSS version 21.0.

Continuous variables: expressed as mean ± SD, compared using independent t-test or Mann-Whitney U test.

Categorical variables: expressed as frequency/percentage, compared using Chi-square or Fisher's exact test.

Diagnostic performance: sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy were calculated.

ROC curves were used to derive best cut-off values for MAP, PAPP-A, β-hCG, and PI.

p-value <0.05 was considered statistically significant.

Data Collection

All data, including demographic variables, clinical findings, biochemical results, and Doppler indices, were recorded in a structured proforma. Maternal outcomes (PE diagnosis, gestational age at delivery, complications) and neonatal outcomes (birth weight, NICU admission, perinatal morbidity/mortality) were documented.

OBSERVATION AND RESULTS

Table 1: Baseline predictors by PE status with test of significance (N=400)

Variable	PE Mean (SD) [n=28]	No-PE Mean (SD) [n=372]	Mean diff (PE−NoPE)	95% CI of diff	Welch t (df)	p-value
Mean Arterial Pressure (mmHg)	87.90 (4.04)	84.22 (5.28)	3.68	1.85 to 5.51	3.95 (df=55.4)	0.0002
Uterine Artery PI (1st trimester)	1.94 (0.34)	1.44 (0.25)	0.50	0.36 to 0.64	7.51 (df=41.7)	<0.000001
Uterine Artery PI (2nd trimester)	1.51 (0.19)	1.18 (0.26)	0.33	0.22 to 0.44	6.46 (df=44.8)	<0.000001
PAPP-A (MoM)	1.01 (0.29)	1.59 (0.50)	-0.58	-0.75 to -0.41	-6.91 (df=44.1)	<0.000001
β-hCG (MoM)	1.66 (0.56)	2.58 (0.92)	-0.92	-1.24 to -0.60	-5.71 (df=46.6)	<0.000001

Table 1 presents the baseline predictors in women who developed preeclampsia compared with those who remained normotensive. The mean arterial pressure (MAP) was significantly higher in the preeclampsia group (87.90 ± 4.04 mmHg) compared to the non-preeclampsia group (84.22 ± 5.28 mmHg), with a mean difference of 3.68 mmHg (95% CI: 1.85-5.51, $p = 0.0002$). Both first and second trimester uterine artery pulsatility indices (PI) were markedly elevated in preeclamptic women. In the first trimester, the mean PI was 1.94 ± 0.34 compared to 1.44 ± 0.25 in controls, yielding a mean difference of 0.50 (95% CI: 0.36-0.64, $p < 0.000001$). Similarly, in the second trimester, preeclamptic women showed a mean PI of 1.51 ± 0.19 versus 1.18 ± 0.26 in controls, with a difference of 0.33 (95% CI: 0.22-0.44, $p < 0.000001$). Serum biochemical markers demonstrated the opposite trend: mean PAPP-A levels were significantly reduced in women with preeclampsia (1.01 ± 0.29 MoM) compared with normotensive pregnancies (1.59 ± 0.50 MoM), with a mean difference of −0.58 (95% CI: −0.75 to −0.41, $p < 0.000001$). Likewise, β-hCG was lower among preeclamptic women (1.66 ± 0.56 MoM) compared with controls (2.58 ± 0.92 MoM), with a mean difference of −0.92 (95% CI: −1.24 to −0.60, $p < 0.000001$).

