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Coagulation and hematological variations in pregnancy: A cross-sectional study on hypertensive and normotensive females

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ABSTRACT

Objective: To evaluate and compare coagulation and hematological parameters in hypertensive and normotensive pregnant women.**Methods:** This present cross-sectional study was carried out in the Departments of Pathology and Obstetrics & Gynaecology at Dr. D. Y. Patil Medical College, Hospital & Research Centre, Pimpri, Pune, India from September 2023 to March 2025. Hematological parameters [platelet count, mean platelet volume (MPV), platelet distribution width (PDW)] were analyzed using an automated hematology analyzer, while coagulation parameters [prothrombin time (PT)/international normalised ratio, activated partial thromboplastin time (aPTT), and *D*-dimer] were assessed by standard automated assays. Results were compared between normotensive and hypertensive groups and correlated with disease severity.**Results:** The study included 212 antenatal females, with 106 normotensive pregnant women and 106 hypertensive women. Hypertensive women include cases of gestational hypertension ($n=55$); mild preeclampsia ($n=39$), and severe preeclampsia ($n=12$). A significant progressive decrease in platelet count and significant increases in MPV, PDW, PT, aPTT, and *D*-dimer levels were associated with increasing severity of pregnancy-induced hypertension ($P<0.001$). Women with severe preeclampsia had the lowest mean platelet counts and the highest coagulation parameter values compared to women with gestational hypertension, mild preeclampsia, and normotensive pregnancies. These findings indicate enhanced platelet activation, endothelial dysfunction, and activation of the coagulation–fibrinolytic system with worsening disease severity.**Conclusions:** Significant hematologic and coagulation abnormalities were present in women with pregnancy-induced hypertension. For better maternal-fetal outcomes and early management, routine monitoring is essential.**KEYWORDS:** Pregnancy-induced hypertension; Coagulationprofile; Platelet indices; Perinatal health; Preeclampsia; Gestational hypertension; Platelet count; *D*-dimer; Maternal outcomes

Key Points

Question: What coagulation and hematological abnormalities are present in pregnancy-induced hypertension, and how do these differ depending on the severity of the condition in comparison to pregnancies with normal blood pressure?**Findings:** The study showed that as pregnancy-induced hypertension severity increased, mean platelet volume, platelet distribution width, prothrombin time, activated partial thromboplastin time, and *D*-dimer levels gradually increased while platelet counts significantly decreased. These alterations, which include increased platelet activation, endothelial damage, and developing coagulopathy, were statistically significant.**Meaning:** Enhancing early risk detection, directing clinical decision-making, and optimizing maternal and fetal outcomes can all be achieved by incorporating their routine examination into prenatal treatment.✉ To whom correspondence may be addressed. E-mail: ypi7884@gmail.com

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1. Introduction

Pregnancy-related hypertensive disorders, particularly pregnancy-induced hypertension (PIH), are major causes of maternal and fetal morbidity and mortality and represent a significant global health concern. PIH is defined as new-onset hypertension after 20 weeks of gestation and includes a spectrum ranging from gestational hypertension to mild and severe preeclampsia[1]. These disorders affect approximately 5%–10% of pregnancies worldwide and contribute substantially to maternal mortality, particularly in low- and middle-income countries where access to adequate antenatal care may be limited[2]. The increasing incidence of PIH has been associated with rising maternal obesity, delayed childbearing, and the use of assisted reproductive technologies[3].

Preeclampsia represents a more severe manifestation of PIH, typically characterized by hypertension accompanied by proteinuria after mid-gestation. It may also be associated with clinical evidence of end-organ dysfunction, including thrombocytopenia, renal impairment, or hepatic involvement[4]. If left untreated, preeclampsia may progress to eclampsia, a life-threatening condition characterized by seizures, and may lead to serious complications such as placental abruption, stroke, multi-organ failure, and fetal death[5]. Although the exact etiology of preeclampsia and eclampsia remains unclear, their pathogenesis is believed to involve endothelial dysfunction, placental hypoperfusion, immune maladaptation, and oxidative stress[6,7], influenced further by genetic, environmental, and immunological factors[8]. PIH is also associated with multi-organ involvement in affected mothers. The characteristic placental lesion observed in preeclampsia is acute atherosclerosis of the decidual arteries, which reduces placental perfusion. Decreased placental blood flow may result in intrauterine growth restriction, fetal distress, and even fetal demise[9].

