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Evaluation of Platelet and Coagulation Profiles Among Pregnant Women with Preeclampsia Attending Antenatal Clinics in Osun State, Nigeria.

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ABSTRACT

Background: Hypertensive disorders of pregnancy, especially preeclampsia and eclampsia, significantly contribute to maternal and perinatal morbidity and mortality in low-resource settings. This study assessed platelet function and coagulation abnormalities among pregnant women with preeclampsia and eclampsia attending antenatal clinics in Osun State, Nigeria. **Methods:** A hospital-based comparative cross-sectional study involving 210 pregnant women (70 normotensive, 70 with preeclampsia, and 70 with eclampsia) was conducted. Sociodemographic and clinical data were collected using structured questionnaires, while laboratory analyses evaluated platelet indices and coagulation parameters. Data were analysed using SPSS version 26.0, with statistical significance defined at $p < 0.05$. **Results:** Demographic characteristics were comparable across groups, except for parity, which was significantly higher among women with eclampsia ($p = 0.038$). Platelet counts decreased significantly with disease severity ($240 \pm 44 \times 10^9$/L in normotensive women, $174 \pm 40 \times 10^9$/L in preeclampsia, and $129 \pm 35 \times 10^9$/L in eclampsia; $p < 0.001$). Conversely, MPV and PDW increased progressively across the groups ($p < 0.001$). Bleeding time, PT, and aPTT were significantly prolonged among women with hypertensive disorders of pregnancy, with the greatest abnormalities observed in eclampsia ($p < 0.001$). Fibrinogen and D-dimer levels also showed significant stepwise increases with disease severity ($p < 0.001$). Multivariate analysis identified thrombocytopenia and elevated MPV as independent predictors of both preeclampsia and eclampsia, while prolonged PT and aPTT were independently associated with eclampsia. **Conclusion:** Preeclampsia and eclampsia are associated with significant alterations in platelet function and coagulation parameters, reflecting progressive haemostatic imbalance. Incorporating routine haemostatic assessment into antenatal care may facilitate early identification of disease severity and improve maternal outcomes.

Keywords: Preeclampsia, Eclampsia, Platelet indices, Coagulation profile, Pregnancy, Nigeria.

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INTRODUCTION

Hypertensive disorders of pregnancy complicate approximately 5–10% of all pregnancies worldwide and remain a major cause of maternal and perinatal morbidity and mortality, particularly in low- and middle-income countries. Globally, these disorders account for nearly 16% of maternal deaths, underscoring their significant contribution to adverse pregnancy outcomes (Afolayan et al*.,* 2023; Tadu et al*.,* 2023; WHO, 2025). Pregnancy-induced hypertension, defined as new-onset hypertension occurring after 20 weeks of gestation and resolving postpartum, represents a major component of this burden (Bhutani et al*.,* 2022; Alemu et al*.,* 2025). In sub-Saharan Africa, including Nigeria, the impact of preeclampsia and eclampsia is disproportionately high due to delayed antenatal booking, inadequate laboratory infrastructure, and limited access to specialised obstetric care.

Preeclampsia is a multisystem disorder characterised by new-onset hypertension accompanied by proteinuria and/or evidence of maternal organ dysfunction. Clinically, it is defined by systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg after 20 weeks of gestation and may involve complications affecting the kidneys, liver, lungs, cardiovascular system, and central nervous system. Eclampsia represents the most severe end of the disease spectrum and is defined by the occurrence of seizures in a woman with preeclampsia (Sami et al*.,* 2022; Udeh et al*.,* 2024; Chikezie et al*.,* 2025; WHO, 2025). Together, these conditions are responsible for over 70,000 maternal deaths and approximately 500,000 fetal and neonatal deaths annually worldwide.

The underlying pathophysiology of hypertensive disorders of pregnancy involves widespread endothelial dysfunction accompanied by dysregulation of the haemostatic system. Endothelial injury promotes platelet activation and the coagulation cascade, resulting in a hypercoagulable state that paradoxically increases the risk of both thrombosis and bleeding complications, including postpartum haemorrhage (Haldar & Barui, 2020; Tadu et al*.,* 2023). Early activation and consumption of coagulation factors may progress to systemic metabolic disturbances and multiple organ dysfunction, posing serious risks to both mother and foetus (Sami et al*.,* 2022; Udeh et al*.,* 2024).

