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Comparison of Mean Uterine Artery PI between Women at High Risk for both Preeclampsia & Fetal Growth Restriction & those at High Risk for either Condition Alone

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HIGHLIGHTS

- Combined risk had highest PI
- PI increased with overlapping risk
- Pairwise differences were significant
- Combined risk showed higher PE
- Doppler enabled early risk stratification

Key Words:

Doppler ultrasonography
Placental insufficiency
First-trimester screening
Pregnancy complications
Impedance
Risk stratification



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ABSTRACT

Introduction: Preeclampsia (PE) and fetal growth restriction (FGR) share common pathophysiological mechanisms, including defective deep placentation, impaired spiral artery remodeling, and increased uteroplacental vascular impedance. Whether their coexistence produces a distinct first-trimester maternal haemodynamic signature remains uncertain. **Aim & Objective:** To compare first-trimester mean uterine artery pulsatility index (PI) among women at high risk for PE alone, FGR alone, and both conditions combined. **Materials & Methods:** This prospective observational comparative study included 150 singleton pregnancies at 11–13⁺6 weeks. Women were stratified using the Fetal Medicine Foundation risk cut-off of 1:150 into three equal groups of 50 (33.3%) each: PE risk, FGR risk, and combined PE+FGR risk. Transabdominal Doppler assessment was performed to derive the mean PI from both uterine arteries. Groups were compared using one-way analysis of variance with post-hoc testing and the chi-square test. **Results:** Mean uterine artery PI was highest in the combined-risk group (2.81 ± 0.16), followed by the PE-only (2.34 ± 0.09) and FGR-only (2.04 ± 0.10) groups [$F(2,147) = 517.05$, $p < 0.001$], with all pairwise differences statistically significant. PE developed in 32 (64.0%) women with combined risk versus 26 (52.0%) with PE risk alone, while FGR occurred in 35 (70.0%) versus 22 (44.0%), respectively. **Conclusions:** First-trimester mean uterine artery PI increased progressively with overlapping placental risk, with the highest impedance observed when PE and FGR risks coexisted. These findings support uterine artery Doppler as a potential tool for early risk stratification of adverse placental outcomes

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INTRODUCTION

Preeclampsia and fetal growth restriction remain leading contributors to maternal and perinatal morbidity and mortality worldwide, and both are rooted in defective transformation of the maternal spiral arteries during early placentation [1]. When trophoblastic invasion fails to convert these vessels from high- to low-resistance channels, uteroplacental impedance stays elevated, a disturbance that can be detected in the uterine arteries weeks before disease becomes clinical [2]. Growth restriction is conventionally divided into an early-onset form (before 32 weeks), dominated by severe placental disease and abnormal multivessel Doppler, and a more prevalent late-onset form in which the fetus may not appear small and cerebral redistribution predominates [1].

The uterine artery pulsatility index measured at 11–13⁺ weeks has become a central biophysical marker of this process, because a raised index reflects the impaired remodelling that underlies both disorders [2,3]. Its predictive performance is strongest when combined with maternal characteristics and biochemical markers such as pregnancy-associated plasma protein-A and placental growth factor, an approach that anchors current first-trimester screening algorithms [4,5]. Reported performance is nonetheless consistently higher for preeclampsia than for isolated growth restriction, in keeping with the more systemic maternal endothelial component of the former [6,7].

Where the two conditions coexist, placental dysfunction is presumed to be more profound, and several cohorts have observed correspondingly higher impedance in pregnancies affected by both disorders than in those affected by either alone [5,8]. However, most evidence derives from outcome-defined cohorts rather than from prospectively risk-stratified groups, and few studies have directly contrasted the mean pulsatility index of

women flagged as high risk for both conditions against those flagged for a single condition. Clarifying whether combined risk carries an incrementally higher index would refine antenatal surveillance intensity, inform counselling, and indicate whether overlapping pathology represents an additive process or a distinct entity. The present study was undertaken to address this gap. **Figure 1.** Mechanism of placental dysfunction in preeclampsia and fetal growth restriction.

