Original Article

Extrinsic Coagulation Pathway Selectivity Of Plateletcrit And Platelet Count In Normal Pregnancy: A Cross-Sectional Study In Ibadan, Nigeria

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ABSTRACT

The relationship between platelet morphological indices and coagulation pathway activity during pregnancy remains incompletely characterised, particularly in sub-Saharan African populations where population-specific haematological norms are limited. This study investigated pathway-selective associations between five platelet indices and three coagulation markers across the three trimesters of normal singleton pregnancy in a Nigerian cohort, with specific focus on whether plateletcrit demonstrates selective coupling to the extrinsic coagulation pathway. A cross-sectional design was employed involving 216 healthy pregnant women (56 in the first trimester, 82 in the second, and 78 in the third) alongside 50 non-pregnant controls recruited from public hospitals in Ibadan, Nigeria. Platelet indices—including platelet count, plateletcrit (PCT), mean platelet volume (MPV), platelet distribution width (PDW), and platelet-large cell ratio (P-LCR)—were measured using an automated haematology analyser, while prothrombin time (PT), international normalised ratio (INR), and activated partial thromboplastin time (APTT) were determined manually. Pearson correlation analysis with 95% confidence intervals and Bonferroni correction (adjusted $\alpha = 0.0033$) was performed, with Spearman analysis used for sensitivity testing. Plateletcrit showed significant positive correlations with PT ($r = 0.252$, 95% CI: 0.121–0.374, $P < 0.001$) and INR ($r = 0.259$, 95% CI: 0.129–0.381, $P < 0.001$), while platelet count also correlated with PT ($r = 0.230$, $P = 0.001$) and INR ($r = 0.248$, $P < 0.001$), with all associations remaining significant after correction. No platelet index demonstrated significant correlation with APTT, and PDW, MPV, and P-LCR showed no meaningful relationships with any coagulation markers; these findings were consistent in Spearman analysis. Overall, the results indicate that plateletcrit and platelet count exhibit selective association with extrinsic coagulation pathway markers (PT and INR) during normal pregnancy, while remaining independent of intrinsic pathway activity. This novel observation in a West African obstetric population suggests that total platelet mass co-varies with extrinsic coagulation efficiency across gestation and highlights the potential utility of plateletcrit as an adjunctive haemostatic monitoring parameter in resource-limited antenatal settings.

Keywords: Haemostasis, Nigeria, Platelet Count, Plateletcrit, Pregnancy, Prothrombin Time

INTRODUCTION

Venous thromboembolism complicates approximately 1 in 1000 pregnancies globally and remains a leading preventable cause of maternal mortality, driven by the physiologically adaptive hypercoagulable state of pregnancy.[1,2] This state is characterised by rising concentrations of procoagulant factors, particularly fibrinogen and factors VII, VIII, IX and X alongside suppression of natural anticoagulants and attenuation of fibrinolysis.[3] The burden is disproportionately high in sub-Saharan Africa, where Nigeria's maternal mortality ratio of approximately 576 per 100 000 live births reflects a well-documented deficit of laboratory-guided haemostatic monitoring capacity.[4]

Platelets participate centrally in coagulation by exposing phosphatidylserine-rich membrane surfaces that serve as catalytic scaffolds for prothrombinase and tenase complex assembly, thereby amplifying both extrinsic and intrinsic pathway activity.[5,6] Automated platelet morphological indices; platelet count, plateletcrit (PCT), mean platelet volume (MPV), platelet distribution width (PDW) and platelet-large cell ratio (P-LCR), are generated routinely from complete blood counts without additional cost and reflect biologically distinct properties of the circulating platelet pool. PCT, the platelet analogue of haematocrit, quantifies total circulating platelet mass and mathematically integrates both count and mean platelet size.[7] In resource-limited settings, these indices represent

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practically accessible haemostatic indicators where dedicated coagulometers are unavailable.[8]

The coagulation cascade is subdivided into the extrinsic pathway initiated by tissue factor and factor VII, assessed by prothrombin time (PT) and the international normalised ratio (INR), and the intrinsic pathway, initiated by contact activation and evaluated by activated partial thromboplastin time (APTT). During normal pregnancy, the extrinsic pathway is most prominently activated, whilst intrinsic pathway clotting times show relatively stable gestational behaviour.[3,9] Whether specific platelet indices couple differentially to these two pathways during pregnancy has not been systematically established in any African obstetric cohort.

