

Frequency of Coagulation Abnormalities in Women with Preeclampsia with Severe Features and Their Association with Maternal Disease Severity and Fetal Outcomes

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Abstract

Background: Preeclampsia with severe features remains a major cause of maternal and perinatal morbidity in South Asia, yet haemostatic derangement and its relevance to fetal compromise are poorly characterised in Bangladesh. **Objective:** To determine the frequency of coagulation abnormalities in women with preeclampsia with severe features and their association with maternal disease severity and fetal outcomes. **Methods:** This analytical cross-sectional study enrolled 216 consecutive women with preeclampsia with severe features at Dhaka Medical College Hospital from March to September 2026. Any coagulation abnormality was defined as platelet count <150,000/ μ L, prothrombin time (PT) >16.1 s, activated partial thromboplastin time (aPTT) >40 s, or D-dimer >1.7 mg/L (fibrinogen-equivalent units). Scans were performed at or after admission blood sampling. Multivariable regression estimated adjusted associations. **Results:** Overall, 140 women (64.8%; 95% CI 58.0 - 71.2) had at least one coagulation abnormality; thrombocytopenia (48.6%) and elevated D-dimer (39.4%) predominated, followed by prolonged aPTT (19.4%) and PT (16.7%). Affected women more often had severe-range blood pressure (93.6% vs 60.5%), serum creatinine $\geq$ 1.1 mg/dL (55.7% vs 18.4%), and alanine ami-

notransferase ≥ 70 U/L (25.7% vs 2.6%) (all $P < 0.001$), and low birthweight (82.1% vs 68.4%; $P = 0.021$). Excess fetal growth restriction (38.6% vs 25.0%) was borderline (chi-square $P = 0.044$; Fisher's exact $P = 0.051$). After adjustment, any coagulation abnormality was associated with an abnormal antenatal scan (adjusted odds ratio 3.20; 95% CI 1.60 - 6.40; $P = 0.001$) and lower birthweight ($\beta -0.151$ kg; 95% CI -0.270 to -0.032 ; $P = 0.013$). Preterm delivery did not differ significantly between groups. **Conclusion:** Nearly two-thirds of women with preeclampsia with severe features have coagulation abnormalities, which indicate more advanced end-organ involvement and placental compromise. A low-cost admission coagulation panel may help prioritise early fetal ultrasound and Doppler assessment in resource-limited settings; its predictive value requires prospective study.

Keywords

Preeclampsia, Severe Features, Coagulation Abnormalities, Thrombocytopenia, D-Dimer, Fetal Growth Restriction, Birthweight, Bangladesh

1. Introduction

Hypertensive disorders of pregnancy are among the most consequential complications of pregnancy, and preeclampsia is their most dangerous form. A 2025 meta-analysis of 70 studies estimated the global prevalence of preeclampsia at 4.43%, with the highest eclampsia rates observed in South Asia and sub-Saharan Africa [1]. In a population-based cohort across eight low- and middle-income countries, major direct maternal morbidity affected 43.9% of pregnancies in South Asia, almost double the sub-Saharan African estimate [2]. In Bangladesh, a hospital-based study in Sylhet identified preeclampsia in 14.4% of pregnant women [3], and preeclampsia/eclampsia accounted for 24% of all maternal deaths in the 2016 national survey, with a cause-specific mortality ratio that has stalled at 46 per 100,000 live births [4]. Hypertensive disorders affect approximately 11% of pregnancies in India [5], account for 29% of maternal deaths in Pakistan [6], and have remained among the five leading causes of maternal mortality in Sri Lanka for two decades [7]. These convergent data identify severe preeclampsia as a persistent regional priority.

Pregnancy is a physiologically hypercoagulable state involving profound changes in haemostasis. Fibrinogen and procoagulant factors VII, VIII, IX and X rise progressively, several to more than twice their non-pregnant levels, protecting the mother against haemorrhage at placental separation [8]. A vital balance between the coagulation, anticoagulant and fibrinolytic systems is therefore necessary to maintain uteroplacental and multi-organ perfusion. In preeclampsia, defective spiral artery remodelling, placental ischaemia and the release of antiangiogenic factors cause systemic endothelial injury [9] [10], tipping this balance towards

platelet activation and consumption, thrombin generation and microvascular fibrin deposition.

