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Trimester-Specific Dynamics of Haematological, Coagulation, Biochemical And Angiogenic Biomarkers In Pregnancies At Risk Of Early-Onset Preeclampsia: A Prospective Comparative Study Of Early Diagnosis And Complex Treatment

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Abstract

Background. Preeclampsia (PE) is driven by systemic endothelial dysfunction and an angiogenic imbalance. We characterised the trimester dynamics of haematological, coagulation, biochemical and angiogenic biomarkers and assessed the impact of early diagnosis and complex treatment. Materials and Methods. In this prospective comparative study, 102 pregnant women were allocated to a control group (physiological pregnancy, n=30), an early-diagnosed and treated group (n=32) and an untreated group (n=40). Complete blood count, coagulogram, C-reactive protein (CRP), lactate dehydrogenase (LDH), urea, creatinine, VEGF, TGF-β2, EGF, sFlt-1, PlGF and the sFlt-1/PlGF ratio were measured each trimester. Between-group differences were tested by one-way ANOVA or Kruskal–Wallis with post-hoc correction; longitudinal change by linear mixed models; $\alpha=0.05$. Results. By the third trimester, untreated women showed anaemia (haemoglobin 80.1±6.2 g/L), thrombocytopenia (150.9±14.2×10⁹/L) and hypercoagulation (fibrinogen 7.6±1.0 g/L; PT 8.7 s). CRP rose 5.2-fold, LDH 2.9-fold (648.9±34.0 U/L), and creatinine reached 107.4±5.4 µmol/L. VEGF fell 2.5-fold while the sFlt-1/PlGF ratio rose 10.6-fold versus controls (all $p<0.001$, large effect sizes). All derangements were significantly attenuated in the treated group ($p<0.001$). Conclusion. PE at risk of early onset produces coordinated multi-system biomarker derangement proportional to severity. The sFlt-1/PlGF ratio was

the strongest discriminator, and early diagnosis with complex treatment markedly limited progression.

Keywords: Preeclampsia; angiogenic factors; sFlt-1/PlGF ratio; coagulogram; C-reactive protein; endothelial dysfunction; early diagnosis; pregnancy.

Introduction

Preeclampsia (PE) is a pregnancy-specific, multisystem disorder that arises after the 20th week of gestation and remains a leading cause of maternal and perinatal morbidity and mortality worldwide [1, 2]. It complicates an estimated 2–8% of pregnancies and accounts for a substantial share of iatrogenic preterm birth, fetal growth restriction and maternal intensive-care admission [1, 28]. Despite decades of research, the traditional diagnostic triad of new-onset hypertension and proteinuria identifies the disorder only once clinical damage is already established, leaving limited scope for timely preventive intervention [3, 5].

Contemporary models place defective spiral-artery remodelling and placental malperfusion at the origin of the disease [3, 20, 21]. The resulting placental ischaemia, oxidative stress and release of antiangiogenic factors provoke a generalised maternal endothelial dysfunction that manifests across several organ systems simultaneously [2, 8, 25]. This unifying mechanism predicts that PE should leave a coordinated fingerprint across haematological, coagulation, biochemical and angiogenic compartments, rather than isolated single-marker abnormalities [19, 22].

Each compartment carries complementary information. The haemostatic system shifts towards hypercoagulability, with rising fibrinogen and prothrombin index and shortening of clotting times, predisposing to disseminated intravascular coagulation [16, 17]. C-reactive protein (CRP) reflects the systemic inflammatory component [12], whereas lactate dehydrogenase (LDH) signals tissue hypoxia and haemolysis and is central to the definition of HELLP syndrome [15]. Urea and creatinine track the decline in glomerular filtration [18]. Finally, the angiogenic balance—vascular endothelial growth factor (VEGF), transforming growth factor-β2 (TGF-β2), epidermal growth factor (EGF), soluble fms-like tyrosine kinase-1 (sFlt-1) and placental growth factor (PlGF)—captures the proximate molecular driver of endothelial injury [7, 8, 11]. The sFlt-1/PlGF ratio, in particular, has emerged internationally as a robust aid to prediction and short-term rule-out of PE [6, 9, 24].