Published data from Chinese10 and Indian11 cohorts have documented trimester-specific changes in platelet count and coagulation parameters independently, but no study from West Africa has applied a multiparameter correlation framework to examine pathway-selective platelet–coagulation associations across gestation.

This study aimed to characterise the bivariate associations between five platelet morphological indices and three coagulation markers across the three trimesters of normal singleton pregnancy in a Nigerian cohort, and to test the hypothesis that PCT as the most integrative measure of total platelet mass would demonstrate selective coupling to the extrinsic coagulation pathway (PT, INR) but not the intrinsic pathway (APTT).

MATERIALS AND METHODS

Study design and setting

A cross-sectional analytical study was conducted from March to August 2025 at three public hospitals in Ibadan, Oyo State, Nigeria: Adeoyo Maternity Teaching Hospital, Yemetu (n = 110); Jericho Specialist Hospital, Magazine Road (n = 60); and Ring Road State Hospital (n = 46). These facilities were purposively selected to represent geographically and socioeconomically diverse urban subpopulations. Sample collection was conducted during routine antenatal clinic sessions at identical calendar periods across all sites to minimise seasonal confounding.

Participants

Two hundred and sixteen healthy singleton pregnant women (aged 15–49 years) attending antenatal care were recruited by proportionate multi-stage sampling and stratified by trimester: first trimester (T1: ≤13 weeks, n = 56); second trimester (T2: 14–27 weeks, n = 82); third trimester (T3: ≥28 weeks, n = 78). Fifty non-pregnant women of reproductive age served as a reference control group from the same facilities. Gestational age was confirmed by last menstrual period, corroborated by first-trimester ultrasound where available.

Inclusion criteria required:

Written informed consent;

Confirmed singleton intrauterine pregnancy or confirmed non-pregnant status;

And active antenatal attendance (pregnant) or absence of known gynaecological pathology (controls).

Exclusion criteria for both groups comprised:

Active or antepartum haemorrhage;

Personal or family history of a diagnosed bleeding or thrombotic diathesis;

Current anticoagulant or antiplatelet therapy;

Autoimmune or inflammatory conditions; haematological malignancy;

Hypertensive disorders of pregnancy;

Diabetes mellitus;

Haematinics commenced within the preceding four weeks;

And non-consent.

Sociodemographic differences between groups (mean age: 28.33 ± 6.09 vs 24.96 ± 6.63 years; higher proportion of single women in controls) are inherent to this obstetric study design and are acknowledged as a limitation.

Laboratory procedures

Nine millilitres of venous blood were collected by standardised antecubital venepuncture under minimal stasis and processed within two hours of collection. Platelet indices were measured on a Sysmex KX-21N five-part automated haematology analyser (Sysmex Corporation, Kobe, Japan) per the manufacturer's standard operating procedure. Internal quality control was performed daily. PT was determined by the manual tilting-tube water-bath method using TECLOT PT-S reagent (Lot No. 10233707; Teco Medical, Germany); INR was derived as INR = (patient PT / mean normal PT)^ISI using a locally determined geometric mean normal PT of 12.8 s and ISI of 1.0. APTT was measured using TECLOT APTT-S reagent (Lot No. 10323628) with 0.025 M calcium chloride. All coagulation assays were performed in duplicate; the mean was used for analysis.