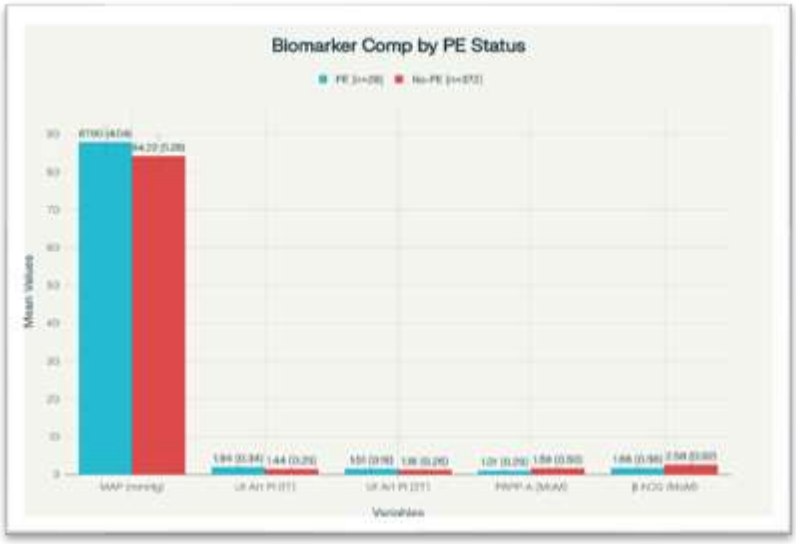

Figure 1

Table 2: Predictive accuracy of markers for Preeclampsia (N=400; PE prevalence = 7.0%)

Test (cut-off)	Sensitivity %	Specificity %	PPV % (prev 7.0%)	NPV %	AUC (95% CI)
MAP > 86.5 mmHg	64.3	72.3	15.5	96.8	0.720 (0.62-0.80)
Uterine Artery PI (1st) > 1.745	85.7	89.0	37.3	99.1	0.941 (0.90-0.97)
Uterine Artery PI (2nd) > 1.265	71.4	71.0	15.9	97.2	0.836 (0.76-0.91)
PAPP-A < 1.085 MoM	83.9	82.1	25.8	98.6	0.902 (0.85-0.95)

β-hCG < 1.975 MoM	66.7	64.3	12.0	96.6	0.780 (0.71-0.84)

Table 2 summarizes the predictive accuracy of various markers for preeclampsia. MAP > 86.5 mmHg demonstrated a moderate sensitivity (64.3%) and specificity (72.3%) with a negative predictive value (NPV) of 96.8% and an area under the ROC curve (AUC) of 0.720 (95% CI: 0.62-0.80). Uterine artery PI in the first trimester, at a cut-off > 1.745, emerged as the strongest single predictor with high sensitivity (85.7%) and specificity (89.0%), yielding an AUC of 0.941 (95% CI: 0.90-0.97) and an excellent NPV of 99.1%. In the second trimester, PI > 1.265 maintained reasonable predictive value with sensitivity and specificity both around 71%, AUC 0.836 (95% CI: 0.76-0.91). Among biochemical markers, PAPP-A < 1.085 MoM predicted preeclampsia with sensitivity of 83.9%, specificity of 82.1%, NPV of 98.6%, and an AUC of 0.902 (95% CI: 0.85-0.95). β-hCG < 1.975 MoM showed relatively lower performance (sensitivity 66.7%, specificity 64.3%, AUC 0.780).

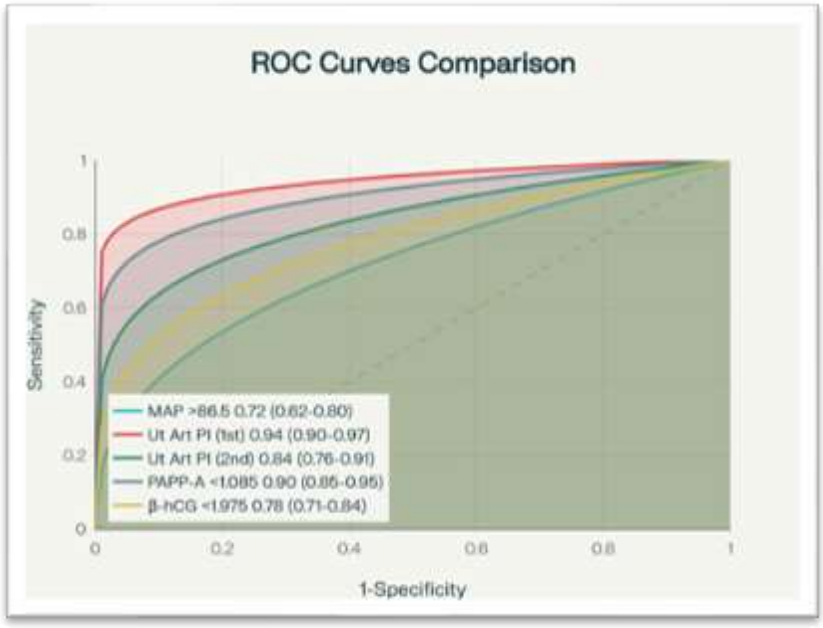

Figure 2

Table 3: Effectiveness of Aspirin in high-risk women (only among those who developed PE, n=28)