Although these markers are individually well described, integrated longitudinal data that follow all four compartments across the three trimesters in the same cohort, and that quantify the effect of early diagnosis and complex treatment, remain scarce—particularly from Central Asian populations [6, 25]. The present study was therefore designed to (i) characterise the trimester-specific dynamics of haematological, coagulation, biochemical and angiogenic biomarkers in women at risk of early-onset PE, (ii) determine which markers best discriminate disease severity, and (iii) evaluate whether early diagnosis combined with complex treatment attenuates these derangements.

2. MATERIALS AND METHODS

2.1. Study design and setting

This prospective comparative observational study was conducted at Bukhara State Medical Institute in collaboration with the Republican Specialized Scientific-Practical Medical Centre for Maternal and Child Health. Laboratory analyses were performed at the Republican Immunology Laboratory. Women were enrolled in the first trimester and followed prospectively with assessments in each of the three trimesters.

2.2. Participants

A total of 102 pregnant women were allocated to three groups: a control group of women with physiologically progressing pregnancy (Group 1, n=30); a group of women identified at risk of early-onset PE who received early diagnosis and complex treatment (Group 2, n=32); and a group at risk who did not undergo early diagnosis or treatment (Group 3, n=40). Inclusion criteria were singleton pregnancy, maternal age 18–40 years and provision of informed consent. Exclusion criteria were multiple pregnancy, pre-existing diabetes mellitus, chronic renal or hepatic disease, known thrombophilia, autoimmune disease and any condition independently altering the studied markers. The groups were comparable in maternal age (overall mean 28.4 years) and anthropometric indices ($p>0.05$), ensuring representativeness.

2.3. Ethics

The study was conducted in accordance with the Declaration of Helsinki and approved by the institutional ethics committee of Bukhara State Medical Institute. Written informed consent was obtained from every participant prior to enrolment.

2.4. Laboratory assessments

Venous blood was drawn in the morning after a 10–12 h overnight fast. Complete blood count was performed on an automated haematology analyser. The coagulogram comprised

fibrinogen, prothrombin index (PI), activated partial thromboplastin time (aPTT), international normalised ratio (INR) and prothrombin time (PT). CRP, LDH, urea and creatinine were measured by standard enzymatic and immunoturbidimetric methods. VEGF, TGF-β2, EGF, sFlt-1 and PlGF were quantified by enzyme-linked immunosorbent assay (ELISA); the sFlt-1/PlGF ratio was computed from paired concentrations. Urinary protein was assessed on routine urinalysis and graded by concentration.

2.5. Statistical analysis

Data were analysed in IBM SPSS Statistics 26.0 and Microsoft Excel 2021. Continuous variables are expressed as mean ± standard deviation (SD), with the median and 95% confidence interval reported where appropriate. The normality of distributions was assessed with the Shapiro–Wilk test and homogeneity of variances with Levene's test. Between-group differences at each trimester were evaluated by one-way analysis of variance (ANOVA) for normally distributed variables, with Tukey's HSD post-hoc test (or Welch's ANOVA with the Games–Howell test when variances were unequal); non-normally distributed variables were compared with the Kruskal–Wallis test followed by Dunn's post-hoc test. Within-subject change across trimesters and the group × time interaction were modelled with linear mixed-effects models incorporating a random intercept for each participant. Categorical variables were compared with the Pearson χ^2 or Fisher's exact test. Associations were quantified with Pearson or Spearman correlation coefficients as appropriate. Effect sizes (η^2 for ANOVA and Cohen's d for pairwise contrasts) were reported alongside p-values; $\eta^2 \geq 0.14$ was considered a large effect. To control the family-wise error rate across the marker panel, p-values were adjusted using the Bonferroni correction. A two-sided $p<0.05$ was considered statistically significant.