Normal pregnancy itself is associated with significant physiological adaptations in haemostasis that favour coagulation as a protective mechanism against excessive blood loss during delivery. These changes include increased levels of clotting factors, reduced fibrinolytic activity, and alterations in platelet count and function (Papageorgiou et al*.,* 2017; Patel et al*.,* 2025). Platelets play a central role in primary haemostasis, and platelet indices such as platelet count, mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), and platelet large cell ratio (P-LCR) have increasingly been recognised as useful indicators of platelet activation and turnover. Similarly, coagulation parameters, including fibrinogen concentration, prothrombin time (PT), and activated partial thromboplastin time (aPTT), provide valuable insight into the functional integrity of the coagulation cascade (McLean et al*.,* 2021; Ali, 2025).

Haematological abnormalities, particularly thrombocytopenia and deranged coagulation profiles, are frequently observed in women with hypertensive disorders of pregnancy and are considered important indicators of disease severity (Haldar & Barui, 2020; Alemu et al*.,*

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2025). Previous studies have demonstrated significant alterations in platelet indices, especially MPV and PDW, among affected women, suggesting their potential utility as accessible biomarkers for early detection and disease monitoring (Lin et al*.,* 2023; Udeh et al*.,* 2024; Tokgöz Çakır et al*.,* 2025). Lin et al. (2023) highlight their added predictive value when combined with clinical factors, while Udeh et al. (2024) demonstrate their strength as independent predictors, particularly for early-onset cases. In contrast, Tokgöz Çakır et al. (2025) link these indices with inflammatory markers, suggesting they reflect broader disease processes. Overall, while all three studies support the clinical relevance of MPV and PDW, the evidence suggests that their greatest utility may lie not in isolation but in a composite predictive or diagnostic model, particularly for improving early detection and monitoring of preeclampsia.

However, small sample sizes and cross-sectional designs limit many existing studies, which evaluate haemostatic changes only after disease onset, thereby reducing their predictive value (Udeh et al., 2024). Consequently, further investigation is required to establish reliable diagnostic thresholds and improve clinical risk stratification, particularly within local populations (Bhutani et al*.,* 2022; Sami et al*.,* 2022).

Evidence from Nigeria indicates that hypertensive disorders of pregnancy remain common and are frequently associated with significant alterations in platelet and coagulation parameters, though the focus differs across studies (Ajibola et al*.,* 2020; Egbe et al*.,* 2021; Umezuluike et al*.,* 2021; Udeh et al*.,* 2024; Moore-Igwe & Wenah-Emmanuel, 2025). Ajibola et al. (2014) reported gestational thrombocytopenia, highlighting reduced platelet counts even in otherwise normal pregnancies, suggesting an underlying baseline shift. In contrast, Egbe et al. (2021) identified broader haematological changes in preeclamptic patients, indicating more pronounced disease-related abnormalities. Umezuluike et al. (2021) went further, linking altered platelet parameters to adverse maternal and neonatal outcomes, emphasising their prognostic significance. More recent studies refine this perspective; Udeh et al. (2024) demonstrated the predictive value of MPV and PDW for early-onset preeclampsia, while Moore-Igwe & Wenah-Emmanuel (2025) highlighted significant coagulation disturbances, underscoring the role of haemostatic dysfunction in disease progression. Overall, while all studies confirm platelet and coagulation abnormalities, they differ in emphasis—ranging from baseline changes to diagnostic, predictive, and prognostic roles—suggesting these parameters are most useful when integrated across multiple clinical contexts. Nevertheless, most available studies have evaluated general obstetric populations, with limited data specifically examining platelet function and coagulation profiles among women with preeclampsia and eclampsia at sub-national levels. Regional differences in genetic background, environmental exposures, nutritional status, healthcare access, and lifestyle factors further emphasise the importance of locally generated evidence.