One of the important pathological features of PIH is its impact on the hemostatic system. Women with PIH frequently exhibit hematological and coagulation abnormalities, including thrombocytopenia, increased platelet activation, and disturbances in coagulation pathways[10]. These alterations create a prothrombotic state and increase the risk of complications such as thromboembolic events, postpartum hemorrhage, and disseminated intravascular coagulation[11]. Several studies have reported significant changes in coagulation parameters, including platelet count, prothrombin time (PT), activated partial thromboplastin time (aPTT), and fibrinogen levels in patients with preeclampsia and eclampsia[12]. Among these abnormalities, thrombocytopenia is often the earliest hematological change detected and is widely considered an indicator of disease severity[13]. PIH is therefore regarded as one of the most serious hypertensive disorders of pregnancy and is associated with increased maternal morbidity and mortality[14].

Timely evaluation of hematological and coagulation profiles in women with PIH is essential for identifying high-risk cases and initiating appropriate clinical management[15]. Routine monitoring of platelet counts helps in the early detection of thrombocytopenia, which may increase the risk of maternal hemorrhage during delivery. Assessment of PT, aPTT, and other coagulation parameters assists in identifying coagulopathy and guiding clinical decisions regarding anticoagulant therapy and timing of delivery[16]. Such evaluation is particularly important in severe preeclampsia and eclampsia, where the risk of coagulation-related complications is significantly increased[6].

Despite extensive research, the prognostic value of specific hematological and coagulation parameters in predicting maternal and perinatal outcomes in PIH remains controversial[17]. Some studies have demonstrated a strong correlation between disease severity and altered coagulation profiles, whereas others have reported inconsistent findings[18]. These variations may be attributed to differences in laboratory methodologies, patient populations, and clinical management practices, which make it challenging to establish standardized diagnostic and management guidelines.

Therefore, the present study aims to compare hematological indices and coagulation profiles between pregnant women with PIH and normotensive pregnant women. Understanding these hemostatic alterations may contribute to improved diagnostic evaluation and better management strategies for complications associated with hypertensive disorders of pregnancy.

2. Methods

2.1. Study design and setting

This cross-sectional study was conducted over a period of 18 months, from September 2023 to March 2025. The study was carried out in the Departments of Pathology and Obstetrics & Gynecology at Dr. D. Y. Patil Medical College, Hospital & Research Centre, Pimpri, Pune, India.

2.2. Study population and sample size

The study included 212 pregnant women in all. There were two primary categories of participants. The control group included 106 age- and gestational age-matched normotensive pregnant women; The study group included 106 pregnant women with hypertensive disorders of pregnancy.

The sample size for the present study was determined based on the expected prevalence of PIH reported in previous studies, which ranges from 5% to 10% of all pregnancies. Using a 95% confidence level and a margin of error of 5%, the minimum required sample size

was calculated using the standard formula:

$$n = Z^2 \times p \times q / d^2$$

Where:

n = required sample size; Z = standard normal deviate at 95% confidence level (1.96); p = estimated prevalence of PIH (10% or 0.10); $q = 1 - p$; d = margin of error (0.05).

Based on this calculation and considering the availability of eligible patients during the study period, a total sample size of 212 pregnant women was included in the study to ensure adequate comparison between hypertensive and normotensive groups.

The participants were divided into two main groups: 1) The control group: 106 normotensive pregnant women; 2) The study group: 106 pregnant women diagnosed with hypertensive disorders of pregnancy.

Based on clinical diagnosis, the study group was further stratified into: 1) Gestational hypertension ($n=55$); 2) Mild preeclampsia ($n=39$); 3) Severe preeclampsia ($n=12$).