The primary objective was to measure and compare the first-trimester mean uterine artery pulsatility index at 11–13⁺ weeks of gestation among 150 women stratified into three equal high-risk groups (preeclampsia only, fetal growth restriction only, and both conditions) over an 18-month period. The secondary objective was to determine whether the combined-risk group demonstrated a significantly higher mean pulsatility index than each single-risk group, thereby testing the pulsatility index as an early marker of overlapping placental disease.

MATERIALS & METHODS

Study design and setting

A prospective, observational, comparative study was conducted in the Department of Radio-Diagnosis, Maharani Laxmi Bai Medical College, Jhansi, over 18 months (November 2024 to October 2026). The study design was defined first because it frames the approach used to answer the research question and to interpret the results that follow.

Participants and eligibility criteria

A total of 150 pregnant women attending the first-trimester antenatal clinic were enrolled after written informed consent. Eligible women had a singleton live pregnancy with a crown-

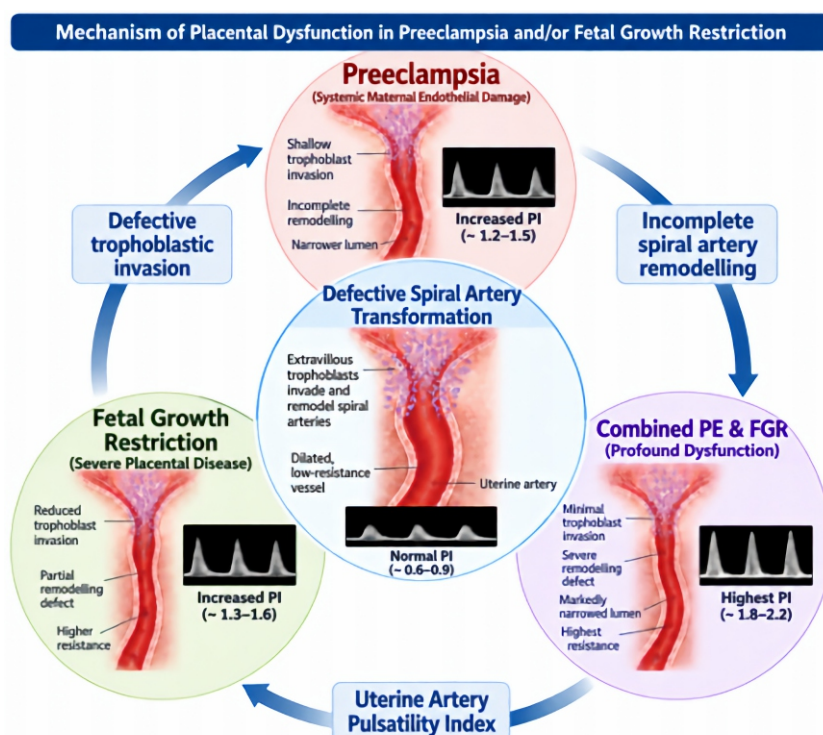


Figure 1: Mechanism of placental dysfunction in preeclampsia and fetal growth restriction.

rump length of 45–84 mm corresponding to 11–13⁺6 weeks, and a Fetal Medicine Foundation risk exceeding the 1:150 cut-off on the respective risk calculators for preeclampsia and for fetal growth restriction. Multiple pregnancies, gestational age outside the 11–13⁺6-week window, crown-rump length above 84 mm, non-viable fetuses, congenital anomalies, and women who declined consent were excluded. Applying these criteria, participants were assigned to three equal groups of 50 each: high risk for preeclampsia only, high risk for fetal growth restriction only, and high risk for both conditions.

Ethics

The study was approved by the Institute Ethics Committee (Human Studies), M.L.B. Medical College, Jhansi (Sr. No. 5484/IEC/23/2025/SC-1; approved 30 April 2025) and was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from every participant, and no personally identifying information was recorded in the analytic dataset.