Statistical analysis

Data were analysed using IBM SPSS Statistics version 27.0. Normality was assessed by the Shapiro–Wilk test. Between-group comparisons were performed by one-way analysis of variance (ANOVA) with eta-squared (η^2) and Tukey's Honestly Significant Difference post-hoc test. Pearson's correlation coefficient (r) with 95% confidence intervals (Fisher's z-transformation) quantified bivariate associations; r^2 indicated variance explained. Bonferroni correction was applied across all 15 correlation pairs (adjusted $\alpha = 0.0033$). Spearman's rank correlation (ρ) was applied for non-normally distributed variables. This study is reported in accordance with the STROBE Statement.[12]

Ethical considerations

Ethical approval was obtained from the Oyo State Ministry of Health Ethical Review Committee, Secretariat, Ibadan, Nigeria (Reference: AD 13/479/041c). Written informed consent was obtained from all participants prior to enrolment. Participation was entirely voluntary; participants were informed of their right to withdraw at any stage without consequence. All data were anonymised and stored confidentially. The study was conducted in accordance with the Declaration of Helsinki (1964, revised 2013).

RESULTS

A total of 266 participants were included, comprising 216 pregnant women (T1 = 56, T2 = 82, T3 = 78) and 50 non-pregnant controls. Among the pregnant participants, the mean age was 28.33 ± 6.09 years, and 82.3% were married. Group-level means for all parameters are presented in Table 1. Platelet count demonstrated a declining trend from the first trimester (194.61 ± 48.07 $\times 10^3/\mu L$) to the second

trimester (177.90 ± 42.05), with partial recovery in the third trimester (187.23 ± 45.04), although values remained lower than those of controls (212.72 ± 35.49; $F_{3,262} = 7.058$, $P < 0.001$, $\eta^2 = 0.075$). Mean platelet volume (MPV) and platelet-large cell ratio (P-LCR) were significantly elevated across all trimesters relative to controls ($P = 0.003$, $\eta^2 = 0.052$; $P = 0.002$, $\eta^2 = 0.054$, respectively), whereas plateletcrit (PCT) was significantly reduced ($P = 0.008$, $\eta^2 = 0.044$). Platelet distribution width (PDW) showed no significant between-group difference ($P = 0.139$, $\eta^2 = 0.021$). Prothrombin time (PT) and international normalised ratio (INR) were significantly shorter in pregnant participants compared with controls ($P = 0.008$, $\eta^2 = 0.044$; $P = 0.003$, $\eta^2 = 0.053$), including in the third trimester (−0.73 s, 95% CI: −1.29 to −0.17 s), although all values remained within conventional reference limits. Activated partial thromboplastin time (APTT) did not differ significantly ($P = 0.643$, $\eta^2 = 0.006$).

The full 5 × 3 Pearson correlation matrix with corresponding 95% confidence intervals is presented in Table 2. PDW was non-normally distributed (Shapiro–Wilk $P < 0.001$); accordingly, Spearman's ρ was adopted as the primary metric for PDW and as a sensitivity analysis for all other indices. Plateletcrit demonstrated significant positive correlations with PT (r = 0.252, 95% CI: 0.121–0.374, $P < 0.001$, $r^2 = 6.4\%$) and INR (r = 0.259, 95% CI: 0.129–0.381, $P < 0.001$, $r^2 = 6.7\%$). Similarly, platelet count correlated with PT (r = 0.230, 95% CI: 0.098–0.354, $P = 0.001$, $r^2 = 5.3\%$) and INR (r = 0.248, 95% CI: 0.117–0.370, $P < 0.001$, $r^2 = 6.2\%$). All four associations remained statistically significant following Bonferroni correction ($\alpha = 0.0033$). No platelet index demonstrated a significant relationship with APTT (all $P \geq 0.118$), and PDW, MPV, and P-LCR showed no significant associations with any coagulation marker. Spearman sensitivity analyses confirmed the robustness of these findings (PCT–PT $\rho = 0.239$, $P = 0.001$; PCT–INR $\rho = 0.253$, $P < 0.001$; platelet count–PT $\rho = 0.221$, $P = 0.001$; platelet count–INR $\rho = 0.242$, $P < 0.001$). The pathway-selectivity pattern (Table 3) therefore demonstrates consistent and exclusive coupling of PCT and platelet count with extrinsic pathway markers, with no evidence of intrinsic pathway association for any platelet index.

