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Original Research Article

Coagulation profile and Platelet indices as a predictive marker in Gestational Hypertension and Pre-eclampsia- A case-control study of 260 cases

Sweta[1], Mayank Agrawal[1], Mukta Saini[1]*, Rajeev Sen[2]

[1]Dept. of Pathology, Shri Atal Bihari Vajpayee Government Medical College, Chhainsa, Faridabad, Haryana, India
[2]Dept. of Pathology, SGT Medical College, Hospital and Research Institute, Gurugram, Haryana, India

Abstract

Introduction: Pregnancy-induced hypertension (PIH) is the leading medical condition during pregnancy that can result in multiple organ systems failing in the mother. PIH is recognized worldwide as a major cause of illness and death in both mothers and newborns. This research aimed to compare platelet levels and blood clotting factors (PT, APTT) between pregnant women with high blood pressure and those with normal blood pressure. The study also sought to determine the severity of PIH.

Materials and Methods: A comparative study was conducted over a one-year period to analyze platelet parameters and coagulation profiles (Prothrombin Time, Activated Partial Thromboplastin Time) in overall 260 pregnant women with 130 PIH cases and 130 normotensive pregnancies treated as control group.

Results: In PIH (includes Gestational Hypertension, Mild Pre-Eclampsia, Severe Pre-Eclampsia), A statistically significant decrease in platelet count was observed compared to the control group. Additionally, both Prothrombin Time (PT) and Activated Partial Thromboplastin Time (APTT) were prolonged, and these differences were also statistically significant.

Conclusion: The severity of PIH is negatively correlated with platelet count, and as thrombocytopenia worsens, the risk of coagulopathy rises. Both the APTT and PT were extended. A standard screening test for coagulopathy in PIH that is quick, easy, and inexpensive is the platelet count.

Keywords: PIH (Pregnancy Induced Hypertension), Platelet Count, PT (Prothrombin time)

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1. Introduction

Pregnancy Induced Hypertension (PIH) is a condition distinguished by the development of elevated blood pressure occurring after the 20-week mark of pregnancy. It might cause mother and new born problems if not addressed effectively. High blood pressure, swelling, headaches, and visual abnormalities are symptoms. PIH can progress to preeclampsia, which involves additional symptoms like protein in urine and can affect organ function.

PIH constitutes a major contributor to maternal and perinatal morbidity and mortality.[1] Data from the National Health Portal India (NHP) indicates that pregnancy-induced hypertensive disorders contribute to approximately 10% of all maternal deaths in Asia, whereas PIH affects approximately 8-10% of women in India.[2] Preeclampsia is more common in India because of impoverishment, poor health, and lack of antenatal care. Hypertension (HTN) is the second most prevalent direct cause of maternal mortality worldwide, especially in developing nations, according to the WHO systematic analysis of "Global Causes of Maternal Deaths" article published in The Lancet in 2014.[2]

Preeclampsia significantly increases the risk of thrombocytopenia, particularly in severe cases and among women with pre-existing vascular conditions. Low platelet counts in pregnant women are associated with more severe illness and potential complications like disseminated intravascular coagulation.

Preeclampsia poses a significant public health concern, especially in developing nations.[3-6] Particularly during and after surgeries like cesarean sections or epidural anaesthesia, coagulation abnormalities related to PIH increase the risk of bleeding.

*Corresponding author: Mukta Saini
Email: muktasaini13@gmail.com

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In India, hypertensive disorders of pregnancy, including preeclampsia affecting 5.4% of pregnancies, occur in 7.8% of cases. Eclampsia and HELLP syndrome (hemolysis, increased liver enzymes, and low platelet count) are severe side effects of preeclampsia. Pre-eclampsia can be discovered and treated early, before convulsions (eclampsia) and other potentially fatal consequences develop.[7]

Hypercoagulability is consistently connected with hypertensive diseases during pregnancy, particularly pre-eclampsia. Thus, standard coagulation testing (Prothrombin time (PT) and APTT) is the most often utilized test to check for indications of DIC and HELLP syndrome-2, as it measures enzyme activity that causes clot formation.