Data collection and measurements

A structured proforma was used to record demographic data, obstetric and medical history, and established risk factors. Maternal blood pressure was measured in both arms with a calibrated sphygmomanometer, two readings were taken per arm, and the mean arterial pressure was calculated. Maternal

serum was collected for pregnancy-associated plasma protein-A and placental growth factor, which fed the Fetal Medicine Foundation risk estimate. Uterine artery Doppler was performed transabdominally on a GE Vivid T8 and a Konica Aeroscan system. In a mid-sagittal view of the uterus and cervix, colour Doppler identified each uterine artery at the cervico-uterine junction; pulsed-wave Doppler was then applied with a sampling gate of approximately 2 mm and an insonation angle below 30°. After at least three uniform consecutive waveforms had been obtained, the right and left pulsatility indices were recorded and the mean uterine artery pulsatility index was calculated as their average (**Figures 2–4**). Because this is a well-established, standardised technique, it was described in brief rather than in full protocol detail.

Statistical analysis

Data were entered in Microsoft Excel and analysed in SPSS version 21.0. Continuous variables were summarised as mean $\pm$ standard deviation and categorical variables as frequency and percentage in n (%) format. After normality was confirmed, the three groups were compared for continuous variables using one-way analysis of variance, with the F statistic and, where the overall test was significant, Tukey post-hoc pairwise comparisons reported with 95% confidence intervals. Categorical variables were compared using the Pearson chi-square test. All tests were two-tailed and a p-value below 0.05 was considered statistically significant.



Figure 2. Normal first-trimester uterine artery Doppler at a crown-rump length of 11 weeks 4 days, showing colour flow at the cervico-uterine junction and a normal pulsatility index below the 95th centile.

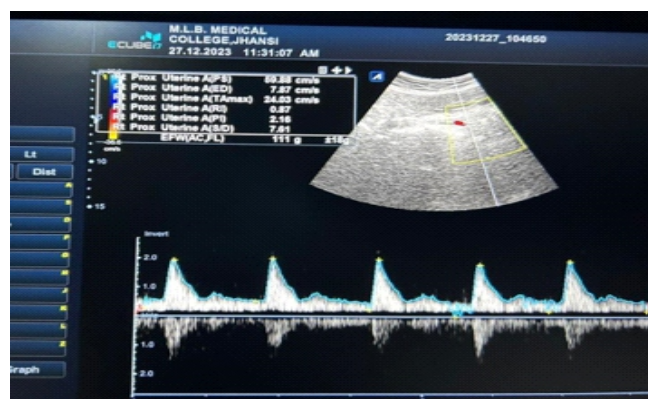


Figure 3. Raised first-trimester uterine artery Doppler at a crown-rump length of 12 weeks 6 days, showing an elevated pulsatility index above the 95th centile with an early diastolic notch.

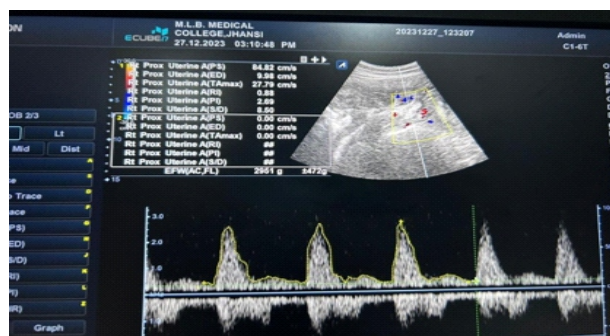


Figure 4. Raised first-trimester uterine artery Doppler at a crown-rump length of 12 weeks 4 days, showing an elevated pulsatility index above the 95th centile.