2.3. Inclusion and exclusion criteria

Pregnant women with a gestational age greater than 20 weeks who were diagnosed with PIH were included as cases in the study. The control group consisted of age- and gestational age-matched normotensive pregnant women attending the antenatal clinic during the study period. The hypertensive cases were clinically classified into gestational hypertension, mild preeclampsia, and severe preeclampsia based on blood pressure measurements and associated clinical findings. Gestational hypertension was defined as systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg without proteinuria. Mild preeclampsia was defined as systolic blood pressure between 140–160 mmHg or diastolic blood pressure between 90–110 mmHg with the presence of proteinuria (+1). Severe preeclampsia was defined as systolic blood pressure ≥ 160 mmHg or diastolic blood pressure ≥ 110 mmHg associated with significant proteinuria and/or clinical features such as persistent headache, oliguria (<400 mL/24 hours), serum creatinine >1.2 mg/dL, elevated liver enzymes [aspartate aminotransferase/alanine aminotransferase (AST/ALT)], pulmonary edema, or seizures not attributable to other causes.

Pregnant women with pre-existing hypertension, diabetes mellitus, renal disease, coagulation disorders, hepatitis, sepsis, idiopathic thrombocytopenic purpura (ITP), seizure disorders, or a history of severe trauma were excluded from the study. Additionally, women with placental abnormalities such as placental abruption or placenta previa were not included. Patients who were taking medications other than routine antenatal supplements (multivitamins, calcium, and iron) that could influence hematological or coagulation parameters were also excluded. Furthermore, women with a history of smoking or alcohol consumption were excluded to avoid potential

confounding effects on hematological parameters.

2.4. Primary and secondary study indices

The primary study indices included evaluation of hematological and coagulation parameters in pregnant women. These parameters comprised platelet count, mean platelet volume (MPV), platelet distribution width (PDW), PT, aPTT, international normalized ratio (INR), and D -dimer levels, which were measured and compared between hypertensive and normotensive pregnant women.

The secondary study indices included assessment of the association between platelet indices and coagulation parameters with the severity of hypertensive disorders of pregnancy. In addition, correlations between the laboratory parameters and clinical features of the disease were evaluated. The study also aimed to assess maternal and perinatal outcomes in relation to the observed hematological and coagulation abnormalities.

2.5. Laboratory procedures

In accordance with manufacturer-recommended procedures, coagulation profiles (PT/INR, aPTT, and D -dimer) were evaluated using the ACL TOP 300 Coagulation Analyzer and platelet parameters were examined using the Beckman Coulter DxH 900 CBC Analyzer.

2.6. Data collection

A standardized proforma was used to record sociodemographic information including maternal age, parity, and gestational age, obstetric history such as gravidity, previous pregnancy complications, and antenatal care status, clinical findings, laboratory data, and maternal and perinatal outcomes.

2.7. Statistical analysis

All collected data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Comparison between normotensive and hypertensive groups was performed using the independent sample t -test for normally distributed continuous variables. When more than two groups were compared (gestational hypertension, mild preeclampsia, and severe preeclampsia), one-way analysis of variance (ANOVA) followed by *post-hoc* analysis was used. The *Chi-square* test was applied to evaluate associations between categorical variables. For non-normal distribution parameters, group comparisons were performed using the Kruskal–Wallis test, and data are expressed as median and interquartile range (IQR). Continuous variables such as platelet count, MPV, PDW, PT, aPTT, INR, and D -dimer levels were expressed as mean $\pm$ standard deviation (mean $\pm$ SD), while categorical

variables were presented as frequency and percentage [$n(\%)$]. A P -value <0.05 was considered statistically significant.

2.8. Ethics statement

The study was approved by the Institutional Ethics Committee (Ethical Clearance No. Dean/North DMC/MC/2021/1031, dated 27-12-2021). Written informed consent was obtained from all participants.

3. Results

3.1. Gravida distribution

The distribution of cases according to gravida status showed that primigravida women accounted for 36.4% of gestational hypertension, 46.2% of mild preeclampsia, and 16.7% of severe preeclampsia cases. Gravida 2 constituted the majority of gestational hypertension cases (50.9%), followed by mild preeclampsia (30.8%) and severe preeclampsia (8.3%). Gravida 3 was observed in 10.9% of gestational hypertension, 17.9% of mild preeclampsia, and 41.7% of severe preeclampsia cases. Multigravida women accounted for 1.8% of gestational hypertension, 5.1% of mild preeclampsia, and 33.3% of severe preeclampsia cases (Figure 1A).

3.2. Age distribution

Participants in the study and control groups were (25.7 ± 4.4) years old on average. The age distribution of the groups did not change statistically significantly ($P=0.13$) (Table 1). According to an analysis of age groups, women between the ages of 18 and 24 years were more likely to have mild preeclampsia and gestational hypertension, whereas women over 30 years were more likely to have severe preeclampsia (Figure 1B).