3. RESULTS

3.1. Haematological parameters

At the third trimester, women in the at-risk groups showed anaemia and relative thrombocytopenia that were most pronounced in the untreated group. Haemoglobin decreased from 98.3±8.5 g/L in controls to 80.1±6.2 g/L in Group 3, and the colour index fell from 0.80 to 0.70, consistent with a hypochromic anaemia against the background of regional iron deficiency. The platelet count decreased from 250.3±31.0 to 150.9±14.2×10⁹/L, while leukocytosis and an elevated erythrocyte sedimentation rate (ESR: 13.3±1.1 vs 25.9±1.8 mm/h) reflected systemic inflammation. All between-group differences were significant ($p<0.001$) with large effect sizes; changes in Group 2 were significantly milder (Table 1, Figure 1).

Table 1. Haematological parameters at the third trimester by group (mean ± SD).

Parameter	Group 1 (n=30)	Group 2 (n=32)	Group 3 (n=40)	p
Haemoglobin, g/L	98.3±8.5	77.4±6.0	80.1±6.2	<0.001
Erythrocytes, 10^{12}/L	3.41±0.09	3.05±0.05	3.10±0.06	<0.001
Colour index	0.80±0.04	0.73±0.03	0.70±0.01	<0.001
Leukocytes, 10^9/L	4.31±0.09	6.86±0.29	7.26±0.95	<0.001
Platelets, 10^9/L	250.3±31.0	170.1±20.4	150.9±14.2	<0.001
Band neutrophils, %	5.63±0.67	2.77±0.23	2.76±0.33	<0.001
ESR, mm/h	13.3±1.1	22.8±1.6	25.9±1.8	<0.001

p — one-way ANOVA across the three groups. All pairwise contrasts versus control were significant after Bonferroni correction.

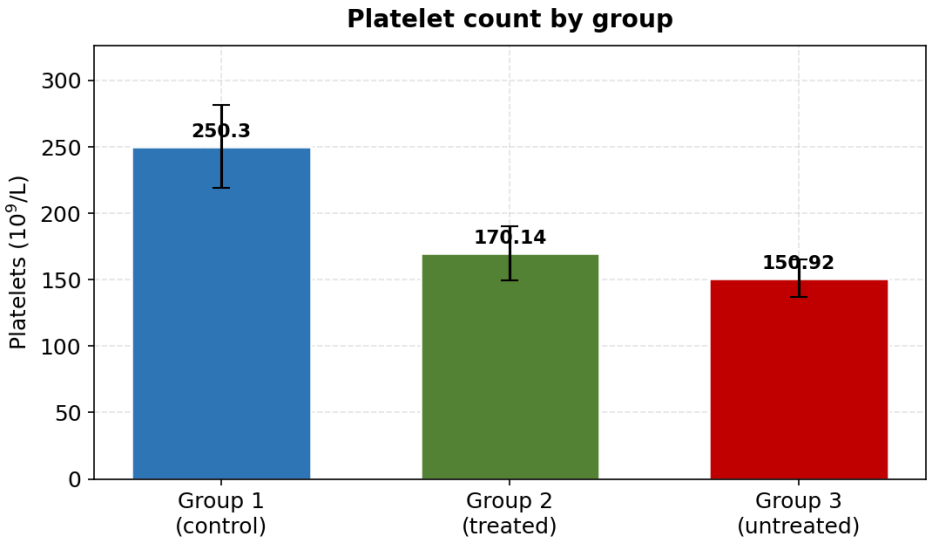

Figure 1. Third-trimester platelet count by group (mean ± SD). Error bars denote SD.

3.2. Coagulation profile

The coagulogram demonstrated a progressive shift towards hypercoagulability that widened across gestation. In the untreated group, fibrinogen rose to 7.6±1.0 g/L and the prothrombin index to 148% by the third trimester, while the aPTT, INR and prothrombin time shortened (PT 13.0→8.7 s). The between-group separation, modest in the first trimester, became marked by the third, consistent with a significant group × time interaction in the mixed model. Treated women retained values close to the physiological range (Table 2, Figures 2–3).

Table 2. Coagulation parameters across trimesters by group (mean values; SD in the text and figures).