A 2024 meta-analysis of 30 studies involving 5964 women demonstrated significantly prolonged PT and aPTT in preeclampsia compared with normotensive pregnancy [11]. Hospital-based studies from China, India and Ethiopia have shown that platelet count falls while aPTT, D-dimer and platelet indices rise with increasing disease severity, and that these changes are associated with adverse pregnancy outcomes and lower birthweight [12]-[15]. Platelet parameters have also been correlated with adverse neonatal outcomes in severe preeclampsia [16].

Important gaps remain. D-dimer rises physiologically through gestation, and a prospective multicentre cohort found no predictive value for placenta-mediated complications [17], while late-pregnancy reference values approach thresholds commonly applied in clinical practice [18]. Most studies compare preeclamptic with normotensive women rather than examining heterogeneity within severe disease, few adjust for confounders such as gestational age, and contemporary prognostic models emphasise angiogenic and metabolic markers that are rarely available in low-resource settings [19]. Data from Bangladesh are particularly scarce.

We therefore aimed to determine the frequency of coagulation abnormalities in women with preeclampsia with severe features and tested the hypothesis that the presence of any abnormality is associated with greater maternal disease severity and is independently associated with adverse fetal outcomes, specifically abnormal antenatal scan findings and lower birthweight.

2. Materials and Methods

2.1. Study Design, Setting, and Duration

We conducted a hospital-based observational, analytical cross-sectional study over a six-month period, from 01 March to 01 September 2026, at the Feto-Maternal Unit, Department of Obstetrics and Gynaecology, Dhaka Medical College Hospital, Dhaka, Bangladesh, a tertiary referral centre for high-risk pregnancies.

2.2. Participants and Sampling

Consecutive women admitted with preeclampsia with severe features during the study period were enrolled until 216 participants were included. With this sample, the prevalence of any coagulation abnormality could be estimated with an absolute precision of approximately $\pm 6.4\%$ at the 95% confidence level. Preeclampsia was defined as new-onset hypertension after 20 weeks of gestation in a previously normotensive woman, with systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg on two measurements at least 4 hours apart, accompanied by proteinuria or evidence of maternal end-organ dysfunction [20]. Severe features included severe-range blood pressure (systolic ≥ 160 mm Hg or diastolic ≥ 110 mm Hg), platelet count $< 100,000/\mu\text{L}$, renal insufficiency (serum creatinine ≥ 1.1 mg/dL or doubling of baseline in the absence of other renal disease), liver trans-

aminases more than twice the upper limit of normal or persistent right upper-quadrant/epigastric pain not explained by another diagnosis, pulmonary oedema, new-onset headache unresponsive to medication and not accounted for by an alternative diagnosis, or visual disturbance. Women with preeclampsia with severe features who also had an eclamptic seizure ($n = 12$) were included in the study group, and seizure episode was analysed as a marker of maternal disease severity (Section 2.4). Women with known bleeding disorders predating pregnancy, current anticoagulant therapy, chronic liver disease, platelet disorders unrelated to preeclampsia, or refusal to participate were excluded.

2.3. Data Collection and Laboratory Assessment

After written informed consent, maternal sociodemographic characteristics, obstetric history, relevant comorbidities, presenting clinical findings, blood pressure, urine protein, antenatal ultrasonographic findings, gestational age at admission and at delivery, mode of delivery, and neonatal birthweight were recorded using a structured data-collection form. Venous blood obtained at the index admission was analysed in the hospital laboratory by standard automated laboratory methods for complete blood count, platelet count, prothrombin time (PT), activated partial thromboplastin time (aPTT), D-dimer, serum creatinine, and alanine aminotransferase. D-dimer was measured in citrated plasma by a quantitative latex-enhanced immunoturbidimetric assay on an automated coagulation analyser and is reported in mg/L fibrinogen-equivalent units (FEU).

