Section: Pathology

Original Research Article

ALTERATIONS IN COAGULATION PARAMETERS IN PREGNANCY INDUCED HYPERTENSION AND THEIR CORRELATION WITH SEVERITY OF PREECLAMPSIA

Rajkumar R.[1], *J. Maheswari*[2], *A. Arputham*[3]

[1]*Assistant Professor, Department of Pathology, Institution Government Pudukkottai Medical College, India.*
[2]*Associate Professor, Department of Pathology, Government Pudukkottai Medical College, India.*
[3]*Associate Professor, Department of Pathology, Pudukkottai Mc, India.*

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Corresponding Author:
Dr Rajkumar R.,
Assistant Professor, Department of Pathology, Institution Government Pudukkottai Medical College, India.
Email: rajkumarabi79@gmail.com

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ABSTRACT

Background: Pregnancy induced hypertension (PIH) is a major cause of maternal and perinatal morbidity and mortality. It is associated with endothelial dysfunction and activation of the coagulation system, leading to alterations in hemostatic parameters. Early identification of these changes is crucial for timely management and prevention of complications. Aim to evaluate alterations in coagulation parameters in pregnancy induced hypertension and correlate them with the severity of preeclampsia.

Materials and Methods: This prospective case-control study was conducted on 200 pregnant women, including 100 normotensive controls and 100 women with PIH. Coagulation parameters including platelet count, bleeding time (BT), clotting time (CT), prothrombin time (PT), and activated partial thromboplastin time (APTT) were assessed. The PIH group was further categorized into mild preeclampsia, severe preeclampsia, and eclampsia. Statistical analysis was performed using appropriate tests, and p-value <0.05 was considered significant. Results: PIH patients showed a significant reduction in platelet count and prolongation of BT, CT, PT, and APTT compared to normotensive women ($p < 0.001$). The prevalence of coagulation abnormalities such as thrombocytopenia and prolonged coagulation times was significantly higher in PIH cases. A strong correlation was observed between worsening coagulation parameters and increasing severity of preeclampsia, with progressive deterioration from mild to severe cases and eclampsia ($p < 0.001$).

Conclusion: Coagulation parameters are significantly altered in PIH and correlate strongly with disease severity. Routine assessment of coagulation profile can aid in early diagnosis, risk stratification, and prevention of complications, thereby improving maternal and fetal outcomes.

Keywords: Pregnancy Induced Hypertension. Coagulation Parameters. Preeclampsia Severity.

INTRODUCTION

Pregnancy Induced Hypertension (PIH) is one of the most common and serious complications of pregnancy, contributing significantly to maternal and perinatal morbidity and mortality worldwide. In India, the incidence of PIH ranges from 5–15% of all pregnancies and accounts for a substantial proportion of maternal deaths, estimated to be around 16%, along with nearly 20% of perinatal mortality. PIH encompasses a spectrum of hypertensive disorders, including gestational hypertension, preeclampsia, and eclampsia, which typically develop after 20 weeks of gestation in previously normotensive women. Preeclampsia is defined by blood pressure ≥140/90 mmHg

associated with proteinuria ≥300 mg/day, and its severity is classified based on blood pressure levels and systemic involvement.[1]

Normal pregnancy is characterized by profound physiological adaptations, particularly within the hematological and coagulation systems. It is a hypercoagulable state, marked by increased levels of clotting factors, reduced fibrinolysis, and enhanced platelet activity, which serve as protective mechanisms against hemorrhage during delivery. However, in PIH, these physiological changes become exaggerated, leading to pathological activation of the coagulation cascade. Endothelial dysfunction, a hallmark of preeclampsia, results in imbalance between procoagulant and anticoagulant mechanisms, promoting platelet activation, thrombin generation, and fibrin deposition.[2]

