Original Article

Evaluation of Coagulation Profile in Pregnancy-induced Hypertensive Patients and Normotensive Pregnancies

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Abstract

Background: Pregnancy-induced hypertension (PIH) remains a major contributor to maternal and perinatal morbidity and mortality worldwide. The present study aimed to evaluate the coagulation profile in women with PIH compared to normotensive pregnancies, and assess severity of coagulation changes associated with PIH. **Materials and Methods:** This prospective observational study was conducted over 12 months and included 100 children aged 1 month to 18 years with convulsive SE. Data were collected on demographics, etiology, seizure characteristics, anti-epileptic drug (AED) use, need for critical care interventions, and early outcomes. Statistical analysis was performed using SPSS v22, with chi-square tests applied for categorical variables ($p<0.05$ considered significant). **Results:** Mean age of participants was 25.87±4.31 years. Women with PIH had significantly lower platelet counts (185 vs. 352 $\times 10^9$/L), lower haemoglobin (10.53 vs. 11.16 g/dL), prolonged PT (18.02 vs. 11.55 sec) and aPTT (53.23 vs. 28.99 sec), elevated INR (1.39 vs. 0.89), and markedly higher D-dimer levels (2.14 vs. 0.71 mg/dL) compared to normotensives (all $p<0.0001$). Peripheral smear revealed predominance of normocytic normochromic (44.1%) and microcytic hypochromic anaemia (39.1%), with mild thrombocytopenia in 1.82% and occasional schistocytes in 0.9% of PIH patients. **Conclusion:** PIH is associated with significant haematological and coagulation abnormalities, which correlate with disease severity. Routine assessment of coagulation profile, particularly PT, aPTT, INR, D-dimer, and platelet count, can serve as cost-effective markers for early detection of complications like HELLP syndrome and disseminated intravascular coagulation, thereby improving maternal and foetal outcomes.

Keywords: Pregnancy-induced hypertension, coagulation profile, platelet count, D-dimer.

Received: 01 July 2025 **Revised: 02 August 2025** **Accepted: 16 September 2025** **Published: 24 September 2025**

Introduction

Pregnancy is a physiological state marked by dynamic alterations in coagulation and haemostasis, designed to prevent haemorrhage during childbirth.[1] In Pregnancy-Induced Hypertension (PIH), these mechanisms become maladaptive, posing maternal and foetal risks.[2] PIH encompasses gestational hypertension, preeclampsia, and eclampsia, defined by hypertension (systolic ≥140 mmHg and/or diastolic ≥90 mmHg) after 20 weeks of gestation, with or without proteinuria and systemic involvement.[3] Globally, PIH complicates 5–10% of pregnancies and remains a leading cause of maternal and perinatal mortality.[4-6] The pathophysiology involves endothelial dysfunction, placental ischemia, and systemic inflammation, resulting in hypercoagulability and consumption of clotting factors.[7-10] Preeclampsia is frequently associated with thrombocytopenia, prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), and elevated fibrin degradation products, with abnormalities worsening with disease severity.[7-10] Such derangements predispose to complications including disseminated intravascular coagulation (DIC) and HELLP syndrome. Regular assessment of coagulation parameters enables early risk detection and intervention.[8,10-12] Key tests—PT, aPTT, platelet count, fibrinogen, and D-dimer—offer a comprehensive view of haemostasis. While normotensive pregnancies show a protective hypercoagulable state, PIH exacerbates platelet consumption and coagulation activation, leading to adverse outcomes such as placental abruption, intrauterine growth restriction, and maternal or foetal mortality.[8,10-13] Platelet indices like Mean Platelet Volume and Platelet Distribution Width have emerged as cost-effective markers of platelet activation and PIH severity.[10,12] This study compares the coagulation profiles of normotensive pregnancies and PIH cases beyond 20 weeks' gestation, and aims to assess the severity of coagulation changes in PIH cases.