The antenatal scan used for outcome classification was the ultrasound and Doppler examination performed during the index admission, at or after admission blood sampling and before delivery. Coagulation status was therefore established before, or at the same time as, the scan findings and never after them. Antenatal scan categories were abstracted from the contemporaneous ultrasound/Doppler report according to the criteria given in Section 2.4. There were no missing observations for variables included in the reported analyses.

2.4. Operational Definitions

For the coagulation analysis, thrombocytopenia was defined as platelet count $<150,000/\mu\text{L}$, the conventional definition of thrombocytopenia in pregnancy [21]; platelet count $<100,000/\mu\text{L}$ was treated as a more severe, nested subgroup. The prespecified study cut-offs for prolonged PT, prolonged aPTT, and elevated D-dimer were >16.1 seconds, >40 seconds, and >1.7 mg/L FEU, respectively; each is the upper reference limit issued by the hospital laboratory for the assay in use. These laboratory limits are not gestation-specific, and the implications for D-dimer are considered in the Discussion. Any coagulation abnormality was defined as the presence of at least one abnormality among platelet count, PT, aPTT, and D-dimer. Participants with none of these abnormalities were classified as having no predefined coagulation abnormality.

Maternal disease-severity markers were severe-range blood pressure, urine pro-

tein 3+/4+, serum creatinine ≥ 1.1 mg/dL, alanine aminotransferase ≥ 70 U/L, and seizure episode. Obstetric outcomes were preterm delivery (<37 weeks) and early preterm delivery (<34 weeks). Fetal and placental outcomes included fetal growth restriction, any abnormal antenatal scan, low birthweight (<2500 g), and birthweight in kilograms. Any abnormal antenatal scan was a composite outcome defined as fetal growth restriction, oligohydramnios, or placental insufficiency documented on antenatal assessment. Fetal growth restriction was defined as an ultrasonographic estimated fetal weight or abdominal circumference below the 10th centile for gestational age [22]. Oligohydramnios was defined as an amniotic fluid index ≤ 5 cm or a single deepest vertical pocket <2 cm [23]. Placental insufficiency was defined as abnormal umbilical artery Doppler velocimetry, that is, resistance indices raised for gestational age or absent or reversed end-diastolic flow [22]. These were the criteria applied by the reporting sonologists in routine practice, and each category was abstracted as documented in the report.

Clinical history/comorbidity burden was calculated as the number of recorded background factors present: family history of cardiovascular disease, hypertension in a previous pregnancy, dyslipidaemia, pregestational diabetes mellitus, polycystic ovary syndrome, chronic kidney disease, hypothyroidism, inherited/acquired thrombophilia, and autoimmune disease.

2.5. Statistical Analysis

Data were entered, cleaned, and analysed using SPSS version 25. Continuous variables were summarised as mean $\pm$ standard deviation (SD) when approximately normally distributed and as median (interquartile range, IQR) when skewed; categorical variables were summarised as frequency and percentage. The prevalence of any and individual coagulation abnormalities was estimated for the full cohort; exact binomial 95% confidence intervals were used for the prevalence estimates displayed in **Figure 1**.

Participants were categorised as having no coagulation abnormality or any coagulation abnormality using the prespecified thresholds. Categorical maternal, obstetric, and fetal variables were compared with the Pearson chi-square test; Fisher's exact test was used when expected cell counts were <5 . Where the chi-square P value lay close to the significance threshold (0.04 to 0.06), the Fisher's exact P value is reported alongside it as a check of robustness, and such results are interpreted as borderline. Continuous variables were compared using the independent-samples t test or Mann-Whitney U test, as appropriate. Crude odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for categorical outcomes.

Multivariable logistic regression was used to estimate the association between any coagulation abnormality and any abnormal antenatal scan, adjusting for age, body mass index, severe-range blood pressure, proteinuria 3+/4+, serum creatinine ≥ 1.1 mg/dL, and clinical history/comorbidity burden. Multivariable linear regression was used to estimate the association between any coagulation abnor-

mality and birthweight, adjusting for the same maternal covariates and gestational age at delivery. Covariates were selected a priori as potential confounders reflecting overall maternal disease severity; because some may also lie on the pathway between haemostatic derangement and placental dysfunction, the adjusted estimates represent associations independent of concurrent disease severity and may be conservative. Adjusted associations are reported as adjusted ORs (aORs) or regression coefficients (β) with 95% CIs. All tests were two-sided, and $P < 0.05$ was considered statistically significant. P values are reported to three decimal places, and values below 0.001 are reported as $P < 0.001$.