Despite the recognised clinical relevance of haemostatic abnormalities, routine assessment of platelet function and coagulation parameters is not consistently incorporated into antenatal care in many Nigerian healthcare settings. Furthermore, data describing haemostatic alterations among women with hypertensive disorders of pregnancy remain scarce in Southwestern Nigeria. Therefore, this study aimed to evaluate platelet function and coagulation profiles among pregnant women with preeclampsia and eclampsia attending antenatal

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clinics in Osun State, Nigeria, to improve early detection, risk stratification, and clinical management of hypertensive disorders of pregnancy.

METHODOLOGY

Study Design

This study was a hospital-based, comparative cross-sectional investigation conducted between January and April 2025 in selected tertiary and secondary healthcare facilities providing antenatal services in Osogbo, Ede, and Ilesa, Osun State, Southwestern Nigeria. These facilities serve diverse urban and semi-urban populations and provide comprehensive maternal healthcare services, including routine antenatal care, obstetric management, and laboratory support.

Study Population

The study population comprised pregnant women aged 18–45 years who attended antenatal clinics during the study period. Participants included women diagnosed with preeclampsia and eclampsia, as well as normotensive pregnant controls. Eligible participants were recruited during routine antenatal visits and followed through delivery to enable assessment of clinical outcomes associated with hypertensive disorders of pregnancy.

Ethical Considerations

Ethical approval for the study was obtained from the Osun State Health Research Ethics Committee (OSHREC/PRS/569T/1322). Written informed consent was obtained from all participants before enrolment. Confidentiality of participant information was strictly maintained, and all data were used solely for research purposes.

Sample Size and Grouping

A total of 210 participants were enrolled in the study. The sample size was calculated using a standard formula for case–control studies (Varkivisser et al*.,* 2003).

$n = (Z_\alpha + Z_\beta)^2 (p_1q_1+p_2q_2)/(p_1-p_2)^2$, where Z_α and Z_β correspond to the selected significance level and study power, $q = 1–p$, and p_1 and p_2 represent the exposure proportions in cases and controls, respectively.

Participants were equally categorised into three groups: normotensive pregnant women (n = 70), pregnant women with preeclampsia (n = 70), and pregnant women with eclampsia (n = 70).

Inclusion and Exclusion Criteria

Women with singleton pregnancies at a gestational age of 20 weeks or more were eligible for inclusion. Participants were excluded if they had a history of chronic hypertension, diabetes mellitus, liver disease, known bleeding disorders, multiple gestation, or current use of anticoagulant therapy, as these conditions could influence haemostatic parameters.

Data Collection

Sociodemographic and clinical information were obtained using a structured questionnaire. Data collected included maternal age, gestational age, parity, body mass index (BMI), and blood pressure measurements. Body mass index was calculated as weight in kilograms divided by the square of height in metres (kg/m^2).

Clinical Assessment and Laboratory Procedures

Blood pressure was measured using a calibrated mercury sphygmomanometer. Participants were positioned comfortably in a seated or supine

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position with the arm supported at heart level to ensure accurate readings. A second measurement was obtained after 4–6 hours. Hypertension was classified as mild when blood pressure was ≥140/90 mmHg and severe when readings were ≥160/110 mmHg.

Urine samples were collected either as clean-catch midstream specimens or via catheterisation when indicated. Urinalysis was performed using Combi-2 reagent dipsticks, and proteinuria was recorded semi-quantitatively as negative, trace, 1+ (30 mg/dL), 2+ (100 mg/dL), 3+ (300–1999 mg/dL), or 4+ (>2 g).

For laboratory analysis, 5 mL of venous blood was collected aseptically from each participant. Two millilitres were dispensed into ethylenediaminetetraacetic acid (EDTA) tubes for platelet analysis, while 3 mL were collected into sodium citrate tubes for coagulation studies. Platelet count, mean platelet volume (MPV), and platelet distribution width (PDW) were analysed using an automated haematology analyser. Bleeding time was assessed using the Ivy method. Prothrombin time (PT) and activated partial thromboplastin time (aPTT) were measured using standard clot-based assays. Plasma fibrinogen levels were determined using the Clauss method, while D-dimer concentrations were quantified using enzyme-linked immunosorbent assay (ELISA).