Because platelet count is a simple, affordable test to determine the severity of PIH and its complications, this study compared the coagulation profile and platelet indices as predictive markers in gestational HTN, mild pre-eclampsia, and severe pre-eclampsia with normal pregnancies.

2. Materials and Methods

After receiving approval from the "Institutional Ethics Committee", a one-year prospective comparison study involving 260 cases—130 of which were PIH patients and 130 of which were normotensive pregnant women—had been performed in the "Department of Pathology, FMHS, SGT Hospital, Gurugram". 2ml of blood sample in EDTA vial and 1.8 ml blood sample in sodium citrate vial was taken and required investigations were done within 30mins of blood collection. Patient data were extracted from medical records using a standardized form. Maternal symptoms upon presentation, obstetrical history, patient demographics, and maternal and perinatal outcomes were all included in this form. After being gathered, the data was examined. Following a clinical examination and investigations, patients had been classified into four groups based on the American College of Obstetricians and Gynecologists' Williams Obstetrics Textbook 25th Edition 2018 table 40-1 classification: normotensive pregnancies, gestational hypertension, mild preeclampsia, and severe preeclampsia. This study exclusively included pregnant women beyond 20 weeks of gestation, encompassing both those with pregnancy-induced hypertension and those with normal blood pressure. Patients with pre-existing/chronic hypertension, eclampsia, known bleeding disorders, anticoagulant medication, labour, established DIC, or pre-existing renal disease are excluded from our study groups.

2.1. Statistical analysis

SPSS software (v.23.0) was employed to perform statistical analyses. Platelet count, PT, and APTT correlations between PIH and normotensive pregnant women were assessed by employing the chi-square test; a threshold <0.05 was considered statistically significant.

3. Results

The study comprised 260 pregnant women in total, 130 of whom were PIH patients as cases and 130 of whom were normotensive pregnant women as controls. In our study on the basis of gravida, the maximum number of patients of primigravida (23.8%) followed by second gravida (17.31%) and third gravida (7.69%) are of gestational hypertension whereas the maximum number of patients of multigravida (7.69%) are of pre-eclampsia. The gravida status of the patients with "pre-eclampsia", severe "pre-eclampsia", and gestational hypertension did not differ statistically significantly. As of now, gestational hypertension is a most common presentation in a pregnant woman, and out of 130 patients of PIH, maximum number of cases were of gestational hypertension.

Table 1 illustrates a comparison of platelet indices between normal pregnancy, gestational hypertension and mild pre-eclampsia, and severe pre-eclamsia. Analysis of platelet parameters revealed significant differences across the groups. With mild "pre-eclampsia" at 1.81 lakhs/dl, severe "pre-eclampsia" at 1.66 lakhs/dl, and gestational hypertension at 2.0 lakhs/dl, the mean platelet counts gradually dropped (p-value = 0.0012). Similarly, mean platelet volume (MPV) demonstrated a significant increase from 8.6 fl in gestational hypertension to 9.7 fl in mild pre-eclampsia and further to 10.3 fl in severe pre-eclampsia (p-value<0.05). Although there were statistically significant variations in platelet distribution width (PDW) between the groups (p-value < 0.05), these changes were not as large as those in platelet count and MPV.

In the gestational hypertension group (n=70), the most frequent platelet count range was 1-2.5 lakhs/μL in 82.86% of cases. Increased mean platelet volume (MPV) was observed in 28.57% of cases (10-12 fl), and increased platelet distribution width (PDW) was found in 35.71% of cases (12-14 fl). In the mild pre-eclampsia group (n=45), 82.22% of cases exhibited a platelet count within the 1-2.5 lakhs/μL range. Increased MPV was observed in 44.44% of cases, and increased PDW was found in 33.33% of cases. Out of total of 15 patients with severe pre-eclampsia, 12(80%) cases have a platelet count of 1-2.5 lakhs whereas 8(50%) cases have increased mean platelet volume, and 5(33.33%) cases have increased platelet distribution width. The platelet count is a simple, rapid, and inexpensive regular screening test for coagulopathy in PIH, and it is negatively correlated with the severity of PIH.