RESULTS

A total of 150 women were analysed in three equal groups of 50 (33.3%) each - high risk for preeclampsia only, high risk for fetal growth restriction only, and high risk for both conditions (**Table 1**). The 25–29-year band was the commonest overall, accounting for 65 (43.3%) women, but the age structure differed markedly across groups ($\chi^2 = 40.76$, $df = 8$, $p < 0.001$): most single-risk women were aged 25–29 years [24 (48.0%) preeclampsia-only and 21 (42.0%) growth-restriction-only], whereas the combined-risk group clustered at 30–34 years [25 (50.0%)]. Only 2 (1.3%) women were younger than 20 years.

Maternal demographic and clinical characteristics diverged significantly across the three groups (**Table 2**). The combined-risk group was older (30.76 ± 2.85 years) than the preeclampsia-only (26.24 ± 4.15) and growth-restriction-only (25.48 ± 3.53) groups [$F(2,147) = 32.33$, $p < 0.001$], and had the highest body mass index (29.33 ± 1.66 kg/m²; $F(2,147) = 84.43$, $p < 0.001$), gravity (3.94 ± 0.87 ; $F(2,147) = 47.86$, $p < 0.001$), and mean arterial pressure (114.88 ± 4.90 mmHg; $F(2,147) = 119.57$, $p < 0.001$). These findings indicate a clustering of maternal cardiometabolic risk in women carrying dual placental risk.

The primary endpoint confirmed a stepwise gradient in impedance (**Table 3**). The mean uterine artery pulsatility index was highest in the combined-risk group (2.81 ± 0.16), intermediate in the preeclampsia-only group (2.34 ± 0.09), and lowest in the growth-restriction-only group (2.04 ± 0.10), with a very large between-group effect [$F(2,147) = 517.05$, $p < 0.001$]. The same ordering held for the right (2.83 ± 0.18 vs 2.36 ± 0.10 vs 2.05 ± 0.12 ; $F(2,147) = 407.31$, $p < 0.001$) and left (2.79 ± 0.15 vs 2.33 ± 0.11 vs 2.02 ± 0.12 ; $F(2,147) = 459.49$, $p < 0.001$) arteries. Every woman in the preeclampsia-only and combined groups

had an index above the 95th centile, whereas all growth-restriction-only women had values below that threshold.

Tukey post-hoc analysis showed that all three pairwise contrasts were significant (**Table 4**). The mean index was 0.30 higher in the preeclampsia-only group than in the growth-restriction-only group (95% CI 0.26–0.34), 0.47 lower in the preeclampsia-only group than in the combined group (95% CI 0.42–0.52), and the widest gap, 0.77, separated the growth-restriction-only and combined groups (95% CI 0.72–0.82); all $p < 0.001$. Impedance therefore increased incrementally with the accumulation of placental risk rather than in a single step. Maternal and neonatal outcomes, stratified by aspirin use, are shown in **Table 5**. Low-dose aspirin was started in 52 (34.7%) women. Preeclampsia developed most often in the combined-risk group [32 (64.0%)], followed by the preeclampsia-only group [26 (52.0%)], with none in the growth-restriction-only group ($\chi^2 = 48.80$, $df = 2$, $p < 0.001$). Fetal growth restriction was likewise commonest in the combined group [35 (70.0%)] and next in the growth-restriction-only group [22 (44.0%)] ($\chi^2 = 53.14$, $df = 2$, $p < 0.001$). Aspirin was associated with a lower incidence of preeclampsia in the preeclampsia-only group [4/16 (25.0%) vs 22/34 (64.7%); $\chi^2 = 5.37$, $p = 0.020$], with a similar but non-significant direction in the combined group [9/19 (47.4%) vs 23/31 (74.2%); $\chi^2 = 2.61$, $p = 0.106$]. A comparable non-significant reduction in growth restriction was seen with aspirin in the growth-restriction-only [5/17 (29.4%) vs 17/33 (51.5%); $\chi^2 = 1.42$, $p = 0.234$] and combined [12/19 (63.2%) vs 23/31 (74.2%); $\chi^2 = 0.26$, $p = 0.611$] groups. Mean birth weight fell progressively from the preeclampsia-only (2750 ± 310 g) to the growth-restriction-only (2280 ± 450 g) and combined (1890 ± 520 g) groups [$F(2,147) = 48.88$, $p < 0.001$].