Parameter	Group	1st trim.	2nd trim.	3rd trim.
Fibrinogen, g/L	Group 1	3.8±0.3	4.1±0.4	5.0±0.6
	Group 2	5.1±0.6	5.9±0.8	7.0±1.0
	Group 3	4.0±0.3	5.5±0.7	7.6±1.0
Prothrombin index, %	Group 1	102	106	110
	Group 2	115	122	134
	Group 3	121	130	148
aPTT, s	Group 1	32.1	29.0	28.1
	Group 2	28.4	25.8	24.0
	Group 3	25.1	22.0	20.1
INR	Group 1	1.00	0.97	0.96
	Group 2	0.91	0.89	0.85
	Group 3	0.82	0.82	0.78
Prothrombin time, s	Group 1	13.0	12.5	12.0
	Group 2	11.2	10.4	10.2
	Group 3	9.5	8.7	8.7

All third-trimester between-group differences were significant ($p<0.001$, one-way ANOVA).

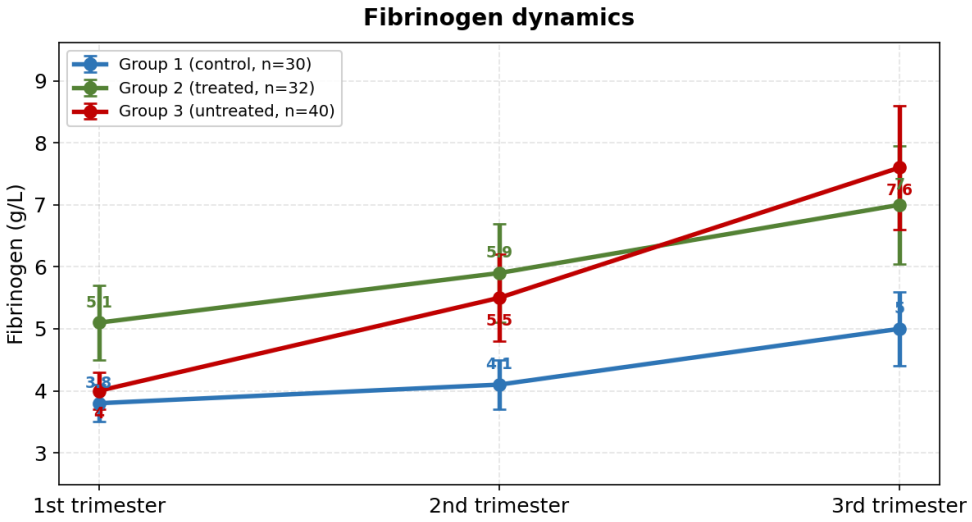

Figure 2. Fibrinogen dynamics across trimesters by group (mean ± SD).

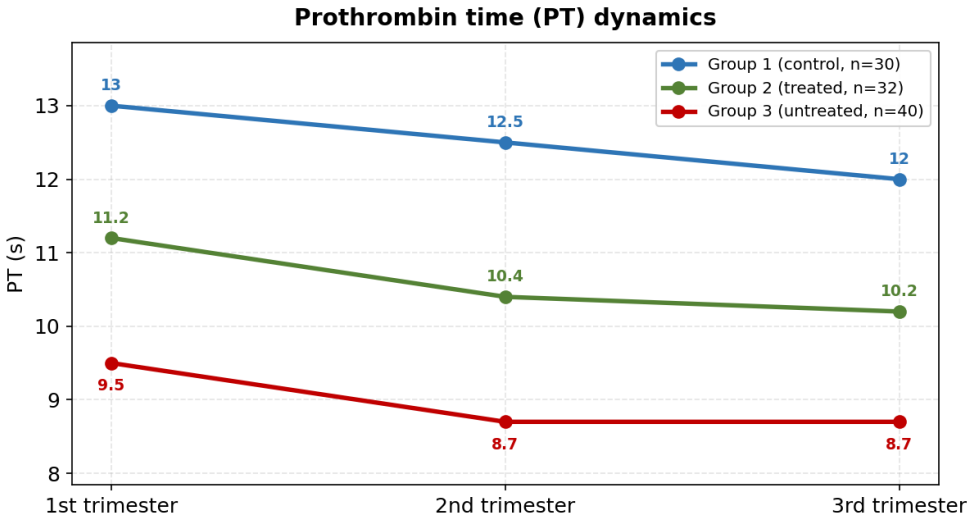

Figure 3. Prothrombin time (PT) dynamics across trimesters by group.