Gestational age was handled differently in the two models. In the scan model, gestational age at delivery was not entered because delivery follows the scan and its timing may be determined by the scan findings; adjusting for it would condition on a consequence of the outcome. Gestational age at admission, although recorded, was not part of the prespecified covariate set, which was restricted to maternal characteristics and markers of concurrent disease severity, and two of the three components of the composite scan outcome are themselves referenced to gestational age (centile-based fetal biometry and gestation-specific Doppler indices). In the birthweight model, gestational age at delivery was included because it is the principal determinant of birthweight, and the aim was to estimate the association with fetal size that is not explained by gestational duration. Because gestational age at delivery may itself be shortened by disease severity, it may act as an intermediate variable; the gestational-age-adjusted coefficient is therefore interpreted as the association that does not operate through earlier delivery, and the unadjusted mean difference, which does not condition on gestational age, is reported alongside it.

Two sensitivity analyses were performed. First, because a platelet count $<100,000/\mu\text{L}$ is both a severe-feature criterion and a component of the exposure, the cohort was restricted to women with a platelet count $\geq 100,000/\mu\text{L}$, all of whom necessarily qualified for severe features through a criterion other than platelet count, and the prevalence of any coagulation abnormality was re-estimated. For the outcome associations, a tipping-point (bounding) analysis was applied to the crude estimates in this restricted cohort: we determined how many of the excluded women would need to have had the outcome for the association to lose statistical significance, and we report the estimate under the most extreme allocation. Second, to gauge the potential influence of gestational age at admission and of other omitted or unmeasured confounders on the scan model, we calculated the E-value for the adjusted odds ratio and for the confidence limit closer to the null, using the square-root transformation for a common outcome [24].

2.6. Ethical Considerations

The study protocol was reviewed and approved by the Institutional Review Board of Dhaka Medical College (IRB No. IRB-DMC/2026/111) and was conducted in accordance with the Declaration of Helsinki (2024). Written informed consent was obtained from all participants prior to enrolment. Participation was volun-

tary, and participants' privacy and confidentiality were maintained throughout the study. Participants were informed of their right to withdraw at any time without penalty or any effect on their clinical care.

3. Results

3.1. Baseline Characteristics

The mean maternal age was 28.5 ± 5.4 years and mean BMI was 30.9 ± 4.4 kg/m²; 25.0% were nulliparous, and the median gravidity was 3 (IQR, 2 - 4). Mean gestational age at delivery was 35.2 ± 2.7 weeks. Anaemia was present in 58.8%, and 27.3% reported a family history of cardiovascular disease; the most frequent background comorbidities were hypertension in a previous pregnancy (23.6%) and pregestational diabetes mellitus (15.7%) (**Table 1**).

Table 1. Baseline maternal and obstetric characteristics (N = 216).

Characteristic	Value
Age, years	28.5 ± 5.4
Body mass index, kg/m ²	30.9 ± 4.4
Education $\leq$ secondary	162 (75.0)
Housewife	162 (75.0)
Lower/lower-middle socioeconomic status	135 (62.5)
Gravidity, median (IQR)	3 (2 - 4)
Nulliparous	54 (25.0)
Para ≥ 3	64 (29.6)
Spontaneous conception	191 (88.4)
Gestational age at delivery, weeks	35.2 ± 2.7
Anaemia (haemoglobin <11 g/dL)	127 (58.8)
Family history of cardiovascular disease	59 (27.3)
Hypertension in a previous pregnancy	51 (23.6)
Pregestational diabetes mellitus	34 (15.7)
Polycystic ovary syndrome	30 (13.9)
Hypothyroidism	26 (12.0)