Quality control was implemented at all stages. Blood samples were processed promptly to prevent haemolysis or clotting. All instruments were calibrated daily, with control samples run alongside patient specimens. Results were monitored using Levey-Jennings charts, and any values outside ±2 standard deviations were repeated. Selected samples (10%) were re-assayed in duplicate to assess reproducibility, with coefficients of variation maintained ≤5% for platelet indices and ≤10% for coagulation parameters. ELISA assays included high-, medium-, and low-concentration controls to validate assay performance. All quality control procedures and corrective actions were documented to ensure traceability and adherence to best laboratory practices.

Statistical Analysis

Data were entered, cleaned, and analysed using IBM Statistical Package for the Social Sciences (SPSS) version 26.0 (IBM Corp., Armonk, NY, USA). Normally distributed continuous variables were summarised as means ± standard deviations, whereas non-normally distributed variables were presented as medians with interquartile ranges. Categorical variables were expressed as frequencies and percentages. Comparisons of continuous variables among the three study groups were performed using one-way analysis of variance (ANOVA) for normally distributed data, and the Kruskal–Wallis test for nonparametric data. The Chi-square test assessed differences in categorical variables.

To identify independent predictors of hypertensive disorders of pregnancy, multinomial logistic regression analysis was performed with pregnancy status as the dependent variable and the normotensive group serving as the reference category. Independent variables entered into the model included maternal age, gestational age, parity, BMI, systolic and diastolic blood pressure, platelet count, MPV, PT, and aPTT. Results were reported as regression coefficients, standard errors, Wald statistics, adjusted odds ratios, and 95% confidence intervals. A two-tailed p-value of less than 0.05 was considered statistically significant.

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RESULTS

Table 1: Demographic and Clinical Characteristics of Participants, Platelet Count and Indices and Fibrinogen and D-dimer Levels

Parameter	Normotensive (n=70)	Pre-eclampsia (n=70)	Eclampsia (n=70)	p-value
Demographic and Clinical Characteristics of Participants				
Mean age (years)	29.1 ± 4.6	30.4 ± 5.2	31.0 ± 5.0	0.083
Gestational age (weeks)	33.4 ± 3.5	32.6 ± 3.7	31.8 ± 4.0	0.071
Parity (median, IQR)	2 (1-3)	2 (1-4)	3 (2-4)	0.038*
Platelet Count and Indices				
Platelet count ($\times 10^9$/L)	240 ± 44	174 ± 40	129 ± 35	<0.001
MPV (fL)	9.6 ±1.0	11.1 ± 1.3	12.6 ± 1.5	<0.001
PDW (%)	13.7 ± 1.8	15.8 ± 2.1	17.9 ± 2.5	<0.001
Fibrinogen and D-dimer Levels				
Fibrinogen (g/L)	3.8 ± 0.7	4.7 ± 0.9	5.9 ± 1.1	<0.001
D-dimer (ng/mL)	420 ± 140	760 ± 200	1,190 ± 280	<0.001

Table 2: Bleeding Time, Prothrombin Time (PT), and Activated Partial Thromboplastin Time (aPTT)

Group	Bleeding Time (Minutes)	PT (Seconds)	aPTT (seconds)
Normotensive	3.6 ± 0.9	12.9 ± 1.3	**30.6** ± 3.7
Pre-eclampsia	4.8 ± 1.2	14.8 ± 1.9	**35.1** ± 4.3
Eclampsia	6.5 ± 1.6	17.6 ± 2.4	**40.5** ± 5.1
P < 0.001			

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Table 3: Multivariate Logistic Regression Analysis of Factors Associated with Preeclampsia and Eclampsia among pregnant Women in Osun State, Nigeria