Materials and Methods

The present cross-sectional study was conducted in the Department of Pathology at Gandhi Medical College, Bhopal, over a period of 18 months (May 2023–October 2024), following approval from the Institutional Ethics Committee. Pregnant women with gestational age >20 weeks admitted to

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DOI:
10.21276/amit.2025.v12.i3.85

How to cite this article: Dad A, Sulya M, Gupta S. Evaluation of Coagulation Profile in Pregnancy-induced Hypertensive Patients and Normotensive Pregnancies. Acta Med Int. 2025;12:307-311.

the Department of Obstetrics and Gynaecology were enrolled. The two groups studied included the normotensive group (pregnant women without hypertension or complications) and the PIH group (women diagnosed with gestational hypertension or preeclampsia, defined as new-onset systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg, with or without proteinuria (≥1+ on dipstick)).

The required sample size was calculated using the formula $4pq/d^2$, with prevalence (p) of hypertensive disorders in pregnancy at 7.8%,[10] q = 92.2%, and allowable error (d) of 5%. This yielded 115, which was rounded to 110 per group. Thus, 110 PIH cases and 110 normotensive pregnant women were included. Inclusion criteria were pregnant women >20 weeks' gestation fulfilling group definitions. Exclusion criteria comprised pre-existing renal disease, diabetes mellitus, chronic hypertension, bleeding or platelet disorders, hepatic disorders, and eclampsia.

After informed consent, demographic, obstetric, and clinical details were recorded on a pre-designed semi-structured proforma. Venous blood samples were obtained aseptically and analyzed within 1–3 hours.

Statistical Analysis: Data were compiled in Microsoft Excel and analyzed using IBM SPSS trial version 25. Continuous variables were summarized as mean ± standard deviation, and categorical variables as frequencies and percentages. Mann-Whitney U test and Chi-square test were applied for analysis. A p-value <0.05 was considered statistically significant.

Results

Most participants (77.3%) were aged 21–30 years, reflecting peak reproductive age. Smaller groups included 15% aged 31–40 years, 6.8% aged ≤20 years and 0.9% aged 41-50 years. The mean age was 25.87 ± 4.31 years (range: 18–41 years). Out of 220 participants, 110 (50%) were normotensive, while the remaining 110 (50%) were diagnosed with Pregnancy Induced Hypertension (PIH). Among those with PIH, 48 women (21.8%) were diagnosed as gestational hypertension and 62 women (28.2%) had pre-eclampsia. Among pregnancy-induced hypertensive women, 47.3% were primigravida and 52.7% multigravida, while in the normotensive group, 44.5% were primigravida and 55.5% multigravida. In terms of parity, most hypertensive women were P1 (45.5%) and P2 (31.8%), whereas normotensives were more evenly distributed across P1 (38.2%), P2 (32.7%), and P3 (16.4%). Very few in both groups had parity ≥4. [Table 1] shows the baseline characteristics of participants. The mean gestational age was 38.04 ± 2.29 weeks. Mean SBP and DBP were 130.00±16.89 mmHg and 85.45±11.83 mmHg, respectively. Platelet counts averaged 270 ×10⁹/L, with mean haemoglobin of 10.85 g/dL, indicating mild anaemia in many cases. As shown in [Table 2], proteinuria was absent in 59.1% but present in 40.9% of women, with 16.4% having 1+ and 11.8% having 2+ albuminuria. Peripheral smear findings [Table 3] revealed normocytic normochromic anaemia (44.1%) and microcytic hypochromic anaemia (39.1%) as the predominant patterns, with thrombocytopenia observed in a small proportion. Occasional schistocytes were seen in 0.9% patients. [Table 4] highlights significant differences between hypertensive and normotensive groups. Women with PIH had lower mean gestational age (37.62 vs. 38.45 weeks, p=0.018), prolonged PT (18.02 vs. 11.55 sec), aPTT (53.23 vs. 28.99 sec), and higher INR (1.39 vs. 0.89), D-dimer (2.14 vs. 0.71 mg/dL), and reduced platelet count (185 vs. 352 ×10⁹/L), all with p<0.0001. Haemoglobin was also significantly lower in the hypertensive group (10.53 vs. 11.16 g/dL, p<0.0001). Table 5 further demonstrates that the majority of hypertensive women had abnormal coagulation parameters—76.4% with PT >16.5 sec, 74.5% with aPTT >42.5 sec, and 87.3% with INR >1.2—whereas nearly all normotensive women had values within normal limits (p < 0.0001).