3.2. Severity Features and Coagulation Profile of the Study Population

Among the 216 women with preeclampsia with severe features, 147 (68.1%) had severe systolic hypertension (≥ 160 mm Hg), while 112 (51.9%) had severe diastolic hypertension (≥ 110 mm Hg). Proteinuria of 3+/4+ was present in 70 (32.4%)

women. Thrombocytopenia with a platelet count $<150,000/\mu\text{L}$ was observed in 105 (48.6%) participants, including 30 (13.9%) with a platelet count $<100,000/\mu\text{L}$. Prolonged PT (>16.1 s) and prolonged aPTT (>40 s) were found in 36 (16.7%) and 42 (19.4%) women, respectively. An elevated D-dimer level (>1.7 mg/L) was observed in 85 (39.4%) participants, while elevated serum creatinine (≥ 1.1 mg/dL) was noted in 92 (42.6%). Overall, 140 (64.8%) women had at least one coagulation abnormality (**Table 2**).

Table 2. Severity features and coagulation profile of women with preeclampsia with severe features (N = 216).

Variable	Frequency (n)	Percentage (%)
Severe systolic BP (≥ 160 mm Hg)	147	68.1
Severe diastolic BP (≥ 110 mm Hg)	112	51.9
Proteinuria (3+/4+)	70	32.4
Platelet count $<150,000/\mu\text{L}$	105	48.6
Platelet count $<100,000/\mu\text{L}$	30	13.9
Prolonged PT (>16.1 s)	36	16.7
Prolonged aPTT (>40 s)	42	19.4
D-dimer >1.7 mg/L	85	39.4
Elevated serum creatinine (≥ 1.1 mg/dL)	92	42.6
Any coagulation abnormality	140	64.8

3.3. Maternal Disease Severity According to Coagulation Status

Compared with women without a coagulation abnormality, those with any coagulation abnormality more often had severe-range blood pressure (93.6% vs 60.5%), serum creatinine ≥ 1.1 mg/dL (55.7% vs 18.4%), and alanine aminotransferase ≥ 70 U/L (25.7% vs 2.6%) (all $P < 0.001$), as well as proteinuria 3+/4+ (37.1% vs 23.7%; $P = 0.044$; Fisher's exact $P = 0.049$). Seizure episodes were numerically more frequent (7.9% vs 1.3%), but the difference was not statistically significant (Fisher's exact $P = 0.060$) (**Table 3**).

3.4. Fetal and Obstetric Outcomes According to Coagulation Status

Women with coagulation abnormalities had higher rates of abnormal antenatal scans (62.1% vs 39.5%; $P = 0.001$) and low birthweight (82.1% vs 68.4%; $P = 0.021$). Fetal growth restriction was numerically more frequent (38.6% vs 25.0%); the pre-specified chi-square test gave $P = 0.044$ (crude OR 1.88; 95% CI 1.01 - 3.50), but the result was not robust to the choice of test (Fisher's exact $P = 0.051$). We therefore regard this difference as borderline and do not interpret it as an established association. After adjustment, coagulation abnormalities remained associated

with increased odds of an abnormal antenatal scan (aOR, 3.20; 95% CI, 1.60 - 6.40; $P = 0.001$) and lower birthweight (β , -0.151 kg; 95% CI, -0.270 to -0.032 ; $P = 0.013$). The birthweight difference was -0.22 kg (95% CI -0.32 to -0.12) before any adjustment and -0.151 kg after adjustment for maternal covariates and gestational age at delivery; the association was therefore attenuated, but not removed, after adjustment that included gestational age at delivery. Preterm and early preterm delivery were not significantly different between groups (Table 4).

Table 3. Maternal clinical and biochemical characteristics according to coagulation status.

Variable	No coagulation abnormality (n = 76)	Any coagulation abnormality (n = 140)	P value
Severe-range blood pressure	46 (60.5)	131 (93.6)	<0.001
Proteinuria 3+/4+	18 (23.7)	52 (37.1)	0.044
Creatinine ≥ 1.1 mg/dL	14 (18.4)	78 (55.7)	<0.001
Alanine aminotransferase ≥ 70 U/L	2 (2.6)	36 (25.7)	<0.001
Seizure episode	1 (1.3)	11 (7.9)	0.060

Table 4. Fetal and obstetric outcomes according to coagulation status and adjusted associations.