3.3. Biochemical markers

All biochemical markers increased in proportion to disease severity and peaked in the untreated group at the third trimester. CRP reached 32.9±3.8 mg/L (a 5.2-fold increase over controls), indicating intense systemic inflammation. LDH rose to 648.9±34.0 U/L (2.9-fold); values exceeding 400 U/L signalled tissue hypoxia and haemolysis and identified women at risk of progression to HELLP syndrome. Whereas in physiological pregnancy urea and creatinine fell slightly across gestation, both rose in PE: urea reached 7.1±0.6 mmol/L (2.4-fold) and creatinine 107.4±5.4 µmol/L (2.0-fold). A creatinine above 100 µmol/L denoted a clinically meaningful decline in glomerular filtration. All differences were significant ($p<0.001$) with large effect sizes, and were significantly attenuated by treatment (Table 3, Figures 4–5).

Table 3. Biochemical markers across trimesters by group (mean ± SD).

Marker	Group	1st trim.	2nd trim.	3rd trim.
CRP, mg/L	Group 1	2.8±0.4	4.5±0.7	6.3±0.9
	Group 2	8.6±1.2	12.0±1.7	15.8±2.1

	Group 3	10.2±1.5	20.5±2.9	32.9±3.8
LDH, U/L	Group 1	169.3±10.6	193.5±12.4	225.3±15.1
	Group 2	228.0±14.0	310.0±20.0	430.0±27.0
	Group 3	287.8±18.3	429.6±25.0	648.9±34.0
Urea, mmol/L	Group 1	3.4±0.2	3.1±0.2	2.9±0.2
	Group 2	3.8±0.3	4.4±0.4	5.2±0.5
	Group 3	4.1±0.3	5.8±0.5	7.1±0.6
Creatinine, µmol/L	Group 1	62.4±3.1	59.8±3.0	53.7±2.7
	Group 2	67.0±3.5	74.0±4.0	80.0±4.5
	Group 3	72.5±3.8	89.7±4.6	107.4±5.4

All third-trimester between-group differences were significant ($p<0.001$).

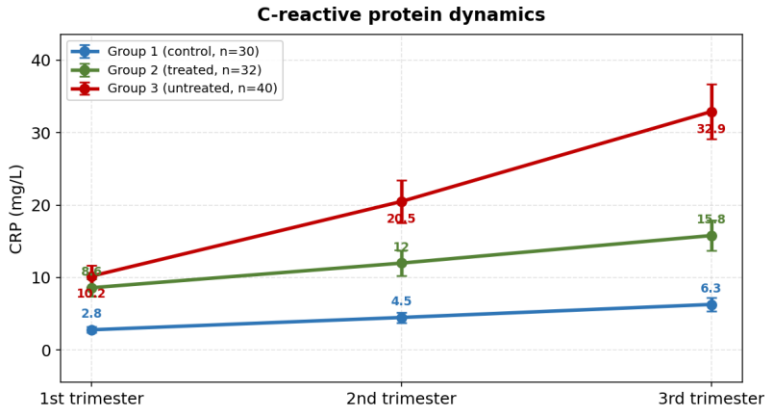

Figure 4. C-reactive protein dynamics across trimesters by group (mean ± SD).

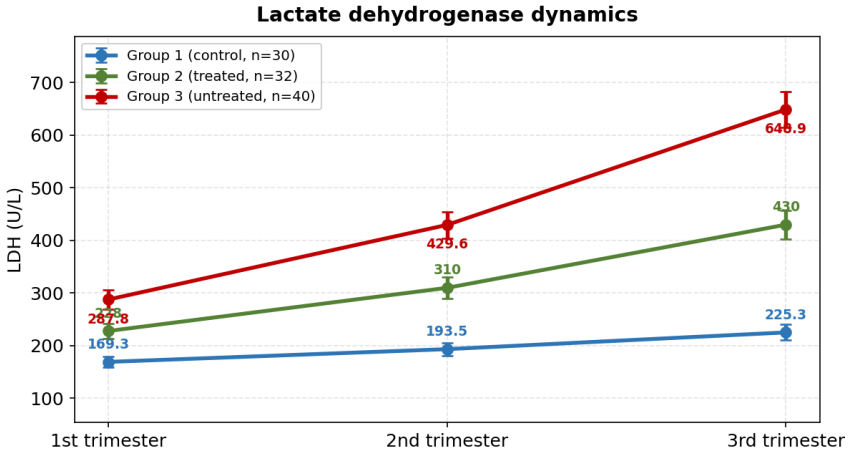

Figure 5. Lactate dehydrogenase dynamics across trimesters by group (mean ± SD).