Table 1: Comparison of platelet indices between normal pregnancy, gestational hypertension, mild pre-eclampsia, and severe pre-eclampsia.

Category	Platelet Count		Mean Platelet Volume (MPV)		Platelet Distribution Width (PDW)	
	Mean ± SD	P value	Mean ± SD	P value	Mean ± SD	P value
Normal Pregnancy (n=130)	2.85 ± 0.58	0.0012*	7.47 ± 1.93	0.04*	10.90 ± 1.32	0.0032*
Gestational Hypertension (n=70)	2.01 ± 0.50		8.65 ± 0.97		11.33 ± 1.11	
Mild Preeclampsia (n=45)	1.81 ± 0.48		9.79 ± 0.68		11.96 ± 0.77	
Severe Preeclampsia (n=15)	1.66 ± 0.47		10.33 ± 1.07		11.70 ± 0.14	

*Statistically significant

Table 2: Comparison of coagulation profile between normal pregnancy, gestational hypertension, mild pre-eclampsia, and severe pre-eclampsia.

Category	Prothrombin Time		Activated Partial Thromboplastin Time	
	Mean ± SD	P value	Mean ± SD	P value
Normal Pregnancy (n=130)	12.95 ± 1.48	0.0021*	25.76 ± 2.99	0.001*
Gestational Hypertension (n=70)	14.59 ± 1.44		33.55 ± 2.44	
Mild Preeclampsia (n=45)	17.61 ± 2.88		38.79 ± 2.52	
Severe Preeclampsia (n=15)	18.88 ± 0.00		41.85 ± 0.00	

*statistically significant

Table 2 illustrates the comparison of coagulation profiles between normal pregnancy, gestational HTN, mild pre-eclampsia, and severe "pre-eclampsia". Based on coagulation profile (PT, APTT), the mean PT and APTT of study groups with gestational hypertension was 14.59 secs, 33.5 secs, for mild pre-eclampsia cases 17.6 secs, 38.7 secs, and for severe pre-eclampsia cases was 18.8 secs, 41.8 secs. With 100% of cases showing extended PT and APTT, the severe pre-eclampsia group had the highest incidence of both conditions. 82.22% of cases in the mild pre-eclampsia group had extended PT, while 66.67% had prolonged APTT. 50% of cases in the gestational hypertension group had prolonged APTT, while 57.14% of cases had prolonged PT. The study groups with mild "pre-eclampsia", severe "pre-eclampsia", and prenatal hypertension all showed statistically significant changes in PT and APTT (p-values of 0.0021 and 0.001, respectively).

4. Discussion

One of the main causes of poor maternal and perinatal outcomes is hypertensive disorders. Other haematological changes may occur as well, however, thrombocytopenia is the most frequent haematological abnormality seen in pregnancies associated with hypertension.

Based on gravida status, maximum pregnant women were found to have a higher prevalence of gestational hypertension (about 23%), and about 17% of cases of gestational hypertension were gravida 2, and 4 (7.69%) cases were multigravida. Half of the cases of preeclampsia were primigravida and half of the patients were in gravida 2 indicating an increased incidence of severe preeclampsia in early gestation age.

In our study, we observed a mean platelet count in normotensive pregnant women at term was 2.85±0.58lakhs/cumm. In contrast, the mean platelet count progressively decreased in the study groups with gestational hypertension (2.01±0.50lakhs/cumm), mild preeclampsia (1.81±0.48lakhs/cumm), and severe preeclampsia (1.66 ± 0.47 lakhs/cumm). This suggests that mean platelet counts decrease as the grades of pregnancy-induced hypertension cases increase. Between the study groups with moderate preeclampsia, severe preeclampsia, and gestational hypertension, there was a significant drop in platelets (p-0.0012). As per 2013 research by Gupta et al.,[8] the mean platelet count in a healthy pregnancy was 3.41 lakhs/cumm, while in cases of severe PIH, it was 1.27 lakhs/cumm, with a significant P-value <0.05.