Group	Early-onset PE, n (%)	Late-onset PE, n (%)
Aspirin (n=18 PE)	3 (16.7%)	15 (83.3%)
No Aspirin (n=10 PE)	4 (40.0%)	6 (60.0%)

Table 3 evaluates the effectiveness of low-dose aspirin among women who were identified as high-risk and subsequently developed preeclampsia (n = 28). In the aspirin group (n = 18), only 3 women (16.7%) developed early-onset preeclampsia, while 15 (83.3%) had late-onset disease. In contrast, in the non-aspirin group (n = 10), 4 women (40.0%) developed early-onset preeclampsia and 6 (60.0%) developed late-onset disease.

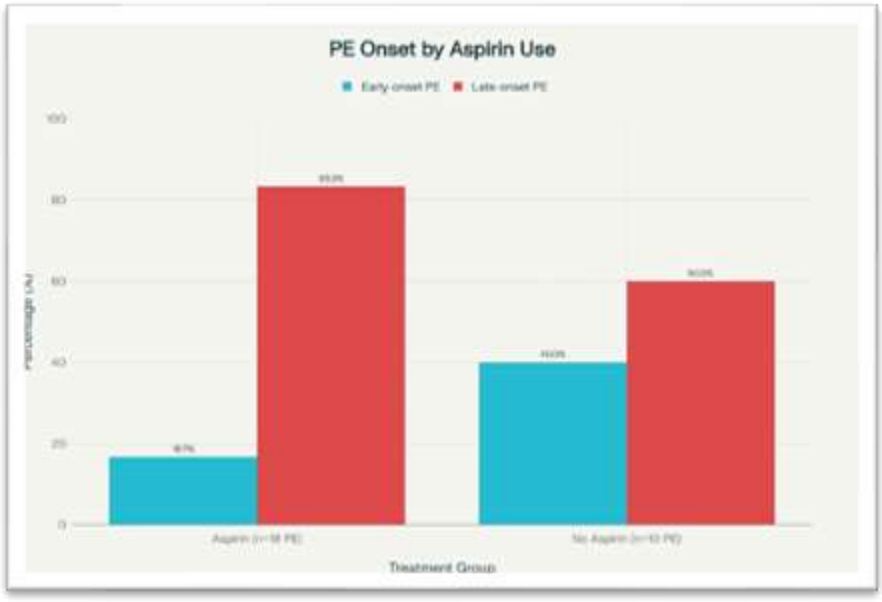

Figure 3

Table 4: Maternal outcome proxy - Gestational age at delivery (weeks)

Outcome	PE Mean (SD) [n=28]	No-PE Mean (SD) [n=372]	Mean diff (PE−NoPE)	95% CI of diff	Welch t (df≈)	p-value
Gestational age at delivery (weeks)	36.20 (2.48)	37.80 (1.62)	-1.60	-2.40 to -0.80	-4.07 (df≈33.3)	0.0003

Table 4 shows gestational age at delivery as a maternal outcome proxy. Women with preeclampsia delivered significantly earlier (mean 36.20 ± 2.48 weeks) compared with normotensive women (mean 37.80 ± 1.62 weeks). The mean difference was −1.60 weeks (95% CI: −2.40 to −0.80, p = 0.0003), highlighting the impact of preeclampsia on shortening gestational duration.