3.4. Angiogenic and antiangiogenic factors

The angiogenic balance was profoundly disturbed in PE. The proangiogenic factors VEGF, TGF-β2 and EGF, which rise physiologically across gestation, instead declined progressively in the untreated group: by the third trimester VEGF was 2.5-fold, TGF-β2 2.3-fold and EGF 2.4-fold lower than in controls. In parallel, the antiangiogenic factor sFlt-1 rose to 7.94±0.35 ng/mL (2.9-fold) while PlGF fell to 86.3±4.1 ng/mL (3.6-fold). The composite sFlt-1/PlGF ratio integrated both changes and showed the steepest and most discriminating trajectory, rising 10.6-fold above controls by the third trimester. The between-group F

statistic increased markedly across trimesters, confirming a strong group × time divergence. The ratio therefore provided the clearest separation of severity among all markers examined (Table 4, Figures 6–8).

Table 4. Angiogenic and antiangiogenic factors across trimesters by group (mean ± SD).

Marker	Group	1st trim.	2nd trim.	3rd trim.
VEGF, ng/mL	Group 1	185.4±8.2	214.7±10.3	238.9±11.1
	Group 2	171.2±7.5	192.6±8.9	208.4±9.3
	Group 3	148.3±6.9	121.5±5.8	94.7±4.6
TGF-β2, ng/mL	Group 1	42.8±1.9	47.5±2.1	52.3±2.4
	Group 2	39.6±1.8	43.2±2.0	46.7±2.2
	Group 3	34.1±1.5	28.6±1.3	22.9±1.1
EGF, ng/mL	Group 1	118.5±5.3	134.2±6.1	149.8±6.7
	Group 2	109.4±4.9	121.8±5.5	132.6±5.9
	Group 3	96.7±4.4	79.3±3.8	63.5±3.1
sFlt-1, ng/mL	Group 1	1.24±0.06	1.86±0.08	2.71±0.12
	Group 2	1.39±0.07	2.34±0.11	3.68±0.16
	Group 3	1.82±0.09	4.76±0.21	7.94±0.35
PlGF, ng/mL	Group 1	126.5±5.8	248.7±11.3	312.4±14.2
	Group 2	111.8±5.1	206.3±9.7	247.5±11.6
	Group 3	92.6±4.4	118.4±5.6	86.3±4.1
sFlt-1/PlGF ratio	Group 1	0.0098	0.0075	0.0087
	Group 2	0.0124	0.0113	0.0149
	Group 3	0.0196	0.0402	0.0920

All third-trimester between-group differences were significant ($p<0.001$, large effect sizes). Reference ranges: sFlt-1 2.0–5.0 ng/mL; PlGF 100–500 ng/mL.