Alterations in coagulation parameters such as platelet count, bleeding time (BT), clotting time (CT), prothrombin time (PT), and activated partial thromboplastin time (APTT) are frequently observed in PIH. Thrombocytopenia is the most common hematological abnormality, reflecting increased platelet consumption and destruction. Progressive derangement of coagulation parameters correlates with disease severity and may indicate impending complications such as disseminated intravascular coagulation (DIC) and HELLP syndrome (Hemolysis, Elevated Liver Enzymes, Low Platelet count), which are life-threatening conditions seen in severe preeclampsia and eclampsia.[3]

The pathophysiology of PIH involves abnormal placentation, leading to reduced uteroplacental perfusion and hypoxia. This triggers the release of antiangiogenic factors and inflammatory mediators, resulting in widespread endothelial injury and activation of the coagulation system. Consequently, the vascular endothelium shifts from an anticoagulant to a prothrombotic state, further exacerbating microvascular thrombosis and organ dysfunction.[4]

Aim

To evaluate alterations in coagulation parameters in pregnancy induced hypertension and correlate them with the severity of preeclampsia.

Objectives

1. To assess coagulation parameters (platelet count, BT, CT, PT, APTT) in pregnant women with PIH.
2. To compare coagulation parameters between normotensive pregnant women and those with PIH.
3. To correlate the degree of alteration in coagulation parameters with the severity of preeclampsia.

MATERIAL AND METHODS

Source of Data: The data were collected from pregnant women attending the Department of Obstetrics, and blood samples were analyzed in the Department of Pathology at a tertiary care teaching hospital. The study included both normotensive pregnant women and those diagnosed with pregnancy induced hypertension in late pregnancy (≥36 weeks).

Study Design

The study was conducted as a prospective case-control study, where pregnant women with PIH were considered as cases and normotensive pregnant women as controls.

Study Location

The study was carried out in the Departments of Obstetrics and Pathology of a tertiary care hospital.

Study Duration

The study was conducted over a period of 18 months.

Sample Size

A total of 200 pregnant women were included in the study:

- 100 normotensive pregnant women (control group)
- 100 pregnant women with pregnancy induced hypertension (study group)

Inclusion Criteria

- Pregnant women ≥36 weeks gestation
- Normotensive pregnant women (control group)
- Pregnant women diagnosed with PIH (BP ≥140/90 mmHg)
- Patients willing to give informed consent

Exclusion Criteria

- Pre-existing hypertension
- Diabetes mellitus
- Renal disease
- Known coagulation disorders
- Multifetal gestation
- Placenta previa or abruption
- Sepsis
- Heavy vaginal bleeding
- Age >35 years
- Smokers

Procedure and Methodology

A total of 200 pregnant women were enrolled after obtaining informed consent. Detailed history including obstetric and medical history was recorded. Clinical examination was performed with special emphasis on blood pressure measurement and detection of proteinuria.

Participants were divided into two groups: normotensive controls and PIH cases. The PIH group was further categorized into mild preeclampsia, severe preeclampsia, and eclampsia based on ACOG criteria.

Venous blood samples were collected under aseptic precautions. The following coagulation parameters were assessed:

- Platelet count using automated hematology analyzer (SYSMEX XP-100)
- Bleeding time by Duke's method
- Clotting time by Wright's capillary method
- Prothrombin time (PT) and APTT using automated coagulation analyzer (SYSMEX CA-50)

Sample Processing

Blood samples were collected in appropriate tubes:

- EDTA tubes for platelet count
- Citrated tubes for PT and APTT

Samples were processed within the recommended time frame. Plasma was separated by centrifugation for coagulation studies. All analyses were performed using standardized laboratory protocols with proper calibration and quality control measures.

Statistical Methods: Data were entered into Microsoft Excel and analyzed using statistical software (SPSS). Descriptive statistics: Mean ± SD, frequency, percentage Inferential statistics: Student's t-test for comparison of means. Chi-square test for categorical variables; Correlation analysis was performed to assess the relationship between coagulation parameters and severity of preeclampsia. A p-value <0.05 was considered statistically significant

Data Collection

Data were collected using a pre-designed and pre-tested structured proforma. Information included demographic details, clinical findings, laboratory parameters, and classification of PIH severity. All collected data were systematically recorded and validated for accuracy before analysis.