Table 1: Baseline characteristics of study participants

Parameter	Minimum	Maximum	Mean±S.D.
Gestational Age (weeks)	30	42	38.04±2.29
SBP- Systolic Blood Pressure (mmHg)	100	160	130.00±16.89
DBP- Diastolic Blood Pressure (mmHg)	60	110	85.45±11.83
Post-prandial Blood Sugar (mg/dL)	94	138.0	127.64±7.60
Random Blood Sugar (mg/dL)	76	135	96.34±11.66
BT- Bleeding time (sec)	72	400	143.39±50.29
CT- Clotting time (sec)	190	740	391.72±201
Platelet count ($x10^9$/L)	70	510	270±101
aPTT	20.0	115.0	41.01±17.75
PT-Prothrombin time	10.0	22.0	14.76±3.55
D-dimer	0.30	5.20	1.41±0.94
Haemoglobin	7.8	13.4	10.85±1.10

Table 2: Distribution of study participants according to urine albumin levels

Urine Albumin levels	Frequency	Percentage (%)
Absent	130	59.1%
Trace	28	12.7%
1+	36	16.4%
2+	26	11.8%

Table 3: Distribution of study participants according to peripheral smear findings

Peripheral Smear Findings	Frequency	Percentage (%)
Dimorphic anaemia	2	0.9%
Dimorphic anaemia with mild thrombocytopenia	1	0.5%
Dimorphic anaemia with moderate thrombocytopenia	1	0.5%
Dimorphic anaemia with neutrophilic leukocytosis	2	0.9%
Microcytic hypochromic anaemia	86	39.1%
Microcytic hypochromic anaemia with mild thrombocytopenia	18	8.2%
Microcytic hypochromic anaemia with moderate thrombocytopenia	3	1.4%
Microcytic hypochromic anaemia with neutrophilic leukocytosis	1	0.5%
Normocytic hypochromic anaemia	3	1.4%
Normocytic normochromic anaemia	97	44.1%
Normocytic normochromic anaemia with mild thrombocytopenia	3	1.4%
Normocytic normochromic anaemia with moderate thrombocytopenia	1	0.5%
Normocytic normochromic anaemia with neutrophilic leukocytosis	1	0.5%
Normocytic normochromic anaemia with neutrophilic leukocytosis with mild thrombocytopenia	1	0.5%
Total	220	100.0
Thrombocytopenia		
Mild thrombocytopenia	4	1.82%
Moderate thrombocytopenia	3	1.36%
Occasional Schistocytes		
Microcytic Hypochromic Anaemia with Occasional Schistocytes	2	0.9%

Table 4: Comparison of mean values of various parameters between pregnancy-induced hypertensive and normotensive groups

Parameters	Pregnancy-induced Hypertensive	Normotensive	p-value
Mean Age (in years) ±S.D.	25.86±4.45	25.88±4.19	0.664
Mean Gestational Age (weeks) ±S.D.	37.62±2.55	38.45±1.94	0.018
Mean SBP (in mmHg) ±S.D.	145.25±8.18	115.30±7.38	<0.0001
Mean DBP (in mmHg) ±S.D.	95.37±7.42	75.89±5.94	<0.0001
Mean PPBS (in mg/dL) ±S.D.	129.38±6.36	125.83±8.36	0.070
Mean RBS (in mg/dL) ±S.D.	97.00±11.87	95.71±11.47	0.152
Mean BT (in seconds) ±S.D.	147.24±53.98	139.71±46.44	0.374
Mean CT (in seconds) ±S.D.	395±103	389±103	0.607
Mean PT (in seconds) ±S.D.	18.02±1.93	11.55±0.83	<0.0001
Mean aPTT (in seconds) ±S.D.	53.23±17.64	28.99±5.72	<0.0001
Mean INR ±S.D.	1.39±0.15	0.89±0.06	<0.0001
Mean D-dimer (mg/dL) ±S.D.	2.14±0.85	0.71±0.21	<0.0001
Mean Hb (mg/dL) ±S.D.	10.53±1.20	11.16±0.91	<0.0001
Mean platelet count ($x10^9$/L) ±S.D.	185±56	352±56	<0.0001