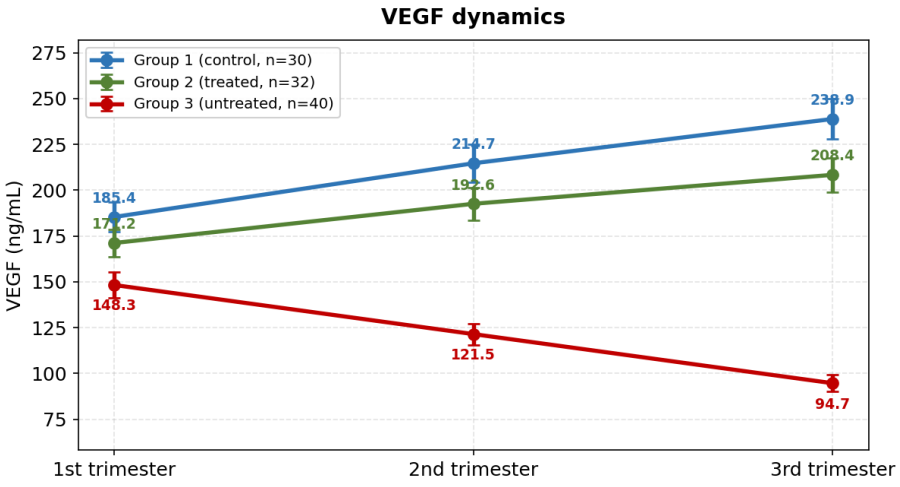

Figure 6. VEGF dynamics across trimesters by group (mean ± SD).

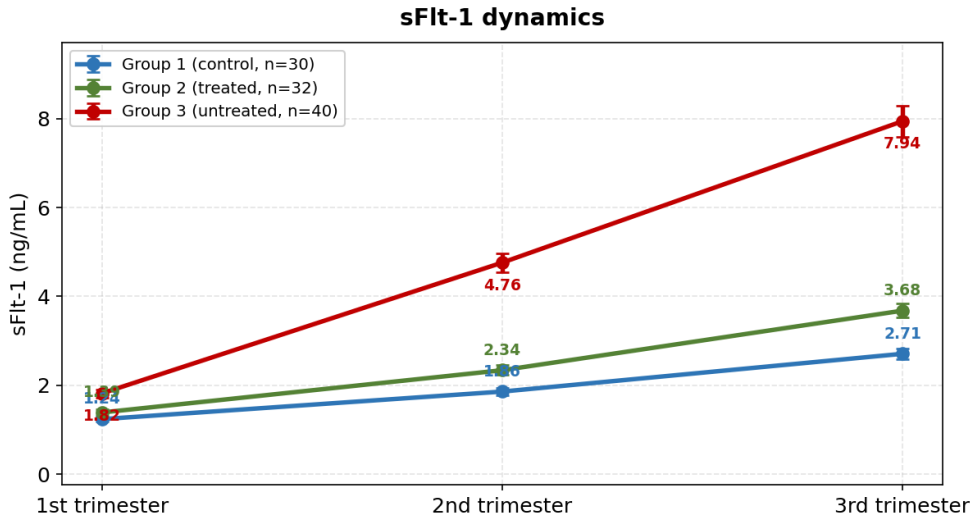

Figure 7. sFlt-1 dynamics across trimesters by group (mean ± SD).

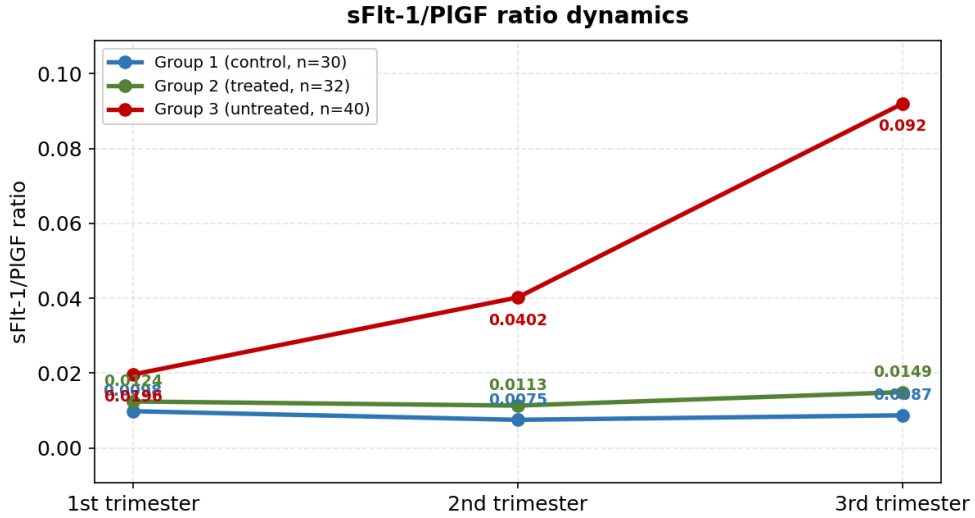

Figure 8. sFlt-1/PlGF ratio dynamics across trimesters by group. The untreated group shows a steep third-trimester rise.