Table 1. Distribution of participants by risk category and maternal age group, n (%)

Age group (years)	PE only (n=50)	FGR only (n=50)	PE + FGR (n=50)	Total (n=150)	p-value
< 20	1 (2.0%)	1 (2.0%)	0 (0%)	2 (1.3%)	< 0.001*
20–24	15 (30.0%)	20 (40.0%)	0 (0%)	35 (23.3%)	
25–29	24 (48.0%)	21 (42.0%)	20 (40.0%)	65 (43.3%)	
30–34	6 (12.0%)	6 (12.0%)	25 (50.0%)	37 (24.7%)	
≥ 35	4 (8.0%)	2 (4.0%)	5 (10.0%)	11 (7.3%)	
Total	50 (100%)	50 (100%)	50 (100%)	150 (100%)	

PE, preeclampsia; FGR, fetal growth restriction. *Pearson chi-square test, $\chi^2 = 40.76$, $df = 8$.

Table 2. Comparison of maternal demographic and clinical characteristics among the three risk groups (mean $\pm$ SD)

Variable	PE only (n=50)	FGR only (n=50)	PE + FGR (n=50)	F (df 2,147)	p-value
Maternal age (years)	26.24 $\pm$ 4.15	25.48 $\pm$ 3.53	30.76 $\pm$ 2.85	32.33	< 0.001
BMI (kg/m ²)	23.71 $\pm$ 1.72	24.60 $\pm$ 3.24	29.33 $\pm$ 1.66	84.43	< 0.001
Gravidity	2.46 $\pm$ 1.01	2.52 $\pm$ 0.65	3.94 $\pm$ 0.87	47.86	< 0.001
MAP (mmHg)	100.26 $\pm$ 10.35	94.26 $\pm$ 3.16	114.88 $\pm$ 4.90	119.57	< 0.001

BMI, body mass index; MAP, mean arterial pressure; SD, standard deviation. One-way analysis of variance.

Table 3. Comparison of uterine artery Doppler parameters among the three risk groups (mean $\pm$ SD)

Doppler parameter	PE only (n=50)	FGR only (n=50)	PE + FGR (n=50)	F (df 2,147)	p-value
Right UtA-PI	2.36 $\pm$ 0.10	2.05 $\pm$ 0.12	2.83 $\pm$ 0.18	407.31	< 0.001
Left UtA-PI	2.33 $\pm$ 0.11	2.02 $\pm$ 0.12	2.79 $\pm$ 0.15	459.49	< 0.001
Mean UtA-PI	2.34 $\pm$ 0.09	2.04 $\pm$ 0.10	2.81 $\pm$ 0.16	517.05	< 0.001

UtA-PI, uterine artery pulsatility index. One-way analysis of variance.

Table 4. Post-hoc pairwise comparison of mean uterine artery pulsatility index between risk groups

Comparison	Mean difference	95% CI	p-value
PE only vs FGR only	0.30	0.26 – 0.34	< 0.001
PE only vs PE + FGR	0.47	0.42 – 0.52	< 0.001
FGR only vs PE + FGR	0.77	0.72 – 0.82	< 0.001

CI, confidence interval. Tukey honestly significant difference test.

Table 5. Maternal and neonatal outcomes stratified by aspirin use, n (%)

Outcome/parameter	PE only (n=50)	FGR only (n=50)	PE + FGR (n=50)	p-value
Aspirin initiated	16 (32.0%)	17 (34.0%)	19 (38.0%)	—
Developed preeclampsia	26 (52.0%)	0 (0%)	32 (64.0%)	< 0.001 [†]
— in aspirin group	4/16 (25.0%)	0 (0%)	9/19 (47.4%)	
— in non-aspirin group	22/34 (64.7%)	0 (0%)	23/31 (74.2%)	
Developed FGR	0 (0%)	22 (44.0%)	35 (70.0%)	< 0.001 [‡]
— in aspirin group	0 (0%)	5/17 (29.4%)	12/19 (63.2%)	
— in non-aspirin group	0 (0%)	17/33 (51.5%)	23/31 (74.2%)	
Mean birth weight (g)	2750 $\pm$ 310	2280 $\pm$ 450	1890 $\pm$ 520	< 0.001 [§]