Mann-Whitney U test applied

Table 5: Comparison of coagulation parameters between pregnancy-induced hypertensive and normotensive groups

Parameters		Pregnancy-induced Hypertensive	Normotensive	Total	p-value
Prothrombin time (in seconds)	≤16.5	26 (23.6%)	110 (100.0%)	136 (61.8%)	<0.0001
	>16.5	84 (76.4%)	0 (0.0%)	84 (38.2%)	
aPTT (in seconds)	≤42.5	28 (25.5%)	109 (99.1%)	137 (62.3%)	<0.0001
	>42.5	82 (74.5%)	1 (0.9%)	83 (37.7%)	
INR	≤1.2	14 (12.7%)	110 (100.0%)	124 (56.4%)	<0.0001
	>1.2	96 (87.3%)	0 (0.0%)	96 (43.6%)	

Chi-square test applied

Discussion

In present study, mean age of participants was 25.87±4.31 years, with no statistically significant difference between PIH group and normotensive group (p=0.664). This aligns with Shekar A. et al.[5] (PIH: 23.98±1.42 vs. Normotensive: 24±3.3 years), Indora P et al.[12] (PE: 25.17±5.67 vs. 23.22±2.66 years, *p*=0.67), Khan MNS et al.[11] (26.19±5.02 vs. 25.17±5.68, *p*=0.384), and Xu C et al.[14] (30.09±4.51 vs. 29.44±3.74, *p*=0.067).

Findings in present study indicated a slightly higher proportion of primigravida women in PIH (47.3%) compared to normotensive group (44.5%). This is supported by Priya MJ et al.[15] who found primigravida status predominant in preeclampsia (55%), severe preeclampsia (75%), and eclampsia (80%). Similarly, Chaithra H. et al.[16] reported 62.7% primigravida in the PIH group versus 44% in controls. Chaware SA et al.[13] documented 62.5% primigravida in PIH. A statistically significant lesser gestational age was observed in PIH vs. normotensives (*p*=0.018) in present study. This was consistent with Shekar A. et al.[5] (260.02±3.47 vs. 270.2±11.4 days) and Xu C et al. (2021)[14] (PE: 35.48±3.67 vs. Controls:

39.47±0.91 weeks, $p<0.001$).

In present study, 16.4% had 1+ albuminuria, and 11.8% had 2+ proteinuria. Dundy G et al.[17] reported progressive severity: 1+ to 3+ proteinuria increasing from mild PE to eclampsia. Chaware SA et al.[13] also found higher proteinuria in severe preeclampsia and eclampsia (≥2+).

A significantly reduced platelet count was seen in PIH versus normotensives ($p<0.0001$) in present study. This observation is consistent with Tasleem S et al.[4] (1.51 vs. 2.31 lakh/cumm, $p<0.0001$), Shekar A. et al.[5] (1.07±0.22 vs. 2.07±0.40 lakh/cumm, $p=0.0001$), Indora P et al.[12] (1.74±0.75 vs. 3.07±0.59 lakh/µL, $p<0.0001$), Bhutani N et al.[10] [(2.85±0.73 (controls), 2.01±0.54 (gestational HTN), 1.81±0.46 (mild PE), 1.66±0.85 (severe PE), $p<0.0001$)], and Boddapati A et al.[18] (p=0.008). Platelet count consistently declines with PIH severity and is one of the most reliable haematological markers in these patients.