3.3. Platelet count

As the severity of pregnancy-related hypertension problems increased, a steady decrease in mean platelet count was noted. The average platelet counts were ($240\,587.64\pm67\,303.17$) μL in normotensive pregnant females, ($199\,057.16\pm46\,168.47$) μL in those with gestational hypertension, ($103\,390.92\pm23\,545.67$) μL in those with mild preeclampsia, and ($37\,886.08\pm10\,806.54$) μL in those with severe preeclampsia. Table 1 shows that this declining trend was statistically significant ($P<0.001$). The pathophysiology of PIH is characterized by endothelial damage and increased platelet activation, which are the likely causes of the observed decrease in platelet count with increasing illness severity (Table 2).

3.4. MPV

MPV showed a notable rising trend as PIH severity increased. In normotensive pregnancies, the mean MPV values were (9.71 ± 1.35) fL; in gestational hypertension, they were (8.87 ± 1.15) fL; in mild preeclampsia, they were (10.91 ± 2.42) fL; and in severe preeclampsia, they were (26.76 ± 11.80) fL. Table 1 shows that this gradual rise was statistically significant ($P<0.001$). Larger and more reactive platelets contribute to the prothrombotic condition seen in severe hypertensive pregnancy disorders, and elevated MPV indicates enhanced platelet activation (Table 2).

3.5. PDW

Similarly, PDW increased significantly in parallel with disease severity. The mean PDW values were (12.92 ± 1.84)% in normotensive pregnancies, (14.09 ± 1.98)% in gestational hypertension, (17.43 ± 3.43)% in mild preeclampsia, and (38.04 ± 18.42)% in severe preeclampsia. This trend was statistically significant ($P<0.001$) (Table 1). The progressive elevation in PDW

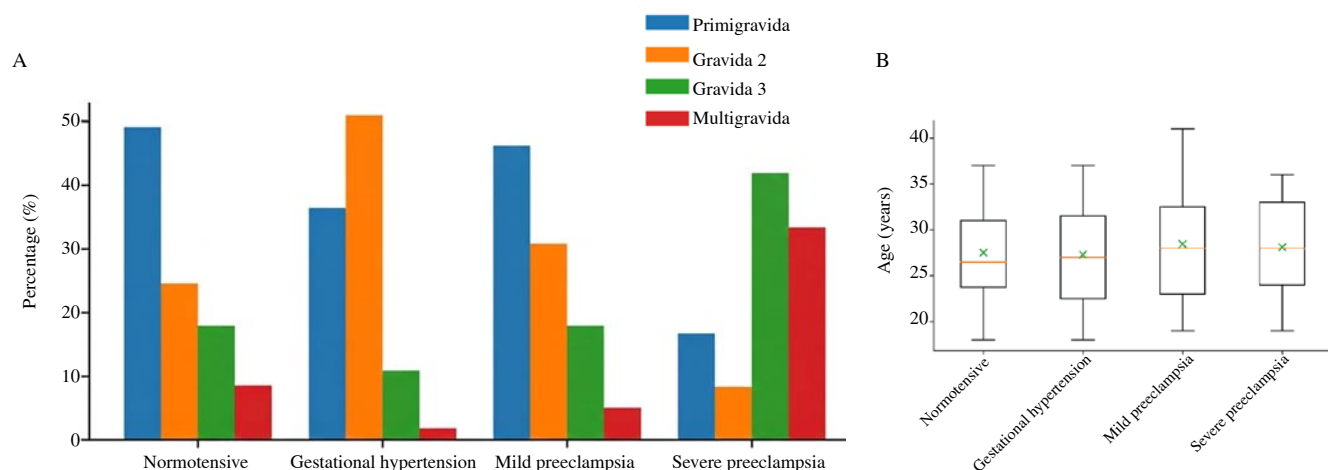


Figure 1. (A) Gravida status in normotensive women or women with gestational hypertension, mild preeclampsia, and severe preeclampsia. **(B)** Comparison of maternal age across hypertensive disorders of pregnancy. Box plots represent median, interquartile range, and range; crosses indicate mean age.

Table 1. Comparison of all parameters between groups.