Table 1: Platelet morphological indices and coagulation parameters by gestational group and controls (mean ± SD)

Parameter	Controls (n=50)	T1 (n=56)	T2 (n=82)	T3 (n=78)	F (df 3,262)	P-value	η^2
Platelet count (×10³/µL)	212.72±35.49	194.61±48.07	177.90±42.05	187.23±45.04	7.058	< 0.001*	0.075
PDW (fL)	9.60±1.34	10.17±1.67	11.46±8.07†	10.44±1.71	1.846	0.139	0.021
MPV (fL)	7.83±0.95	8.15±0.75	8.39±0.75	8.20±0.84	4.740	0.003*	0.052
P-LCR (%)	12.41±5.95	14.71±5.00	15.81±5.29	15.73±5.30	5.005	0.002*	0.054
PCT (%)	0.16±0.02	0.15±0.03	0.15±0.02	0.15±0.03	4.052	0.008*	0.044
PT (seconds)	14.93±0.96	14.37±1.22	14.64±1.31	14.20±1.31	4.065	0.008*	0.044
INR	1.07±0.07	1.02±0.09	1.04±0.10	1.01±0.10	4.894	0.003*	0.053
APTT (seconds)	31.43±3.32	30.68±7.07	31.68±7.81	32.09±5.46	0.558	0.643	0.006

*T1–T3 = first to third trimester; PDW = platelet distribution width; MPV = mean platelet volume; P-LCR = platelet-large cell ratio; PCT = plateletcrit; PT = prothrombin time; INR = international normalised ratio; APTT = activated partial thromboplastin time; η^2 = eta-squared. *P < 0.05 (ANOVA, Tukey HSD). †T2 PDW SD elevated due to outlier influence. All PT/INR values remained within clinical reference bounds.*

Table 2: Pearson correlation coefficients (95% CIs) between platelet indices and coagulation markers in pregnant women (n = 216)

Index (mean±SD)	PT r (95% CI)	PT P	INR r (95% CI)	INR P	APTT r (95% CI)	APTT P	ρ PT	ρ INR
Plt count 185.60±45.04 ×10³/µL	0.230 (0.098–0.354)	0.001*†	0.248 (0.117–0.370)	<0.001*†	0.084 (−0.044–0.215)	0.217	0.221*	0.242*
PDW (fL) 10.76±5.16	0.027 (−0.108–0.161)	0.693	0.022 (−0.113–0.156)	0.750	0.082 (−0.053–0.213)	0.232	0.031	0.026
MPV (fL) 8.26±0.79	−0.044 (−0.178–0.091)	0.522	−0.072 (−0.206–0.064)	0.291	0.002 (−0.133–0.137)	0.973	−0.039	−0.068
P-LCR (%) 15.49±5.22	0.124 (−0.011–0.256)	0.070	0.099 (−0.036–0.232)	0.147	0.046 (−0.089–0.180)	0.497	0.120	0.096
PCT (%) 0.15±0.03	0.252 (0.121–0.374)	<0.001*†	0.259 (0.129–0.381)	<0.001*†	0.107 (−0.028–0.239)	0.118	0.239*	0.253*

*PT mean: 14.41 ± 1.29 s; INR mean: 1.03 ± 0.10; APTT mean: 31.57 ± 6.84 s. 95% CIs by Fisher's z-transformation. *P < 0.05 (uncorrected). †Bonferroni-corrected (α = 0.0033). Spearman ρ: *P < 0.05.*

Table 3: Pathway-selectivity summary: significance pattern of platelet–coagulation correlations

Platelet Index	Extrinsic – PT (r; P)	Extrinsic – INR (r; P)	Intrinsic – APTT (r; P)	Coupling Pattern	Bonferroni
Platelet count	0.230; P=0.001	0.248; P<0.001	0.084; P=0.217	Extrinsic-selective	Survives
PCT	0.252; P<0.001	0.259; P<0.001	0.107; P=0.118	Extrinsic-selective	Survives
PDW	0.027; P=0.693	0.022; P=0.750	0.082; P=0.232	None	Does not survive
MPV	−0.044; P=0.522	−0.072; P=0.291	0.002; P=0.973	None	Does not survive
P-LCR	0.124; P=0.070	0.099; P=0.147	0.046; P=0.497	None	Does not survive

PCT = plateletcrit; PT = prothrombin time; INR = international normalised ratio; APTT = activated partial thromboplastin time. Bonferroni-adjusted α = 0.0033.