RESULTS

Table 1: Demographic and Clinical Characteristics of Study Population

Variable	Normotensive (n=100)	PIH (n=100)	Test Statistic	95% CI	P-value
Age (years) Mean ± SD	25.87 ± 3.41	26.92 ± 3.76	t = 2.03	0.03 to 2.06	0.044
Gestational age (weeks)	36.74 ± 0.81	36.51 ± 0.89	t = 1.92	-0.01 to 0.47	0.056
Primigravida n (%)	47 (47.0)	58 (58.0)	$\chi^2 = 2.42$		0.119
Systolic BP (mmHg)	118.62 ± 7.95	152.38 ± 12.47	t = 23.14	30.64 to 36.91	<0.001
Diastolic BP (mmHg)	76.31 ± 5.62	98.74 ± 8.21	t = 21.87	20.04 to 24.81	<0.001
Proteinuria ≥1+ n (%)	4 (4.0)	83 (83.0)	$\chi^2 = 112.36$		<0.001

Table 1 compares the demographic and clinical characteristics between normotensive pregnant women and those with pregnancy induced hypertension (PIH). The mean age was slightly higher in the PIH group (26.92 ± 3.76 years) compared to the normotensive group (25.87 ± 3.41 years), and this difference was statistically significant (t = 2.03, p = 0.044), indicating a mild association between increasing maternal age and PIH. The mean gestational age was comparable between the two groups (36.74 ± 0.81 vs 36.51 ± 0.89 weeks) with no statistically significant difference (p = 0.056), suggesting uniformity in gestational period at assessment. A higher proportion of primigravida women was observed in the PIH group (58.0%) compared to controls (47.0%), though this difference was not statistically significant (p = 0.119). As expected, both systolic and diastolic blood pressures were significantly elevated in the PIH group (152.38 ± 12.47 mmHg and 98.74 ± 8.21 mmHg) compared to normotensive women (118.62 ± 7.95 mmHg and 76.31 ± 5.62 mmHg), with highly significant p-values (<0.001). Proteinuria (≥1+) was markedly more prevalent in the PIH group (83.0%) than in controls (4.0%), and this difference was highly statistically significant (χ^2 = 112.36, p < 0.001).

Table 2: Assessment of Coagulation Parameters in PIH

Parameter	Normotensive (n=100) Mean ± SD	PIH (n=100) Mean ± SD	Test Statistic	95% CI	P-value
Platelet count (×10⁵/μL)	2.41 ± 0.56	1.68 ± 0.49	t = 9.86	0.58 to 0.87	<0.001
Bleeding Time (min)	2.83 ± 0.61	3.96 ± 0.74	t = 11.02	0.93 to 1.33	<0.001
Clotting Time (min)	5.12 ± 0.78	6.34 ± 0.92	t = 10.14	0.99 to 1.46	<0.001
Prothrombin Time (sec)	12.84 ± 0.89	14.72 ± 1.18	t = 12.49	1.58 to 2.17	<0.001
APTT (sec)	29.46 ± 2.17	33.81 ± 2.94	t = 12.06	3.64 to 5.06	<0.001

Table 2 demonstrates the comparison of coagulation parameters between normotensive and PIH groups. The mean platelet count was significantly reduced in the PIH group (1.68 ± 0.49 ×10⁵/μL) compared to the normotensive group (2.41 ± 0.56 ×10⁵/μL), indicating thrombocytopenia in hypertensive pregnancies (p < 0.001). Bleeding time was significantly prolonged in PIH patients (3.96 ± 0.74 minutes) compared to controls (2.83 ± 0.61 minutes), reflecting impaired platelet function. Similarly, clotting time was also prolonged in the PIH group (6.34 ± 0.92 minutes vs 5.12 ± 0.78 minutes), suggesting activation of coagulation pathways. Prothrombin time was significantly higher in PIH patients (14.72 ± 1.18 seconds) compared to normotensive women (12.84 ± 0.89 seconds), indicating involvement of the extrinsic pathway. Activated partial thromboplastin time (APTT) was also significantly prolonged in the PIH group (33.81 ± 2.94 seconds vs 29.46 ± 2.17 seconds), indicating intrinsic pathway alterations.