Previous research by Abass et al.[9] reported a significant decline in platelet count across different severities of pregnancy-induced hypertension (PIH). Platelet counts were found to be 2.44 ± 0.11 lakhs/cumm in normal pregnancies, 1.82 ± 0.19 lakhs/cumm in non-severe PIH, and 1.42 ± 0.23 lakhs/cumm in severe PIH (p-value < 0.05). Our study observed a similar trend in Mean Platelet Volume (MPV). In pregnant women with normotension, the mean MPV was 7.47 ±1.93 fl (range: 8–12); in those with gestational hypertension, it increased substantially to 8.65 ± 0.97, in moderate preeclampsia, it was 9.79 ± 0.68, and in severe preeclampsia, it was 10.33 ± 1.07 (p=0.04). This steady increase in MPV with increasing PIH severities is consistent with earlier research findings.

The study conducted by Reddy et al.,[10] and Temur et al.[11] observed that MPV was significantly high in the pre-eclampsia group with a p-value <0.001. Numerous

investigations with varying degrees of success have confirmed PDW as a reliable indicator of platelet dysfunction in PIH cases. The control group in our research had a mean Platelet Distribution Width (PDW) of 10.90 ± 1.32 fl. With values of 11.33 ± 1.11 fl in prenatal hypertension, 11.96±0.77fl in mild preeclampsia, and 11.7±0.14fl in severe eclampsia (p-value = 0.0032), there was a notable rise in PDW in the PIH group as the PIH grades improved. When our study was compared to that of Reddy et al.,[10] Temur et al.[11] found that the pre-eclampsia group had a considerably greater PDW (p-value <0.001).

In our study, Prothrombin Time (PT) progressively increased with the severity of pregnancy-induced hypertension (PIH). Mean PT was 12.95 ± 1.48 seconds in normotensive pregnancies, 14.59 ± 1.44 seconds in gestational hypertension, 17.61 ± 2.88 seconds in mild preeclampsia, and 18.88 ± 0.0 seconds in severe preeclampsia. 57.14% of instances of prenatal hypertension, 82.22% of cases of mild preeclampsia, and all cases of severe preeclampsia showed a substantial extension of PT (p-value = 0.0021). These results are consistent with earlier studies. According to others study the mean PT for severe PIH (9.8 seconds) and non-severe PIH (10.1 seconds) was significantly lower than that of normal pregnancies (10.9 seconds) (p-value < 0.0001).[12] In a similar vein, Walker et al.[13] discovered that women with severe preeclampsia and eclampsia had significantly higher mean PT values (p < 0.05).

In the current study, a significant increase in Activated Partial Thromboplastin Time (aPTT) was observed with increasing severity of pregnancy-induced hypertension (PIH).[1] Mean aPTT was 41.85 ± 0.00 seconds in normotensive pregnancies, 38.79± 2.52 seconds in gestational hypertension, 33.55 ± 2.44 seconds in mild preeclampsia, and 25.76 ± 2.99 seconds in severe preeclampsia (p-value = 0.001). A study done by Weinstein et al.[14] observed that the "Activated partial thromboplastin" time was found to be 38.7 sec in severe preeclampsia and in normal pregnant women it was found to be 34.24 secs. The mean APTT was 25.11 seconds for patients with mild GH, 25.97 seconds for those with severe GH, 26.74 seconds for those with mild preeclampsia, and 27.33 seconds for those with severe preeclampsia, per a study by Suzuki et al.[15]

5. Conclusion

Pregnancy-induced hypertension and normal pregnancies are both predicted by the coagulation profile and platelet indices. The risk of coagulopathy increases as thrombocytopenia increases, and the platelet count is inversely correlated with the severity of PIH. The mean values of MPV and PDW also increased with increasing grade and were significant. PT and APTT both are prolonged in cases of PIH. In order to reduce maternal and fetal mortality and morbidity, this study should be conducted based on abnormalities in predictive markers (platelet indices and coagulation profile) related to gestational hypertension, pre-eclampsia at an earlier stage, and the potential for management of complications arising in relation to pregnancy-induced hypertension.

6. Source of Funding

None.

7. Conflict of Interest

None.

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