This study reported prolonged PT in PIH cases versus normotensives ($p<0.0001$). Comparable findings were reported by Shekar A. et al.[5] (15.31±1.13 vs. 13.10±0.93, $p=0.0001$), Meena P et al.[20] (14.30±1.67 vs. 12.62±0.82; $p=0.000$) and Chaithra H. et al.[16] (16.70±1.82 vs. 12.25±1.12; $p<0.001$). Similarly, Bhutani N et al.[10] reported a progressive increase in PT with severity (16.59s-gestational hypertension, 17.61s-mild preeclampsia, 18.88s-severe preeclampsia; 12.95s-normotensive women ($p<0.0001$). Indora P et al.[12] showed mean PT values of 13.86±1.84 s in preeclampsia and 16.01±2.23 s in eclampsia versus 11.23±1.25 s in normotensive pregnancies ($p < 0.0001$). On other hand, Tasleem S et al.[4] ($p=0.085$) and Sharma UK et al.[19] ($p>0.05$) found non-significant differences, likely due to smaller sample sizes or milder PIH cases. However, majority studies corroborate our findings, reinforcing the value of PT as a reliable and accessible marker of coagulation dysfunction in hypertensive disorders of pregnancy.

The aPTT was significantly prolonged in PIH group ($p<0.0001$) in present study. This finding corroborates with Shekar A. et al.[5] (35.20±4.85 vs. 29.40±2.28, $p=0.0001$), Meena P et al.[20] (33.99±4.50 vs. 31.00±1.62; $p=0.000$), and Chaithra H. et al.[16] (32.73±2.30 vs. 25.59±3.45, $p<0.001$). Bhutani N et al.[10] observed progressive aPTT elevation with disease severity—41.85s-severe preeclampsia, 38.79s-mild preeclampsia, 33.55s-gestational hypertension, as against 25.76s- normotensive pregnancies (p<0.0001). Indora P et al.[12] found increased aPTT in preeclampsia (31.52±7.81 s) and eclampsia (34.23±5.22 s) compared to controls (27.84±4.64 s; p=0.0004). However, Tasleem S et al.[4] and Priya MJ et al.[15] reported non-significant values ($p=0.34$, $p=0.370$), suggesting variability depending on PIH severity. Despite these few discrepancies, majority studies support findings of our study, affirming the relevance of aPTT as a sensitive marker of coagulation dysfunction in hypertensive disorders of pregnancy.

INR was markedly raised in hypertensive cohort ($p<0.0001$) in present study. This mirrors findings of Priya MJ et al.[15] where INR was elevated in 35–70% hypertensive cases, and Boddapati A et al.[18] ($p = 0.05$).

This study observed no statistically significant difference in BT ($p=0.374$) or CT ($p=0.607$) between PIH and normotensive women. The findings regarding BT are supported by multiple studies. Priya MJ et al.[15] reported no significant difference in BT between groups (p = 0.76). Similarly, Sharma UK et al.[19] found that BT increased only between mild and severe PE groups (BT: 87.87±7.48 s in mild PE vs. 102.50±10.15 s in severe PE, and 103.27±16.78 s in eclampsia), though this was not significant (p>0.05). Chaithra H. et al.[16] also reported a marginally higher BT in PE cases (2.56 minutes vs. 2.30 minutes). Conversely, our findings diverge from those of Tasleem S et al,[4] who found a statistically significant prolongation of BT in PE cases (2.56±0.67 minutes) compared to normotensive controls (2.30±0.32 minutes; p=0.02), suggesting that in certain populations, BT may serve as a sensitive marker of platelet dysfunction or vascular changes associated with PE. Further, Bhutani N et al.[10] reported BT>5 minutes in 61.11% mild PE and 50% severe PE patients (p=0.05). These findings suggest that while BT may not always be significantly altered, in more advanced or specific clinical subsets, it may be prolonged and clinically relevant.