[†] $\chi^2 = 48.80$, $df = 2$ (preeclampsia across groups). [‡] $\chi^2 = 53.14$, $df = 2$ (FGR across groups). [§]One-way ANOVA, $F(2,147) = 48.88$. Aspirin vs preeclampsia: PE-only $\chi^2 = 5.37$, $p = 0.020$; PE+FGR $\chi^2 = 2.61$, $p = 0.106$. Aspirin vs FGR: FGR-only $\chi^2 = 1.42$, $p = 0.234$; PE+FGR $\chi^2 = 0.26$, $p = 0.611$.

DISCUSSION

This prospective comparison shows that the first-trimester mean uterine artery pulsatility index rises in a graded fashion across risk categories, reaching its highest value in women flagged as high risk for both preeclampsia and fetal growth restriction. The very large F statistic (517.05) and the significance of every pairwise contrast argue that this is not a marginal difference but a robust separation, consistent with the interpretation that overlapping risk corresponds to more severe defective placentation rather than to the simple summation of two independent processes.

The most plausible explanation lies in the shared upstream mechanism. Both disorders originate in incomplete trophoblastic conversion of the spiral arteries, which keeps uteroplacental impedance high and is transmitted upstream as an elevated pulsatility index [2,3]. When invasion is severely deficient, both fetal nutrient delivery and maternal endothelial signalling are compromised, so the two syndromes co-manifest and impedance is correspondingly greater. This gradient is coherent with the

reference literature: Springer and colleagues reported higher first-trimester impedance in growth-restricted pregnancies than in normal ones (1.82 ± 0.70 vs 1.55 ± 0.47) [6], and Siricharoenthai and Phupong found a higher index in early-onset preeclampsia than in controls (2.3 ± 0.5 vs 1.7 ± 0.5) [9]. Our combined-risk mean of 2.81 sits above both, in line with the expectation that dual pathology carries the greatest impedance.

The clustering of older age, higher body mass index, greater gravidity, and higher mean arterial pressure in the combined-risk group mirrors established maternal determinants of placental disease and helps explain why these women occupied the highest impedance stratum [5,10]. The finding that every preeclampsia-risk and combined-risk woman exceeded the 95th-centile index, while all isolated growth-restriction-risk women fell below it, reinforces the recognised observation that first-trimester uterine artery Doppler discriminates preeclampsia more sharply than isolated growth restriction [11,7]. This asymmetry probably reflects the stronger systemic maternal endothelial component of preeclampsia, whereas late-onset growth restriction may arise

from subtler placental compromise that becomes evident only as fetal demand increases [1].

Our diagnostic direction agrees with recent screening cohorts. Parry and colleagues reported that a first-trimester index predicted preeclampsia with a sensitivity of 75% and specificity of 86% [11], and Lee and colleagues found that a first-trimester uterine artery notch carried a high adjusted odds ratio for preeclampsia (8.65) alongside an excellent negative predictive value (99.6%) [12]. Large-cohort and meta-analytic data similarly link a raised index to preterm preeclampsia and to small-for-gestational-age birth [8,7]. Increasingly, the index is combined with circulating markers - including macrophage-stimulating protein, high-temperature-requirement protease A4, hypoxia-inducible factor-1 α , fatty-acid-binding protein-4, and SerpinA5 - to lift predictive accuracy beyond that of either component alone [13-17], and multimodal algorithms of this kind now underpin routine first-trimester practice [4,5].