DISCUSSION

This study provides the first evidence, within a West African obstetric population, of a statistically robust and pathway-selective relationship between platelet mass indices and coagulation pathway activity during normal pregnancy. Specifically, both plateletcrit and platelet count exhibit selective association with extrinsic pathway markers (PT and INR), with consistent effect sizes, persistence after multiple-testing correction, and confirmation through non-parametric sensitivity analysis. The absence of any association with APTT across all platelet indices establishes a clear pathway-discriminant pattern that has not been previously reported in African obstetric literature.

The observed positive correlations between plateletcrit and PT/INR warrant careful interpretation and should not be construed as indicating a causal prolongation of extrinsic pathway clotting with increasing platelet mass.[11] In normal pregnancy, shortening of PT is primarily driven by progressive increases in clotting factors, particularly factor VII and factor X.[3,10] The positive association observed in this study is therefore most plausibly explained by a compositional effect arising from shared gestational dynamics.[13] As gestation advances, platelet

count tends to decline, while extrinsic pathway activity increases, resulting in shorter PT values. Consequently, individuals with relatively higher platelet counts are more likely to be at earlier gestational stages and thus exhibit comparatively less shortened PT.[14] This reflects a cross-sectional confounding structure driven by gestational timing rather than a direct mechanistic interaction between platelets and coagulation factors.[15] This interpretation is further supported by the absence of trimester-stratified correlation analyses in the present dataset, representing a key methodological limitation that should be addressed in future longitudinal studies incorporating direct measurement of factor VII activity.[16]

The slightly stronger correlations observed for plateletcrit compared with platelet count alone ($r = 0.252–0.259$ vs $0.230–0.248$) are biologically plausible, given that plateletcrit integrates both platelet number and mean platelet volume, thereby providing a more comprehensive estimate of total platelet biomass. However, the relatively modest r^2 values (5.3–6.7%) indicate that the majority of variability in extrinsic pathway markers remains attributable to coagulation factor concentrations. Platelet indices should therefore be regarded as secondary or adjunctive correlates rather than primary determinants of coagulation pathway function.[17]

The absence of significant associations with APTT is mechanistically consistent with the established biology of the intrinsic pathway, which is regulated predominantly by contact activation factors and amplification via factors VIII and IX.[18] Platelet involvement in this pathway occurs primarily at the level of membrane-bound tenase complex formation rather than through total platelet mass.[5] The lack of between-group differences in APTT ($P = 0.643$) further supports the relative stability of intrinsic pathway activity during normal pregnancy, in agreement with existing literature.[10] This dissociation carries important clinical implications, indicating that alterations in platelet count or plateletcrit should not be interpreted as reflective of intrinsic pathway dysfunction in obstetric patients.

Although MPV and P-LCR were significantly elevated in pregnant participants, consistent with enhanced platelet activation during gestation, these morphological indices did not demonstrate any measurable association with coagulation pathway markers. This suggests that physiological platelet activation in uncomplicated pregnancy remains below the threshold required to influence global clotting times. [14,19] The dissociation between morphological activation markers and functional coagulation indices further supports the relative utility of plateletcrit over size-based indices in haemostatic assessment. This observation aligns with the findings which indicate that elevated MPV is associated with pathological states such as pre-eclampsia rather than normal pregnancy.[20,11]

From a laboratory medicine perspective, these findings have potential relevance in resource-limited settings such as Nigeria, where automated haematology analysers are more widely available than dedicated coagulation testing platforms.[4,8] The observed associations suggest that plateletcrit and platelet count may offer limited adjunctive insight into haemostatic status in contexts where PT/INR measurement is not readily accessible. However, given the modest effect sizes and the cross-sectional nature of the data, these indices cannot substitute for direct coagulation testing. Prospective studies incorporating clinical outcomes are required to determine whether integration of platelet indices into antenatal screening protocols can provide meaningful diagnostic or prognostic value.