Parameters	Normotensive (n=106)	Gestational hypertension (n=55)	Mild preeclampsia (n=39)	Severe preeclampsia (n=12)	Total (n=212)	P-value
Age, years	25.3±4.0	26.0±4.4	25.8±4.9	28.6±5.0	25.7±4.4	0.13
Gestational age, weeks	35.4±3.0	35.5±2.5	35.2±3.0	35.6±2.7	35.4±2.8	0.93
Platelet count, ×10 ³ /uL	240587.64±67303.17	199057.16±46168.47	103390.92±23545.67	37886.08±10806.54	193100.53±83098.23	<0.001
Mean platelet volume (MPV), fL	9.71±1.35	8.87±1.15	10.91±2.42	26.76±11.80	10.68±5.06	<0.001
Platelet distribution width (PDW), %	12.92±1.84	14.09±1.98	17.43±3.43	38.04±18.42	15.47±7.48	<0.001
Prothrombin time (PT), seconds	11.1±0.9	12.9±1.5	14.3±2.1	20.4±3.1	12.7±2.7	<0.001
Activated partial thromboplastin time (aPTT), seconds	25.2±2.1	27.5±3.1	30.4±4.7	37.8±2.7	27.5±4.4	<0.001
D-dimer, ng/mL	666.54±95.88	792.85±249.19	1426.97±953.88	3361.67±1925.30	991.75±891.88	<0.001

Data are expressed as mean±SD.

indicated increased heterogeneity in platelet size, reflecting enhanced platelet activation, turnover, and consumption—characteristic of the hypercoagulable state in advanced PIH (Table 2).

3.6. PT

The mean PT values increased progressively with worsening severity of PIH. The mean PT values were (11.1±0.9) seconds in normotensive pregnancies, (12.9±1.5) seconds in gestational hypertension, (14.3±2.1) seconds in mild preeclampsia, and (20.4±3.1) seconds in severe preeclampsia. The difference among the groups was statistically significant ($P<0.001$) (Table 1).

3.7. aPTT

As PIH severity increased, aPTT likewise showed a substantial extension ($P<0.001$). In normotensive pregnancies, the mean aPTT values were (25.2±2.1) seconds; in gestational hypertension, they were (27.5±3.1) seconds; in mild preeclampsia, they were (30.4±4.7) seconds; and in severe preeclampsia, they were (37.8±2.7) seconds (Table 1). In severe PIH, this gradual extension indicates a higher risk of hemostatic imbalance and bleeding due to disruption of the intrinsic coagulation pathway (Table 2).

3.8. D-dimer levels

Mean D-dimer levels demonstrated a statistically significant rise corresponding to the increasing severity of PIH ($P<0.001$). The mean values were (666.54±95.88) ng/mL in normotensive pregnancies, (792.85±249.19) ng/mL in gestational hypertension, (1426.97±953.88) ng/mL in mild preeclampsia, and (3361.67±1925.30) ng/mL in severe preeclampsia (Table 1). The marked elevation of D-dimer levels reflects enhanced thrombin generation and fibrinolytic activity associated with advancing PIH. These findings underscore the diagnostic and prognostic utility of D-dimer as a biochemical marker for disease severity and thrombotic risk in hypertensive disorders of pregnancy.

4. Discussion

Preeclampsia and other hypertensive disorders of pregnancy (HDP) continue to be a major global cause of maternal and perinatal morbidity and mortality. These disorders are characterized by widespread endothelial dysfunction, enhanced inflammatory responses, platelet activation, and dysregulation of the coagulation–fibrinolytic system. Understanding the hematological and coagulation changes associated with disease severity is essential for early risk assessment and timely clinical intervention. Parity has long been recognized as an important epidemiological factor in hypertensive disorders of pregnancy. Primigravida women are more frequently affected by gestational hypertension and mild preeclampsia, whereas women with higher parity may be more prone to severe disease, possibly due to cumulative vascular or placental maladaptation across multiple pregnancies[19,20]. This trend suggests that immunological and physiological changes related to parity may influence the onset and progression of hypertensive disorders of pregnancy. Maternal age is another well-recognized risk factor for HDP. Younger women are often more susceptible to gestational hypertension and milder forms of preeclampsia, whereas advanced maternal age has been consistently associated with severe preeclampsia and its complications[19,20]. This association may be explained by age-related endothelial dysfunction, reduced vascular compliance, and a higher prevalence of metabolic comorbidities.