Table 3: Comparison of Coagulation Abnormalities Between Groups

Parameter Category	Normotensive n (%)	PIH n (%)	χ^2 Value	P-value
Thrombocytopenia (<1.5 lakh)	9 (9.0)	46 (46.0)	33.84	<0.001
Prolonged BT (>4 min)	6 (6.0)	39 (39.0)	29.17	<0.001
Prolonged CT (>6 min)	11 (11.0)	42 (42.0)	23.64	<0.001
Prolonged PT (>14 sec)	8 (8.0)	44 (44.0)	31.72	<0.001
Prolonged APTT (>35 sec)	7 (7.0)	37 (37.0)	26.18	<0.001

Table 3 presents the comparison of categorical coagulation abnormalities between normotensive and PIH groups. Thrombocytopenia (<1.5 lakh/μL) was significantly more common in the PIH group (46.0%) compared to the normotensive group (9.0%), indicating increased platelet consumption in PIH ($p < 0.001$). Prolonged bleeding time (>4 minutes) was observed in 39.0% of PIH patients compared to only 6.0% in controls, showing significant impairment in primary hemostasis. Similarly, prolonged clotting time (>6 minutes) was present in 42.0% of PIH cases compared to 11.0% of normotensive women. Prolongation of prothrombin time (>14 seconds) was seen in 44.0% of PIH patients versus 8.0% in controls, while prolonged APTT (>35 seconds) was noted in 37.0% of PIH cases compared to 7.0% of normotensive women. All these differences were highly statistically significant ($p < 0.001$), indicating that coagulation abnormalities are markedly more frequent in PIH patients.

Table 4: Correlation of Coagulation Parameters with Severity of Preeclampsia (PIH group n=100 subdivided)

Parameter	Mild (n=39) Mean ± SD	Severe (n=41) Mean ± SD	Eclampsia (n=20) Mean ± SD	F value	P-value
Platelet count ($\times 10^5$/μL)	1.92 ± 0.44	1.53 ± 0.39	1.21 ± 0.32	18.47	<0.001
Bleeding Time (min)	3.52 ± 0.62	4.11 ± 0.71	4.76 ± 0.84	15.93	<0.001
Clotting Time (min)	5.88 ± 0.71	6.51 ± 0.83	7.18 ± 0.92	17.26	<0.001
Prothrombin Time (sec)	14.08 ± 0.94	15.02 ± 1.11	16.21 ± 1.27	21.64	<0.001
APTT (sec)	32.41 ± 2.51	34.29 ± 2.83	36.72 ± 3.12	19.88	<0.001

Table 4 illustrates the correlation between coagulation parameters and severity of preeclampsia within the PIH group. A progressive decline in platelet count was observed with increasing severity, from mild (1.92 ± 0.44 $\times 10^5$/μL) to severe (1.53 ± 0.39 $\times 10^5$/μL) and eclampsia (1.21 ± 0.32 $\times 10^5$/μL), which was statistically highly significant ($p < 0.001$). Conversely, bleeding time showed a gradual increase with severity, rising from 3.52 ± 0.62 minutes in mild cases to 4.76 ± 0.84 minutes in eclampsia. Similarly, clotting time increased progressively across severity groups (5.88 ± 0.71 to 7.18 ± 0.92 minutes). Prothrombin time also showed a stepwise rise (14.08 ± 0.94 to 16.21 ± 1.27 seconds), indicating worsening coagulation dysfunction. APTT followed a similar pattern, increasing significantly from mild (32.41 ± 2.51 seconds) to eclampsia (36.72 ± 3.12 seconds).

DISCUSSION

The present study evaluated the alterations in coagulation parameters in pregnancy induced hypertension (PIH) and their correlation with the severity of preeclampsia.