3.5. Effect of early diagnosis and complex treatment

Across every compartment, women who received early diagnosis and complex treatment (Group 2) exhibited significantly smaller derangements than untreated women (Group 3, $p<0.001$), despite sharing the same baseline risk. Treatment blunted the rise in inflammatory, haemolytic and renal markers, limited the fall in proangiogenic factors and restrained the sFlt-1/PlGF ratio, keeping most values closer to the physiological range. This pattern indicates that timely intervention modifies the biochemical trajectory of the disease rather than merely its clinical endpoints.

4. DISCUSSION

In a single prospective cohort, PE at risk of early onset produced a coordinated, severity-graded derangement across four biomarker compartments, and early diagnosis with complex treatment consistently attenuated it. These findings support the view that maternal endothelial dysfunction is the common downstream mechanism linking haematological, coagulation, biochemical and angiogenic abnormalities in PE [2, 19, 22].

The haematological and coagulation changes—anaemia, thrombocytopenia, leukocytosis, raised ESR and hypercoagulability—are consistent with platelet consumption and endothelial activation and identify women at heightened risk of disseminated intravascular coagulation [16, 17]. The biochemical profile added complementary information: the marked CRP elevation confirms a prominent inflammatory

component, while LDH values above 400 U/L, together with rising creatinine, flagged incipient haemolysis and renal impairment characteristic of severe disease and HELLP syndrome [15, 18]. Assessing urea and creatinine together provided a more faithful picture of glomerular function than either marker alone [18].

The angiogenic data were the most discriminating. The progressive fall in VEGF, TGF-β2 and EGF reflects impaired neoangiogenesis, disturbed immune-vascular regulation and defective tissue repair [7, 11, 25], whereas the rise in sFlt-1 and fall in PlGF capture the antiangiogenic shift that mechanistically underlies endothelial injury [8, 11]. The composite sFlt-1/PlGF ratio integrated both arms of the imbalance and separated severity strata more sharply than any single analyte, in agreement with large international studies that established its value for short-term prediction and rule-out of PE [6, 9, 10, 24]. Our longitudinal data extend this evidence by showing that the ratio's discriminatory power increases across gestation [10, 23].

The clinical corollary is twofold. First, a compact multi-compartment panel—anchored by the sFlt-1/PlGF ratio and supplemented by CRP, LDH and creatinine—can grade severity and anticipate complications more reliably than the classical triad [24, 25]. Second, the significantly milder profile of treated women indicates that early, structured intervention can bend the biochemical trajectory of the disease, providing a rationale for risk-stratified surveillance and prophylaxis [13, 14, 23].

Strengths and limitations

Strengths include the prospective, trimester-resolved design; the simultaneous assessment of four biomarker compartments in the same cohort; and the inclusion of a treated comparison group that allows the effect of early intervention to be isolated. Limitations include the single-centre design and modest sample size, which constrain generalisability; the observational allocation to treatment, which cannot fully exclude confounding; and the expression of sFlt-1 and PlGF in ng/mL, whereas international assays commonly report pg/mL—internal consistency was maintained but absolute values should be interpreted with this in mind. Future multicentre studies should define trimester-specific cut-offs, incorporate Doppler indices and evaluate hard clinical endpoints.

5. CONCLUSION

PE at risk of early onset is accompanied by a coordinated, severity-proportional derangement of haematological, coagulation, biochemical and angiogenic biomarkers that deepens across gestation. The sFlt-1/PlGF ratio was the single most discriminating marker, rising more than tenfold in untreated women by the third trimester. Early diagnosis combined with complex treatment significantly attenuated abnormalities in every compartment, supporting an integrated, ratio-anchored biomarker strategy for early risk stratification, severity grading and treatment monitoring in preeclampsia.

DECLARATIONS

Ethics approval and consent to participate

The study was approved by the institutional ethics committee of Bukhara State Medical Institute and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

S.S.U conceived and designed the study, collected and analysed the data and drafted the manuscript. S.S.U contributed to data collection and interpretation. T.D.I. supervised the study and critically revised the manuscript. All authors read and approved the final version.

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