Platelet abnormalities are among the earliest and most sensitive laboratory indicators of disease progression in preeclampsia. Thrombocytopenia in HDP results from increased platelet activation and aggregation at sites of endothelial injury, leading to accelerated platelet consumption[21–26]. Placental ischemia stimulates the release of inflammatory cytokines and anti-angiogenic factors, which shorten platelet lifespan and impair megakaryopoiesis. Numerous studies have consistently demonstrated a progressive decline in platelet count with increasing disease severity, highlighting its value as a marker of worsening pathology[19–26]. However, some studies have reported nonsignificant differences, which may reflect variations in diagnostic criteria, timing of sample collection, and

Table 2. Comparison of platelet count, mean platelet volume, platelet distribution width, prothrombin time, and activated partial thromboplastin time categories across hypertensive disorders of pregnancy.

Parameters	Normotensive (n=106)	Gestational hypertension (n=55)	Mild preeclampsia (n=39)	Severe preeclampsia (n=12)	Total (n=212)	P-value
Platelet count, cells/ μ L						<0.001
Normal (150 000–450 000)	101 (95.3)	48 (87.3)	1 (2.6)	0 (0.0)	150 (70.8)	
Borderline (140 000–149 000)	1 (0.9)	6 (10.9)	0 (0.0)	0 (0.0)	7 (3.3)	
Mild thrombocytopenia (50 000–139 999)	4 (3.8)	1 (1.8)	38 (97.4)	1 (8.3)	44 (20.8)	
Moderate thrombocytopenia (20 000–49 999)	0 (0.0)	0 (0.0)	0 (0.0)	9 (75.0)	9 (4.2)	
Marked thrombocytopenia ($\leq$ 20 000)	0 (0.0)	0 (0.0)	0 (0.0)	2 (16.7)	2 (0.9)	
Mean platelet volume (MPV), fL						<0.001
<7	2 (1.9)	1 (1.8)	0 (0.0)	0 (0.0)	3 (1.4)	
Mild (7–9)	29 (27.4)	31 (56.4)	7 (17.9)	0 (0.0)	67 (31.6)	
Moderate (9–11)	60 (56.6)	22 (40.0)	19 (48.7)	0 (0.0)	101 (47.6)	
Severe (>11)	15 (14.2)	1 (1.8)	13 (33.3)	12 (100.0)	41 (19.3)	
Platelet distribution width (PDW), %						<0.001
<12	41 (38.7)	9 (16.4)	0 (0.0)	0 (0.0)	50 (23.6)	
Mild (12–16)	60 (56.6)	37 (67.3)	14 (35.9)	0 (0.0)	111 (52.4)	
Moderate (16–20)	5 (4.7)	9 (16.4)	18 (46.2)	2 (16.7)	34 (16.0)	
Severe (>20)	0 (0.0)	0 (0.0)	7 (17.9)	10 (83.3)	17 (8.0)	
Prothrombin time (PT), seconds						<0.001
<12	90 (84.9)	10 (18.2)	5 (12.8)	0 (0.0)	105 (49.5)	
Mild (12–14)	16 (15.1)	37 (67.3)	13 (33.3)	0 (0.0)	66 (31.1)	
Moderate (14–16)	0 (0.0)	6 (10.9)	18 (46.2)	2 (16.7)	26 (12.3)	
Severe (>16)	0 (0.0)	2 (3.6)	3 (7.7)	10 (83.3)	15 (7.1)	
Activated partial thromboplastin time (aPTT), seconds						<0.001
<26	62 (58.5)	12 (21.8)	9 (23.1)	0 (0.0)	83 (39.2)	
Mild (26–32)	43 (40.6)	37 (67.3)	15 (38.5)	1 (8.3)	96 (45.3)	
Moderate (32–36)	1 (0.9)	6 (10.9)	11 (28.2)	1 (8.3)	19 (9.0)	
Severe (>36)	0 (0.0)	0 (0.0)	4 (10.3)	10 (83.3)	14 (6.6)	

Data are expressed as n (%). Chi-square test is used.

population characteristics[27,28]. In addition to platelet count, platelet indices such as MPV and PDW provide valuable insights into platelet activation and turnover. Elevated MPV indicates the presence of larger, younger, and metabolically more active platelets released from the bone marrow in response to peripheral destruction[20,29–31]. Increased MPV has been frequently associated with severe preeclampsia and is believed to contribute to the prothrombotic state observed in advanced disease[19,22]. Although most studies support the role of MPV as a marker of increased platelet reactivity in HDP, some authors have questioned its diagnostic consistency across different populations[28,32].