Demographic and Clinical Characteristics (Table 1): In the present study, the mean age of women with PIH (26.92 ± 3.76 years) was slightly higher than that of normotensive women (25.87 ± 3.41 years), and this difference was statistically significant ($p = 0.044$). This finding is consistent with the study by Peng et al. (2024),[1] who reported that increasing maternal age is associated with a higher risk of hypertensive disorders due to vascular and metabolic changes. However, some studies like Bhutani et al. (2022),[2] observed no significant association between age and PIH, suggesting that age alone may not be a strong independent risk factor.

Gestational age was comparable between the two groups in this study ($p = 0.056$), indicating uniformity in the timing of evaluation. Similar findings were reported by Tadu et al. (2023),[3] where gestational age did not significantly differ between PIH and control groups. The proportion of primigravida was higher in the PIH group (58%), though not statistically significant, aligning with the observations of Rafaqat et al. (2023),[4] who reported that primigravidity is a known risk factor for preeclampsia due to immunological maladaptation.

As expected, systolic and diastolic blood pressures were significantly elevated in PIH cases ($p < 0.001$), which is consistent with diagnostic criteria and similar findings by Zeng et al. (2022).[5] Proteinuria was significantly higher in PIH patients (83%), confirming disease severity, in agreement with Alemu et al. (2025).[6]

Coagulation Parameters in PIH (Table 2): The present study demonstrated a significant reduction in platelet count in PIH patients ($p < 0.001$), which is consistent with studies by Jin et al. (2023),[7] who reported thrombocytopenia as the most common hematological abnormality in PIH. This decrease is attributed to increased platelet activation and consumption due to endothelial damage.

Bleeding time and clotting time were significantly prolonged in PIH cases, reflecting impaired primary hemostasis and ongoing coagulation activation. Similar findings were observed by Kou et al. (2025),[8] who reported prolonged bleeding parameters in severe preeclampsia due to platelet dysfunction.

Prothrombin time (PT) and activated partial thromboplastin time (APTT) were also significantly prolonged in PIH patients ($p < 0.001$), indicating involvement of both extrinsic and intrinsic coagulation pathways. These findings are in agreement with Tadu et al. (2023),[3] who demonstrated that endothelial injury in PIH leads to activation of the coagulation cascade and consumption of clotting factors, resulting in prolonged PT and APTT.

Comparison of Coagulation Abnormalities (Table 3): Categorical analysis showed that thrombocytopenia, prolonged BT, CT, PT, and APTT were significantly more frequent in PIH patients compared to normotensive women ($p < 0.001$). Nearly half of PIH patients (46%) had thrombocytopenia, which is consistent with the findings of Jin et al. (2023),[7] who reported a high prevalence of thrombocytopenia in hypertensive pregnancies.

The increased proportion of prolonged PT and APTT in PIH patients reflects the progression towards consumptive coagulopathy and possible development of disseminated intravascular coagulation (DIC). Similar observations were reported by Gopinath et al. (2025),[9] who found that coagulation abnormalities worsen with disease severity and may indicate impending complications such as HELLP syndrome.

Correlation with Severity of Preeclampsia (Table 4): The present study demonstrated a strong correlation between worsening coagulation parameters and increasing severity of preeclampsia. Platelet count showed a progressive decline from mild to eclampsia cases, while BT, CT, PT, and APTT showed a stepwise increase, all statistically significant ($p < 0.001$). These findings are consistent with Kou et al. (2025),[8] who reported that platelet count and coagulation parameters can serve as markers of disease severity.

The progressive derangement of coagulation parameters reflects increasing endothelial dysfunction, platelet consumption, and activation of the coagulation cascade in severe disease. Similar trends were reported by Zhang et al. (2023),[10] who emphasized that severe preeclampsia and eclampsia are associated with higher risk of coagulopathy and adverse outcomes.

CONCLUSION

The present study demonstrates that pregnancy induced hypertension (PIH) is significantly associated with marked alterations in coagulation parameters. Compared to normotensive pregnant women, PIH patients showed a significant reduction in platelet count along with prolongation of bleeding time, clotting time, prothrombin time, and activated partial thromboplastin time, indicating activation and consumption of the coagulation cascade. These changes reflect the underlying endothelial dysfunction and hypercoagulable state characteristic of preeclampsia.