Outcome	No abnormality (n = 76)	Any abnormality (n = 140)	Crude association (95% CI); P	Adjusted association (95% CI); P
Obstetric outcomes				
Preterm delivery (<37 wk)	44 (57.9)	97 (69.3)	OR 1.64 (0.92 - 2.93) P = 0.093	—
Early preterm delivery (<34 wk)	18 (23.7)	37 (26.4)	OR 1.16 (0.61 - 2.21) P = 0.658	—
Fetal and placental outcomes				
Fetal growth restriction	19 (25.0)	54 (38.6)	OR 1.88 (1.01 - 3.50) P = 0.044	—
Any abnormal antenatal scan	30 (39.5)	87 (62.1)	OR 2.52 (1.42 - 4.46) P = 0.001	aOR 3.20 (1.60 - 6.40) P = 0.001
Birthweight outcomes				
Low birthweight (<2500 g)	52 (68.4)	115 (82.1)	OR 2.12 (1.11 - 4.06) P = 0.021	—
Birthweight, kg	2.30 $\pm$ 0.32	2.08 $\pm$ 0.40	MD -0.22 kg (-0.32 to -0.12) P < 0.001	β -0.151 kg (-0.270 to -0.032) P = 0.013

Crude P values for categorical outcomes are from the Pearson chi-square test; for fetal growth restriction, Fisher's exact $P = 0.051$. OR, odds ratio; aOR, odds ratio adjusted for age, body mass index, severe-range blood pressure, proteinuria 3+/4+, serum creatinine ≥ 1.1 mg/dL and clinical history/comorbidity burden; MD, unadjusted mean difference; β , coefficient adjusted for the same covariates and gestational age at delivery.

Figure 1 presents the frequency of each coagulation abnormality with exact bi-

nomial 95% CIs: any coagulation abnormality 64.8% (95% CI 58.0 - 71.2), platelet count <150,000/ μ L 48.6% (41.8 - 55.5), D-dimer >1.7 mg/L 39.4% (32.8 - 46.2), aPTT >40 s 19.4% (14.4 - 25.4), and PT >16.1 s 16.7% (12.0 - 22.3).

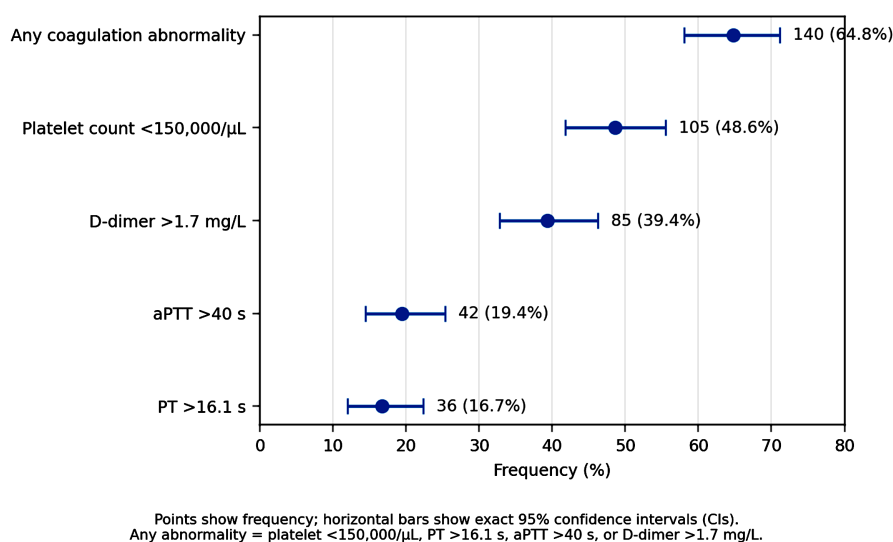


Figure 1. Frequency (with exact 95% confidence intervals) of coagulation abnormalities in preeclampsia with severe features (N = 216).