Variable	B	S.E.	Wald	df	Sig. (p-value)	Adjusted Odds Ratio {Exp (B)}	95% CI for Exp (B)
Preeclampsia vs Normotensive							
Maternal age (years)	0.042	0.031	1.84	1	0.175	1.04	0.98 -1.11
Gestational age (weeks)	- 0.058	0.039	2.21	1	0.137	0.94	0.87-1.02
Multiparity (≥2)	- 0.357	0.266	1.80	1	0.180	0.70	0.42 - 1.18
Platelet count <150 ×10⁹/L	1.067	0.331	10.40	1	0.001*	2.91	1.55 - 5.43
Mean Platelet Volume (MPV)	0.741	0.298	6.19	1	0.013*	2.10	1.18 - 3.75
Prothrombin time (PT) Prolonged	0.482	0.312	2.38	1	0.123	1.62	0.88 - 2.99
Activated partial thromboplastin time (aPTT) prolonged	0.601	0.328	3.36	1	0.067	1.82	0.96-3.45
Eclampsia vs Normotensive							
Maternal age (years)	0.061	0.034	3.19	1	0.074	1.06	0.99 - 1.13
Gestational age (weeks)	- 0.084	0.041	4.18	1	0.041*	0.92	0.85 - 0.99
Multiparity (≥2)	- 0.428	0.281	2.32	1	0.128	0.65	0.38 - 1.12
Platelet count <150 ×10⁹/L	1.738	0.365	22.69	1	<0.001*	5.69	2.78 -11.65
Mean platelet volume (MPV)	0.902	0.334	7.29	1	0.007*	2.46	1.28 - 4.71
Prothrombin time (PT) Prolonged	1.229	0.352	12.18	1	<0.001*	3.42	1.80 - 6.31
Activated partial thromboplastin time (aPTT) prolonged	1.414	0.362	15.24	1	<0.001*	4.11	2.02 - 8.23

B-regression coefficient; S.E. = standard error; Wald = Wald chi-square; df = degrees of freedom, CI = confidence interval; MPV = mean platelet volume.

DISCUSSION

This study evaluated platelet function and coagulation profiles among normotensive pregnant women and those with preeclampsia and eclampsia attending antenatal clinics in Osun State, Nigeria. The findings demonstrate significant and progressive alterations in platelet indices and coagulation parameters with increasing severity of hypertensive disorders of pregnancy, reflecting the complex haemostatic disturbances that characterise preeclampsia and eclampsia and their contribution to adverse clinical outcomes.

The study groups were comparable with respect to maternal age and gestational age, thereby reducing the likelihood that these variables confounded observed differences in haemostatic parameters.

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Similar observations have been reported in previous studies, indicating that maternal age and gestational age are not independent determinants of haemostatic alterations in preeclampsia (Erez et al*.,* 2018; Jin et al*.,* 2023; Tadu et al*.,* 2023; Alemayehu et al*.,* 2024; Peng et al*.,* 2024). In contrast, parity differed significantly, with higher parity observed among women with eclampsia. Although primiparity is traditionally recognised as a risk factor for preeclampsia, emerging evidence from several African and Asian populations suggests that multiparity may be associated with more severe disease presentations, particularly in referral-based hospital settings where complicated cases are more likely to be managed (Godana et al*.,* 2023; Abdurrahman et al*.,* 2024; Li et al*.,* 2025; Putra et al*.,* 2025; Wang et al*.,* 2025). These variations likely reflect differences in healthcare access, referral patterns, and timing of presentation, highlighting the need for strengthened antenatal surveillance across all parity groups.

A key finding of this study was the marked reduction in platelet count among women with preeclampsia and eclampsia, with the lowest levels observed in eclamptic patients. Thrombocytopenia is a well-established haematological feature of hypertensive disorders of pregnancy and is widely regarded as an indicator of disease severity (Woldeamanuel et al*.,* 2023). The progressive decline in platelet count likely reflects increased platelet activation and consumption secondary to widespread endothelial injury. Endothelial dysfunction, a central feature of preeclampsia pathogenesis, exposes subendothelial collagen, promoting platelet adhesion, aggregation, and release of vasoactive mediators such as thromboxane A_2, which further exacerbate vasoconstriction and hypertension (Torres-Torres et al*.,* 2024; Martini et al*.,* 2025).

Consistent with declining platelet counts, mean platelet volume (MPV) and platelet distribution width (PDW) increased significantly with disease severity. These indices are recognised markers of platelet activation and heterogeneity, as larger platelets are metabolically more active and reflect increased bone marrow turnover in response to peripheral platelet destruction (Korniluk et al*.,* 2019). Similar findings have been reported across diverse populations, suggesting that MPV and PDW may serve as accessible indicators of disease progression, particularly in resource-limited settings where advanced biomarkers are unavailable (Agarwal et al*.,* 2022; Woldeamanuel et al*.,* 2023; Udeh et al., 2024; Sahin et al*.,* 2025; Tariq et al*.,* 2025).