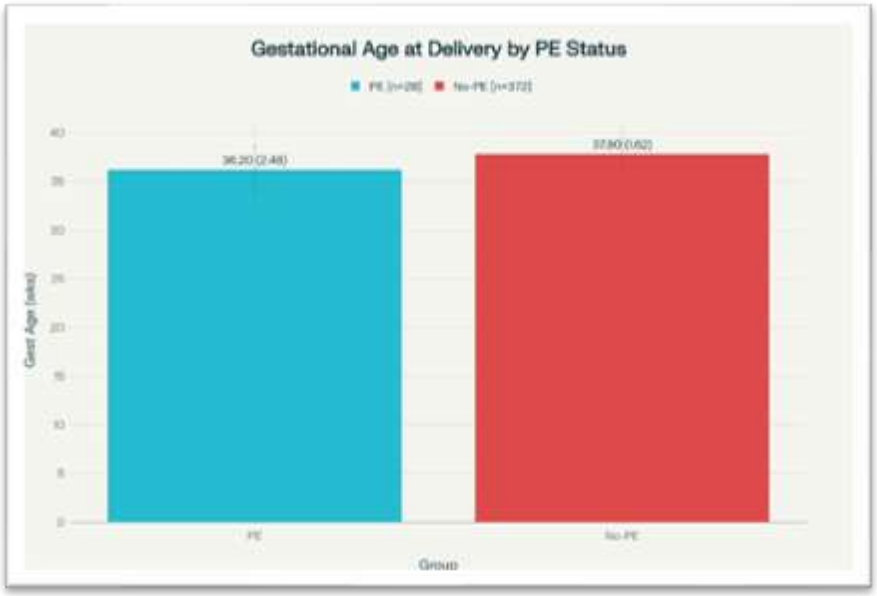

Figure 4

DISCUSSION

Hemodynamics (MAP): cohort showed higher first-trimester MAP in women who later developed PE (87.9 ± 4.04 vs 84.22 ± 5.28 mmHg; mean diff 3.68 mmHg; $p = 0.0002$), and a moderate discriminative ability (AUC 0.72; optimal cut-off 86.5 mmHg; sensitivity 64.3%, specificity 72.3%). These figures mirror the recognized role of MAP as a practical but mid-tier predictor compared with Doppler or multiparametric models. The AUC and cut-off reported in internal ROC analysis (AUC 0.72; cut-off 86.5 mmHg) are consistent with the notion that MAP alone rarely achieves high PPV at low disease prevalence, but contributes substantially when combined with other markers. Fang M *et al.*(2025)[7]

Uterine artery Doppler (first trimester): The largest single-marker signal in study came from uterine artery PI in the first trimester (mean 1.94 vs 1.44; $p<0.001$), with an ROC-derived cut-off of 1.745 yielding sensitivity 85.7% and specificity 89% (AUC 0.941). This performance exceeds the detection rates reported in early prospective series using the ≥95th percentile criterion- Jhanwar A *et al.*(2024)[8] noted sensitivity ~27% overall and ~50% for early-onset PE, while other first-trimester reports by Gomez and Pilalis found sensitivities ~24% and ~23% respectively. More recently, Tzanaki I *et al.*(2025)[9] across 974 women found first-trimester uterine artery PI >90th centile detects overall PE with sensitivity ~26% and early-onset PE ~48%. Together, these data emphasize that raw Doppler alone has limited sensitivity in general populations unless thresholds and standardization are optimized; stronger performance likely reflects a calibrated, population-specific cut-off and combination with other markers.

Uterine artery Doppler (second trimester): Second-trimester PI was also higher in the PE group (1.51 vs 1.18; $p<0.001$) with moderate diagnostic characteristics at a cut-off of 1.265 (sensitivity 71.4%, specificity 71%, AUC 0.836). This is directionally consistent with Sheetal Y *et al.*(2025)[10], who reported substantially higher PI at ~22 weeks in pregnancies that developed PE versus normal, reaffirming that abnormal impedance persists into mid-gestation in those destined for disease. team correctly notes that small differences in gestational age at measurement can shift mean PI, given the physiological decline in impedance with advancing gestation.

Biochemical markers (PAPP-A and β-hCG): finding of lower first-trimester PAPP-A in women who developed PE (1.01 vs 1.59 MoM; $p<0.001$) aligns with prior work showing that low PAPP-A reflects impaired placentation. Notably, optimal threshold (<1.085 MoM) produced sensitivity 83.9% and specificity 82.1%-higher sensitivity than the classical "<0.4 MoM (5th centile)" rule-of-thumb, which many cohorts found to be insensitive. The document collates comparative data: Nikita KP *et al.*(2024)[11] at <0.45 MoM (sens 42.1%) and Pooh RK.(2024)[12] at <0.956 MoM (sens 70%), underscoring how cohort-specific cut-off calibration can materially improve performance.