3.5. Sensitivity Analyses

All 30 women with a platelet count <100,000/ μ L were, by definition, in the coagulation-abnormality group. Among the remaining 186 women, who had a platelet count $\geq$ 100,000/ μ L and therefore qualified for severe features through a criterion other than platelet count, 110 (59.1%; 95% CI 51.7 - 66.3) had at least one coagulation abnormality, compared with 64.8% in the full cohort. In the tipping-point analysis of this restricted cohort, the crude association with an abnormal antenatal scan remained statistically significant unless at least 28 of the 30 excluded women (93.3%) had an abnormal scan, a proportion well above the 62.1% observed in the coagulation-abnormality group as a whole. Under the most extreme allocation, in which all 30 excluded women had an abnormal scan, the association was attenuated but remained in the same direction (51.8% vs 39.5%; crude OR 1.65; 95% CI 0.91 - 2.98; P = 0.097). The crude birthweight difference would have been abolished only if the mean birthweight of the 30 excluded women had been approximately 1.27 kg or less, against a group mean of 2.08 kg. The crude association with low birthweight was less robust: it would lose statistical significance if 26 or more of the 30 excluded women (86.7%) had a low-birthweight infant, a proportion close to the 82.1% observed in the group overall.

For the scan model, the E-value was 2.98 for the adjusted odds ratio and 1.84 for its lower confidence limit. An omitted or unmeasured confounder would therefore need to be associated with both coagulation abnormality and an abnormal scan by a risk ratio of at least 2.98, beyond the measured covariates, to explain away the observed association, and by at least 1.84 to move the confidence interval

to include the null. For comparison, delivery before 34 weeks and before 37 weeks, the available proxies for earlier gestational age at presentation, were only 1.11 and 1.20 times as frequent, respectively, in women with a coagulation abnormality as in those without (**Table 4**).

4. Discussion

In the present cross-sectional analysis of 216 women with preeclampsia with severe features, nearly two-thirds (64.8%) had at least one coagulation abnormality, driven mainly by thrombocytopenia (48.6%) and elevated D-dimer (39.4%), whereas prolonged aPTT (19.4%) and PT (16.7%) were less frequent. Abnormal coagulation clustered with severe-range blood pressure (93.6% vs 60.5%), renal impairment (55.7% vs 18.4%) and hepatic involvement (25.7% vs 2.6%), and was independently associated with threefold higher odds of an abnormal antenatal scan and a 151-g lower birthweight.

These findings indicate that haemostatic derangement in severe preeclampsia is not an isolated laboratory observation but a marker of more advanced endothelial and multi-organ disease. Because the birthweight association persisted after adjustment for gestational age at delivery, and preterm delivery did not differ between groups, the deficit is more plausibly explained by impaired fetal growth from placental malperfusion than by earlier delivery alone. This inference should be made with caution. Gestational age at delivery may itself be shortened by severe disease, so the adjusted coefficient (−151 g) excludes any part of the association that operates through earlier delivery, whereas the unadjusted difference (−220 g) includes it, together with any confounding by the maternal covariates. Both estimates point in the same direction, and birthweight-for-gestational-age centiles would give a more direct measure of fetal growth in future work. The fetal growth restriction comparison itself was borderline and sensitive to the statistical test used, and we do not rely on it. The predominance of thrombocytopenia and D-dimer elevation over prolonged clotting times is also biologically coherent: platelet activation and consumption at injured endothelium, with local fibrin turnover, typically precede the depletion of circulating coagulation factors that prolongs PT and aPTT in more advanced consumptive coagulopathy.

Our results agree with meta-analytic evidence of prolonged PT and aPTT in preeclampsia [11] and with Peng *et al.*, who found that falling platelet count and rising aPTT and D-dimer tracked disease severity and poor pregnancy outcomes [12]. Lower platelet counts in severe disease [13], reduced neonatal birthweight [14], and similar derangements in an Ethiopian cohort [15] and in relation to adverse neonatal outcomes [16] further support our observations. In contrast, the AngioPred cohort reported no predictive value of D-dimer [17], and Li *et al.* identified placental growth factor and cholesterol, rather than conventional coagulation tests, as independent predictors in severe preeclampsia [19]. Notably, our D-dimer threshold (1.7 mg/L FEU, the laboratory's upper reference limit) is more than three times the 0.5 mg/L FEU cut-off conventionally used outside pregnancy,

yet it lies between the mean late-pregnancy (1.48 mg/L) and term (1.89 mg/L) values reported in a large pregnancy reference dataset [18]; some physiological elevations, particularly near term, were therefore probably classified as abnormal. If unrelated to fetal status, such misclassification would be expected to bias estimates toward the null, although this cannot be verified in our data. D-dimer results are also assay-dependent, and values expressed in FEU are approximately twice those expressed in D-dimer units, so the threshold should not be transferred to other platforms without local verification.