Regarding CT, Indora P et al.[12] observed no statistically significant difference in CT values among PE (282±56.14 s), eclampsia (304.07±66.37 s), and control groups (309.66±77.31 s) aligning with our results. Priya MJ et al.[15] similarly reported non-significant differences (p=0.198). Sharma UK et al.[19] also demonstrated a relatively stable trend in CT values across different severity groups (range: 144.50-156.17s). In contrast, Tasleem S et al.[4] observed only a modest difference in CT (4.13±0.47 vs. 3.98±1.09 minutes); p=0.37, still broadly aligning with our findings.

The present study reported significantly elevated D-dimer in PIH ($p<0.0001$). Dundy G et al.[17] also demonstrated substantial D-dimer elevation (2038.74±510.7 (mild), 5922.4±3441.45 (severe), 5565.07±2434.63 (eclampsia) vs. 413.12±58.52 (controls), $p<0.0001$), and Khan MNS et al.[11] found significant associations with elevated fibrin degradation products ($p=0.006$). Xu C et al.[14] also demonstrated higher D-dimer levels in preeclamptic women (807.6±644.35 mg/L) in comparison to healthy pregnant controls (687.13±342.87 mg/L), p=0.028). These findings reinforce the role of elevated D-dimer as a reliable marker of endothelial dysfunction and ongoing intravascular coagulation in hypertensive disorders of pregnancy, supporting its utility in clinical assessment and risk stratification of affected women.

The present study also demonstrated a significant lower mean haemoglobin level PIH (10.53±1.20 g/dL) compared to normotensive pregnant women (11.16±0.91 g/dL), p<0.0001), indicating a notable association between hypertensive disorders and anaemia. This is consistent with Boddapati A et al,[18] who reported a mean haemoglobin level of 10.6±2.1 g/dL among PIH patients, with a further decline in HELLP syndrome cases (9.03±2.77 g/dL) compared to non-HELLP PIH patients (10.63±2.12 g/dL; $p=0.0464$). Dundy G et al.[17] reported marginal haemoglobin differences across groups—10.8 g/dL-mild preeclampsia, 11.1 g/dL-severe preeclampsia, and 11.5 g/dL-eclampsia, p>0.05. Chaware SA et al.[13] similarly recorded comparable haemoglobin values in mild (11 g/dL), severe (11 g/dL), and eclampsia (11.2 g/dL) groups. However, the downward trend in haemoglobin with advancing PIH severity underscores the possible contribution of

haemodilution, microangiopathic haemolysis, or nutritional deficiencies in PIH-related anaemia, warranting attention to haemoglobin monitoring as part of comprehensive antenatal care.

The peripheral smear analysis revealed normocytic normochromic anaemia in 44.1% and microcytic hypochromic anaemia in 39.1% of participants, with mild thrombocytopenia present in 1.82% and moderate thrombocytopenia in 1.36%, indicating underlying haematological alterations associated with PIH. Occasional schistocytes were seen in 0.9% patients. These findings are supported by Boddapati A et al,[18] who reported characteristic haemolytic changes such as schistocytes and anisopoikilocytosis in patients with HELLP syndrome—a severe variant of PIH—highlighting the diagnostic relevance of peripheral smear in such cases. Similarly, Shakarwal S et al.[7] observed deranged peripheral smears in 100% of HELLP patients, with associated thrombocytopenia, prolonged bleeding and clotting times, and elevated INR and PT, underscoring the utility of smear findings in detecting haematological complications in hypertensive pregnancies. This concordance reinforces the importance of peripheral smear examination as an adjunctive tool in identifying severe manifestations of PIH, including HELLP syndrome.

Conclusion

This study highlights the value of coagulation profile assessment in the early diagnosis and monitoring of hypertensive disorders in pregnancy. Changes in platelet count, PT, aPTT, and D-dimer levels correlate with disease severity and may serve as cost-effective markers for identifying complications like HELLP syndrome and DIC. These findings also support a pathophysiological link between endothelial dysfunction and coagulation abnormalities in PIH. Routine monitoring can guide timely interventions, and further large-scale studies are needed to define predictive cut-off values and improve outcomes.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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