Similarly, PDW reflects variability in platelet size and serves as an indirect indicator of asynchronous platelet production and consumption. Progressive elevation of PDW has been reported in preeclampsia and eclampsia, correlating with endothelial injury and increasing disease severity[20,27,29,30,33]. Increased PDW reflects dynamic thrombopoiesis driven by ongoing platelet destruction and widespread microvascular thrombosis. Despite a few studies reporting nonsignificant findings, the majority of available evidence supports PDW as a useful supplementary parameter in assessing disease progression[19,28].

Coagulation abnormalities represent another important feature of severe HDP. Prolongation of PT reflects activation of the extrinsic coagulation pathway due to endothelial injury and the release of

tissue factor from damaged trophoblasts and monocytes[19,20,34,35]. As the disease progresses, continuous thrombin generation leads to consumption of coagulation factors, shifting the hemostatic balance toward consumption coagulopathy. Several studies have reported that prolonged PT is associated with an increased risk of maternal complications, including placental abruption and disseminated intravascular coagulation[23,34–36].

Prolongation of aPTT is another indicator of intrinsic pathway dysfunction and consumption of coagulation factors. Endothelial activation and microvascular thrombosis are strongly influenced by anti-angiogenic mediators such as soluble fms-like tyrosine kinase-1 (sFlt-1), which promote fibrin deposition and progressive depletion of coagulation proteins[20,24,37,38]. The consistent observation of aPTT prolongation in severe disease highlights its potential role as a marker of coagulation imbalance and impending hemostatic failure.

Elevation of D-dimer levels reflects the fibrinolytic response to widespread intravascular fibrin formation. Increased levels indicate enhanced thrombin generation, activation of plasmin, and accelerated fibrin degradation[35,38–43]. Several studies have demonstrated a strong association between elevated D-dimer levels and disease severity, suggesting that it may be useful for identifying patients at increased risk of thrombotic and obstetric complications. Although pregnancy itself is a physiologically hypercoagulable state, disproportionately elevated D-dimer levels in HDP suggest

pathological activation of the coagulation–fibrinolytic system.

The existing body of evidence therefore supports the concept that progressive platelet activation, consumption, and coagulation dysfunction play central roles in the pathophysiology and severity of hypertensive disorders of pregnancy. Incorporating platelet indices and coagulation parameters into routine antenatal evaluation may facilitate early identification of high-risk patients and timely escalation of clinical care. However, larger prospective studies are required to establish optimal cutoff values and standardized protocols for their clinical application.

This study is limited by a small sample size, particularly with only twelve severe preeclampsia cases, reducing generalizability. As a single-centre study, selection bias may exist, and variations in clinical protocols could affect result reliability. Additionally, maternal and neonatal outcomes were not tracked beyond the immediate postpartum period, limiting insights into the long-term effects of hematologic and coagulation disturbances in PIH.

In conclusion, significant hematological and coagulation abnormalities in PIH patients are revealed by this investigation. Platelet activation, endothelial damage, and coagulation imbalance were reflected in the gradual abnormalities in platelet count, MPV, PDW, PT, aPTT, and *D*-dimer that increased with the severity of the disease. Early diagnosis, illness progression, and clinical decision-making are all aided by these characteristics. Maternal-foetal outcomes and risk prediction may be enhanced by routine prenatal evaluation.

Conflict of interest statement

The authors declare no conflicts of interest to disclose.

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Authors' contributions

Concept and design were performed by Dr Anoushka Sharma and Dr Yaminy Pradeep Ingale; data collection was carried out by Dr Anoushka Sharma and Dr Shilpa Kshirsagar; data handling was done by Dr Anoushka Sharma and Dr Narendra C. Kale; analysis was made by Madhura Gandhi; manuscript preparation was performed by Dr Anoushka Sharma, Dr Yaminy Pradeep Ingale and Dr Charusheela Gore.

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