Furthermore, a strong correlation was observed between the severity of preeclampsia and the degree of derangement in coagulation parameters. As the disease progressed from mild preeclampsia to severe preeclampsia and eclampsia, there was a progressive decline in platelet count and a significant increase in coagulation times. This highlights the role of coagulation parameters as potential markers for assessing disease severity and predicting complications.

Thus, routine evaluation of coagulation profile in pregnant women with PIH is essential for early detection of abnormalities, timely intervention, and prevention of life-threatening complications such as disseminated intravascular coagulation and HELLP syndrome. Monitoring these parameters can significantly improve maternal and fetal outcomes.

limitations of the study

- The study was conducted in a single tertiary care center, which may limit the generalizability of the findings.
- The sample size, although adequate, may not represent the entire population diversity.
- Only selected coagulation parameters (platelet count, BT, CT, PT, APTT) were evaluated; advanced markers like D-dimer, fibrinogen levels, and antithrombin III were not included.
- The cross-sectional nature of assessment limits the ability to establish temporal causality.
- Follow-up of patients for maternal and fetal outcomes was not included.
- Possible confounding factors such as nutritional status and subclinical infections were not fully assessed.
- Inter-observer variability in manual methods like bleeding time and clotting time may have influenced results.

REFERENCES

1. Peng J, Zhao Q, Pang W, Li Y, Dong X. Changes of coagulation function and platelet parameters in preeclampsia and their correlation with pregnancy outcomes. The Journal of Clinical Hypertension. 2024 Oct;26(10):1181-7.
2. Bhutani N, Jethani V, Jethani S, Ratwani K. Coagulation profile and platelet parameters in pregnancy induced hypertension cases and normotensive pregnancies: a cross-sectional study. Annals of Medicine and Surgery. 2022 Aug 1;80:104124.
3. Tadu S, Yerroju K, Gudey S. A comparative study of coagulation profile in normal pregnancy, mild

preeclampsia, and severe preeclampsia patients. Journal of South Asian Federation of Obstetrics and Gynaecology. 2023 Apr 19;15(1):71-5.
4. Rafaqat S, Khalid A, Riaz S, Rafaqat S. Irregularities of coagulation in hypertension. Current hypertension reports. 2023 Oct;25(10):271-86.
5. Zeng L, Liao C. Multivariate logistic regression analysis of preeclampsia in patients with pregnancy induced hypertension and the risk predictive value of monitoring platelet, coagulation function and thyroid hormone in pregnant women. American journal of translational research. 2022 Sep 15;14(9):6805.
6. Alemu N, Teketelew BB, Admas S, Marelgn L, Eyayu Y, Woldu B. Coagulation profiles and platelet parameters among preeclampsia, eclampsia, and normotensive pregnant women attending Comprehensive Specialized Hospital maternity wards, Northwest Ethiopia. PLoS One. 2025 Jul 21;20(7):e0328578.
7. Jin PP, Ding N, Dai J, Liu XY, Mao PM. Investigation of the relationship between changes in maternal coagulation profile in the first trimester and the risk of developing preeclampsia. Heliyon. 2023 Jul 1;9(7).
8. Kou X, Li Q, Pan Z, Yang J. Coagulation indices for predicting hypertensive pregnancy disorders. American Journal of Translational Research. 2025 Feb 15;17(2):1106.
9. Gopinath D, Varghese RG, Raveendran C. Hematological and coagulation parameters in pregnancy-induced hypertension: A case–control study. Indian Journal of Obstetrics and Gynecology Research. 2025;12(4):706-12.
10. Zhang Y, Li H, Guo W, Zhao H, Zheng N, Huang Y. Predictive value of coagulation function and D-dimer for pregnancy outcome in pregnancy-induced hypertension. American Journal of Translational Research. 2023 Feb 15;15(2):1150.