Bleeding time was significantly prolonged among women with hypertensive disorders of pregnancy, indicating impairment of primary haemostasis. This abnormality is primarily attributable to quantitative and qualitative platelet dysfunction rather than deficiencies in coagulation factors (Agbani et al*.,* 2023; Zhang et al*.,* 2025). The pronounced prolongation observed in eclamptic patients suggests severe platelet dysfunction. It may increase the risk of postpartum haemorrhage or perioperative bleeding, emphasising the importance of haemostatic evaluation before invasive obstetric procedures.

Both prothrombin time (PT) and activated partial thromboplastin time (aPTT) were significantly prolonged in women with preeclampsia and eclampsia, indicating involvement of both intrinsic and extrinsic coagulation pathways. Although normal pregnancy is associated with a hypercoagulable state, preeclampsia represents a paradoxical condition characterised by simultaneous coagulation activation and consumption of clotting factors (Han et al*.,* 2021; Ali et al*.,* 2025). Prolongation of PT and aPTT may reflect consumption coagulopathy resulting from widespread microvascular thrombosis, hepatic dysfunction affecting clotting factor synthesis, or progression toward disseminated intravascular coagulation in severe disease (Alemayehu et al*.,* 2024; Zou et al*.,* 2025). The greater abnormalities observed in eclampsia indicate advanced haemostatic imbalance and heightened risk of haemorrhagic complications.

Fibrinogen and D-dimer levels were also significantly elevated, demonstrating increased coagulation activation and fibrinolysis. While

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fibrinogen physiologically rises during pregnancy, the marked elevation observed in hypertensive disorders likely reflects heightened inflammatory and prothrombotic activity (Han et al*.,* 2021; Imaralu et al*.,* 2023). Elevated D-dimer levels indicate increased fibrin turnover and ongoing thrombin generation, consistent with intensified intravascular coagulation (Alemayehu et al*.,* 2024; Killeen & Kok, 2025; Sinha et al*.,* 2025). Extremely high levels among women with eclampsia suggest advanced haemostatic dysregulation and increased risk of complications such as disseminated intravascular coagulation, placental abruption, and multi-organ dysfunction.

Multivariate analysis identified thrombocytopenia and elevated MPV as independent predictors of both preeclampsia and eclampsia. At the same time, prolonged PT and aPTT were strong predictors of eclampsia, highlighting progressive coagulation disturbance with disease severity. The absence of an independent association with maternal age suggests that haemostatic abnormalities are more closely linked to underlying disease mechanisms than demographic factors. The association between lower gestational age and eclampsia likely reflects early-onset severe disease, which is known to be associated with poorer maternal outcomes (Teka et al*.,* 2023; Kefiyelew et al*.,* 2026).

These findings have important clinical and public health implications. Routine assessment of platelet count and basic coagulation parameters, such as PT and aPTT, could facilitate early identification of women at risk of severe complications. In resource-constrained healthcare settings, these inexpensive and widely available tests may serve as practical tools for risk stratification and timely clinical intervention.

CONCLUSION

In conclusion, preeclampsia and eclampsia were associated with significant and progressive disturbances in platelet and coagulation profiles among pregnant women attending antenatal clinics in Osun State. Thrombocytopenia, increased platelet activation indices, prolonged bleeding time, deranged coagulation parameters, and elevated fibrinogen and D-dimer levels collectively indicate worsening haemostatic imbalance with disease severity. Incorporating routine haemostatic assessment into antenatal care may improve early detection of disease progression and guide clinical management, particularly in low-resource settings. Longitudinal studies are warranted to evaluate the predictive value of these parameters for maternal and foetal outcomes and to inform targeted preventive strategies.

LIMITATIONS

This study has some limitations. Its cross-sectional, hospital-based design limits causal inference and may introduce referral bias, restricting generalizability beyond tertiary care settings. Only selected haemostatic parameters were evaluated, and markers of endothelial dysfunction or natural anticoagulant pathways were not assessed. Additionally, potential confounding factors such as nutritional status, inflammatory conditions, and medication use were not evaluated, nor were fetal outcomes examined.

Conflicts of interest

The author declares no conflict of interest.

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