The timing of the measurements determines how far these findings can inform practice. Blood was sampled at admission and the scan was performed during the same admission, at or after sampling, so the coagulation result was available before or together with the scan findings, but the two were effectively concurrent. Our data therefore show that an abnormal admission coagulation profile identifies women who are more likely to have an abnormal scan at that admission; they do not show that it predicts fetal deterioration developing later. Clinically, where angiogenic biomarkers are unaffordable, as in much of Bangladesh, a simple admission panel of platelet count, PT, aPTT and D-dimer could help to prioritise early ultrasound and Doppler assessment, closer multidisciplinary review and neonatal preparedness when scanning capacity is limited. It should not be used to withhold or delay fetal assessment in women with normal results, 39.5% of whom also had an abnormal scan. Our data also underscore the need for gestation-specific haemostatic reference intervals and justify prospective evaluation of whether coagulation-guided surveillance improves perinatal outcomes.

This study has limitations. The single-centre cross-sectional design precludes causal and temporal inference, and tertiary referral may limit generalisability. Coagulation tests were measured once at admission using laboratory reference limits that are not gestation-specific, and platelet count partly overlaps the diagnostic criteria for severe features. This overlap may inflate the prevalence estimate, which nevertheless remained high (59.1%) after exclusion of women with a platelet count $<100,000/\mu\text{L}$. It is unlikely to have produced the associations with maternal severity markers, because women without a coagulation abnormality could enter the cohort only through a non-platelet severe feature, a selection effect that would favour higher, not lower, frequencies of those markers in the comparison group. The crude scan and birthweight associations were robust to all but extreme assumptions about the excluded women, but the bounding analysis was unadjusted and the low-birthweight association was less robust. Gestational age at admission was not included in the scan model, so residual confounding by early-onset disease cannot be excluded, although the E-value indicates that a confounder of considerable strength would be required to account for the association. Gestational age at delivery, adjusted for in the birthweight model, may be an intermediate variable, and birthweight centiles were not analysed. Fibrinogen, thrombin time and angiogenic markers were not measured. Finally, antenatal scan findings were abstracted from routine reports prepared without study-specific masking of sonologists

to clinical or laboratory findings, introducing possible inter-observer variability and ascertainment bias.

5. Conclusion

Coagulation abnormalities affect nearly two-thirds of women with preeclampsia with severe features and identify those with greater hypertensive, renal and hepatic severity, threefold higher odds of abnormal antenatal scans, and lower birth-weight. A low-cost admission coagulation panel may help identify women who warrant prioritised fetal ultrasound and Doppler assessment in resource-limited settings, although the scan association was concurrent with admission testing, and the panel's ability to predict subsequent fetal deterioration, and hence its clinical utility, requires prospective confirmation. Prospective multicentre studies using gestation-specific reference ranges, serial fetal assessment, angiogenic markers, and maternal and neonatal hard outcomes are now warranted.

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Author Contributions

Arifa Sharmin was responsible for the conceptualization, study design, methodology, overall supervision, project administration, statistical analysis, interpretation of findings, original manuscript drafting, critical revision, and final manuscript preparation. Quazi Rubyata Sania contributed to study methodology; Hafiza Farzana to patient recruitment; Urmila Majumder to clinical data collection; Jesika Rizvi Tamanna to laboratory data assessment; Raunak Jahan to fetal outcome assessment; Nasrin Akter to data validation; and Alif Laila to interpretation of study findings. All authors reviewed and approved the final manuscript and agreed to be accountable for the accuracy and integrity of the work.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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