The aspirin observations, although secondary, are internally coherent. Preeclampsia and growth restriction were consistently less frequent among aspirin-treated women, and the reduction reached significance for preeclampsia in the preeclampsia-only group. The non-significant effect in the combined-risk group is unsurprising: this study was not powered for stratified outcome analysis, and the residual risk in women with dual pathology is known to be substantial. This pattern is consistent with the randomised evidence, in which aspirin selected purely on uterine artery Doppler criteria did not reduce the composite of preeclampsia and low birth weight [18], and with routine-care cohorts reporting only modest algorithm-based detection of early preeclampsia [5]; together these data indicate that prophylaxis is best guided by multimodal risk rather than impedance alone.

Beyond the incidence figures, the progressively lower birth weight across the groups foreshadows a more demanding neonatal course for dual-risk pregnancies, in which growth-restricted infants are prone to hypoglycaemia, hypocalcaemia, polycythaemia, impaired thermoregulation, and respiratory compromise [1]. Early identification of the highest-impedance pregnancies therefore has value not only for maternal surveillance but also for anticipatory neonatal planning.

Strength and Limitations The strengths of this work include a prospective design, a standardised transabdominal Doppler protocol with strict insonation criteria, balanced groups of equal size that minimised sampling bias, and the reporting of exact test statistics for every comparison. Several limitations temper the findings. It was a single-centre study with 150 participants, which limits generalisability and statistical power for the stratified aspirin sub-analyses; the groups were defined by first-trimester risk rather than by confirmed final diagnosis, so some risk-based misclassification is possible; several categorical cells were small, so the age-distribution chi-square should be read cautiously; aspirin was not randomised, leaving its apparent benefit open to confounding by indication; and biochemical markers were used only for risk assignment rather than analysed as predictors. Multicentre studies with larger samples and standardised,

outcome-anchored cut-offs are needed to confirm these results and to define impedance thresholds for combined placental disease.

CONCLUSION

First-trimester mean uterine artery pulsatility index differs significantly across high-risk groups and is highest when preeclampsia and fetal growth restriction risks coexist, marking the most severe placental impedance. Measured at 11–13⁺⁶ weeks, it is a simple, non-invasive tool for early risk stratification that can direct intensified surveillance and timely preventive care. Integrating it with maternal characteristics and biochemical markers, and validating impedance thresholds in larger multicentre cohorts, should be the next step.

LIMITATIONS & FUTURE PERSPECTIVES

The study's limitations include a single-centre setting, a relatively small sample size, and a short study duration, which may limit the broader applicability of the results. Future studies should incorporate multi-centre designs with larger populations to enhance validity, assess long-term outcomes, and investigate advanced diagnostic & management approaches. Such efforts will improve overall patient care & help minimize complications.

CLINICAL SIGNIFICANCE

The clinical significance of this study lies in its potential to bridge the gap between research findings and practical health-care, diagnosis, and treatment outcomes. By highlighting real-care applications. It emphasizes the importance of translating scientific observations into meaningful improvements in patient world relevance, the study contributes to evidence based medical practice and supports informed clinical decision making. Ultimately, the findings aim to enhance patient quality of life, optimize therapeutic strategies, and promote better disease management in clinical settings.

ABBREVIATIONS

PE: Preeclampsia

FGR: Fetal Growth Restriction

PI: Pulsatility Index

FMF: Fetal Medicine Foundation

UA: Uterine Artery

Doppler: Doppler Ultrasonography

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AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They

participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by the International Committee of Medical Journal Editors (ICMJE) guidelines.

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CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

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All necessary consent & approval was obtained by authors.

CONSENT FOR PUBLICATION

All necessary consent for publication was obtained by authors.

DATA AVAILABILITY

All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a reasonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)

The authors confirm that no AI & LLM tools were used in the writing or editing of the manuscript, and no images were altered or manipulated using AI & LLM.

AUTHOR'S NOTE

This article serves as an important educational tool for the scientific community, offering insights that may inspire future research directions. However, they should not be relied upon independently when making treatment decisions or developing public health policies.

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