LIMITATIONS

The cross-sectional design precludes within-woman longitudinal inference; observed correlations reflect between-subject variation and cannot establish causality. The absence of trimester-stratified analyses means gestational-age confounding cannot be excluded, this is the most critical analytical limitation. The manual coagulation methodology introduces higher variability than automated optical coagulometers, and ISI = 1.0 limits INR comparability. Factor VII and fibrinogen were not measured, precluding direct mechanistic testing. No external quality assurance was conducted for the haematology analyser. The control and pregnant groups were not age-matched, though the 3.4-year mean difference is unlikely to materially confound correlation findings.

CONCLUSION

Plateletcrit and platelet count demonstrate statistically robust, extrinsic-pathway-selective positive correlations with PT and INR during normal singleton pregnancy in a Nigerian cohort, surviving Bonferroni correction and Spearman sensitivity analysis, whilst all five platelet indices remain independent of intrinsic pathway activity. PCT is identified as the most informative platelet index for extrinsic pathway co-variation.

RECOMMENDATIONS

Based on the findings of this study, plateletcrit and platelet count should be considered as practical adjunctive markers for haemostatic assessment in pregnant women, particularly in resource-limited settings where full coagulation testing is not readily available, given their selective association with extrinsic pathway markers and independence from intrinsic pathway activity. Implementation should be accompanied by the development of population-specific reference ranges for Nigerian and broader sub-Saharan African cohorts to improve interpretive accuracy. Longitudinal studies with serial measurements across gestation, including direct assays of factor VII and factor X, are recommended to clarify mechanistic relationships and account for compositional gestational changes. Furthermore, prospective investigations correlating platelet indices with maternal and fetal outcomes, such as postpartum haemorrhage, pre-eclampsia, and thrombotic events, are warranted to determine their predictive utility and clinical relevance. Laboratory capacity building, training in interpretation of plateletcrit relative to coagulation indices, and integration of automated haematology analysers into routine antenatal screening could enhance early detection of haemostatic perturbations, while clinicians should exercise caution not to infer causality from cross-sectional associations, recognizing that observed correlations reflect compositional gestational dynamics rather than direct modulation of clotting.

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Competing interests

The authors declare that they have no financial or personal relationships with other people or organisations that could inappropriately influence (bias) this work.

Authors' contributions

A.B.E. contributed to conceptualisation, data collection, laboratory analysis, formal analysis, data curation, writing, original draft, and writing, review and editing. M.A.M. contributed to conceptualisation, supervision, and writing, review and editing. O.T.O. contributed to data collection, laboratory analysis, and writing, review and editing. F.O.A. contributed to formal analysis and writing, review and editing. A.H.O. contributed to data collection and writing, review and editing.

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Data availability

The data supporting the findings of this study are available from the corresponding author, A.B.E., upon reasonable request.