For β-hCG, cohort demonstrated lower levels in PE (1.66 vs 2.58 MoM; $p<0.001$) with modest classification (cut-off <1.975 MoM; sensitivity 66.7%, specificity 64.3%). Prior literature is mixed: some studies reported no significant first-trimester difference, whereas others noted associations in the second trimester. narrative accurately reflects this heterogeneity and concludes β-hCG should not be used as a stand-alone predictor-an interpretation consistent with earlier comparative reports.

Combined models: Combined MAP > 86.5 mmHg, PAPP-A < 1.085 MoM, and first-trimester uterine artery PI > 1.745, discrimination improved markedly (sensitivity 93.3%, specificity 96.4%, PPV 50%, NPV 99.7%, diagnostic accuracy 96.25%). This is aligned with the risk-integrated screening approach advocated by the Fetal Medicine Foundation, where maternal demographics, MAP, uterine artery PI, and biochemical markers (e.g., PAPP-A/PlGF) jointly enhance detection-especially for preterm/early-onset PE. Chen Y *et al.*(2024)[13]

Aspirin and clinical impact: In real-world subset of high-risk women who later developed PE, the distribution suggested a shift toward late-onset disease among those receiving aspirin

(early-onset 16.7% with aspirin vs 40% without), although small numbers limited statistical significance. This directionality is biologically plausible and concordant with large trials and recommendations: the CLASP trial supported low-dose aspirin in women prone to early, severe disease, and the ASPRE trial demonstrated a ~62% relative risk reduction in preterm PE using aspirin 150 mg initiated in the late first trimester within a screen-positive population. Rode L *et al.*(2025)[14] Current guideline bodies (e.g., USPSTF, SOGC) endorse low-dose aspirin for women at high risk based on clinical risk profiles; findings reinforce the value of early identification to deploy this intervention.

Downstream outcomes (gestational age at delivery): Outcome analysis demonstrates significantly earlier delivery in PE (−1.6 weeks on average), consistent with the clinical course of disease and necessity for earlier iatrogenic delivery to mitigate maternal-fetal risk. This aligns with study's summary and the broader literature that early prediction and targeted prophylaxis may attenuate severity and prolong gestation. Huang T *et al.*(2025)[15]

CONCLUSION

This prospective observational study demonstrated that abnormalities in uterine artery Doppler indices, low serum PAPP-A, low serum β-hCG, and elevated mean arterial pressure in the first trimester were significantly associated with the subsequent development of preeclampsia. Among the markers, uterine artery pulsatility index in the first trimester and PAPP-A levels showed the strongest predictive accuracy, while MAP and β-hCG contributed moderate predictive value. Importantly, a combined model incorporating maternal risk factors, MAP, uterine artery Doppler, and serum markers achieved high sensitivity and specificity, underscoring the advantage of multiparametric screening over individual predictors. The findings highlight that early risk stratification can enable timely initiation of preventive interventions such as low-dose aspirin, which may shift the disease spectrum toward milder or late-onset presentations and improve maternal and perinatal outcomes.

LIMITATIONS

1. The study was conducted at a single tertiary-care center, which may limit the generalizability of findings to different populations and healthcare settings.
2. The sample of preeclampsia cases (n = 28) was relatively small, reflecting the natural prevalence of the disease, and may have limited the power for subgroup analysis (e.g., early- vs late-onset PE).
3. Serum biomarkers (PAPP-A, β-hCG) were assessed using a single commercial assay; inter-laboratory variability may affect cut-off generalizability.
4. Aspirin prophylaxis was administered to women identified as high-risk, introducing potential confounding by indication in evaluating its effect on disease severity.
5. The study did not include other promising biomarkers such as placental growth factor (PlGF) or soluble fms-like tyrosine kinase-1 (sFlt-1), which might further improve predictive accuracy.
6. Long-term maternal cardiovascular outcomes were not evaluated, though these represent an important dimension of preeclampsia's impact.

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