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REFERENCES

1. Nichols KM, Henkin S, Creager MA. Venous thromboembolism associated with pregnancy. J Am Coll Cardiol. 2020;75(6):652–663. https://doi.org/10.1016/j.jacc.2019.12.051
2. Kozak M, Cosmi E, Gerotziafas G, Marschang P, Stanek A. Venous thromboembolism in pregnancy: recent advances. Pol Arch Intern Med. 2025;135(3):16877. https://doi.org/10.20452/pamw.16877
3. Babik B, Kupcsulik P, Fazakas J. Gestational hemostasis: a natural model for hemostasis resuscitation. Perioper Med (Lond). 2021;10:33. https://doi.org/10.1186/s13741-021-00225-0
4. Olamijulo JA, Olorunfemi G, Osman N. Predictors and causes of in-hospital maternal deaths at a tertiary hospital in South-Western Nigeria. Niger Postgrad Med J. 2022;29(4):325–333. https://doi.org/10.4103/npmj.npmj_180_22
5. Agbani EO, Skeith L, Lee A. Preeclampsia: platelet procoagulant membrane dynamics and critical biomarkers. Res Pract Thromb Haemost. 2023;7(2):100075. https://doi.org/10.1016/j.rpth.2023.100075
6. Forstner D, Guettler J, Gauster M. Changes in maternal platelet physiology during gestation and their interaction with trophoblasts. Int J Mol Sci. 2021;22(20):11164. https://doi.org/10.3390/ijms222011164
7. Temur M, Tasgoz FN, Cift T, Serpim G, Ustunyurt E. Role of platelet indices in prediction of preeclampsia. Ginekol Pol. 2021;92(11):792–796. https://doi.org/10.5603/GP.a2021.0116
8. Price MA, Fast PE, Mshai M, Lambrick M, Machira YW, Gieber L, **et al.** Region-specific laboratory reference intervals are important: a systematic review of the data from Africa. *PLOS Glob Public Health*. 2022;2(11):e0000783. doi:10.1371/journal.pgph.0000783.
9. Kaserer A, Castellucci C, Henckert M, Breymann C, Spahn DR. Patient blood management in pregnancy. Transfus Med Hemother. 2023;50(4):257–267. https://doi.org/10.1159/000529568
10. Wang W, Long K, Deng F, Ye W, Zhang P, Chen X, et al. Changes in levels of coagulation parameters in different trimesters among Chinese pregnant women. *J Clin Lab Anal*. 2021;35(4):e23724. doi:10.1002/jcla.23724
11. Umezuluike BO, Anikwe CC, Nnachi OO, Iwe BC, Ifemelumma CC, Dimejesi IB. Correlation of platelet parameters with adverse maternal and neonatal outcomes in severe preeclampsia: A case-control study. *Heliyon*. 2021;7(12):e08484. doi:10.1016/j.heliyon.2021.e08484.
12. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP, et al.; STROBE Initiative. The STROBE statement: guidelines for reporting observational studies. *Lancet*. 2007;370(9596):1453-1457. doi:10.1016/S0140-6736(07)61602-X.
13. Fu Y, Liu J, Jinfang L, Dai Y, Ding X, Dong H, et al. Reference intervals for coagulation parameters in non-pregnant and pregnant women. *Sci Rep*. 2022;12:14216. doi:10.1038/s41598-022-18145-0
14. Forstner D, Guettler J, Gauster M. Changes in maternal platelet physiology during gestation and their interaction with trophoblasts. *Int J Mol Sci*. 2021;22(23):12713. doi:10.3390/ijms222312713
15. Babker AM, Elnaim EO. Hematological changes during all trimesters in normal pregnancy. *J Drug Deliv Ther*. 2020;10(2):1-4. doi:10.22270/jddt.v10i2.3958.
16. Haslinger C, Korte W, Hothorn T, Brun R, Greenberg C, Zimmermann R. The impact of prepartum factor XIII activity on postpartum blood loss. *J Thromb Haemost*. 2020;18(9):2285-2294. doi:10.1111/jth.14889
17. Sang Y, Roest M, de Laat B, de Groot PG, Huskens D. Interplay between platelets and coagulation. *Blood Rev*. 2020;46:100733. doi:10.1016/j.blre.2020.100733.

18. Park JH, Park SH. Back to basics: the coagulation pathway. *Blood Res*. 2024;59(1):1-12. doi:10.5045/br.2024.2024067

19. Guettler J, Forstner D, Gauster M. Maternal platelets at the first trimester maternal-placental interface – small players with great impact on placenta development. *Placenta*. 2021;114:34-42. doi:10.1016/j.placenta.2021.09.00.

20. Sachan R, Patel S, Vandana, Sachan S, Shyam S. Role of platelet count and mean platelet volume and red cell distribution width in the prediction of preeclampsia in early pregnancy. *J Fam Med Prim Care*. 2021;10(11):4018-4023. doi:10.4103/jfmpc.jfmpc_2347_20