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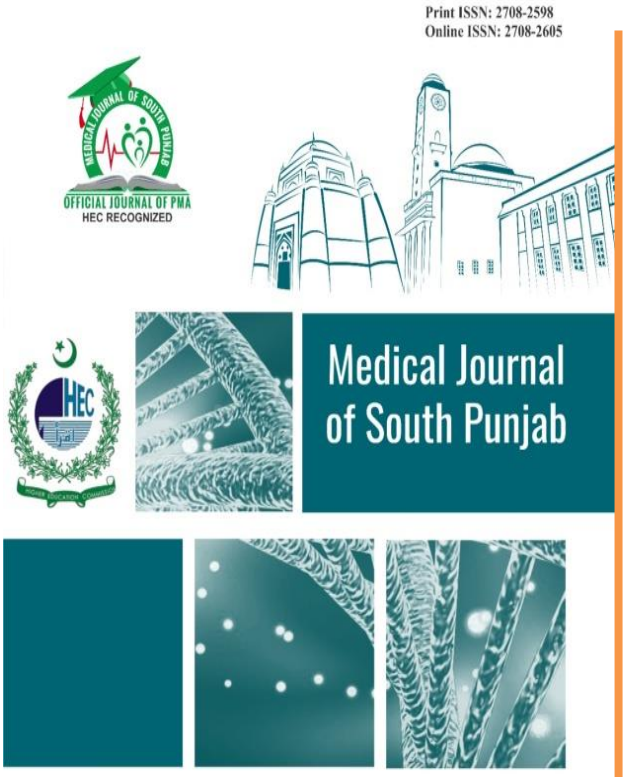

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Original Article

Platelet Count and Its Prognostic Value in Pregnancy-Induced Hypertension

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ABSTRACT

Objective: To determine the pattern of decline in platelet count across the spectrum of pregnancy-induced hypertension in preeclampsia and eclampsia.

Methods: This prospective observational study was conducted at the Obstetrics and Gynecology Department of CMH Hospital, Multan, Pakistan, from 3rd April 2024 to 4th October 2024. A total of 200 pregnant women beyond 28 weeks of gestation were enrolled, comprising 100 women diagnosed with PIH (study group) and 100 normotensive women (control group). Platelet counts were monitored at four-week intervals for the control group and weekly for the study group until delivery. Statistical analyses, including Chi-square tests, independent t-tests, and one-way ANOVA, were utilized to assess the correlation between platelet dynamics and PIH severity.

Results: The mean platelet counts demonstrated a significant progressive decline correlating with disease severity: $2.09 \pm 0.43 \times 10^5/mm^3$ in mild preeclampsia, $1.58 \pm 0.42 \times 10^5/mm^3$ in severe preeclampsia, and $0.96 \pm 0.38 \times 10^5/mm^3$ in eclampsia. The overall mean platelet count for the PIH group ($1.74 \pm 0.56 \times 10^5/mm^3$) was significantly lower than that of the normotensive controls ($2.28 \pm 0.46 \times 10^5/mm^3$, $p < 0.001$). Severe thrombocytopenia ($<1 \times 10^5/mm^3$) was predominantly observed in eclampsia cases (33.3%). Adverse maternal and fetal outcomes were disproportionately clustered among patients with platelet counts $\leq 1.5 \times 10^5/mm^3$.

Conclusion: Platelet count serves as a highly effective, economical, and accessible biomarker for predicting the progression of PIH and anticipating adverse maternal and fetal outcomes. Routine antenatal platelet monitoring should be integrated into the standard management protocols for hypertensive pregnancies to facilitate early intervention and mitigate severe complications.

Keywords: Eclampsia, Platelet count, Preeclampsia, Prognostic marker, Thrombocytopenia

1. INTRODUCTION

Pregnancy induces a physiological state of controlled systemic inflammation, accompanied by immunological adaptations essential for fetal tolerance and maternal homeostasis.[1,2] In preeclampsia, this regulated inflammatory response becomes pathological, leading to systemic endothelial dysfunction and multiorgan injury.[3] Hypertensive disorders of pregnancy, primarily preeclampsia and eclampsia, complicate 5–10% of pregnancies globally and remain a leading contributor to maternal and perinatal morbidity and mortality.[4,5] These conditions are characterized by widespread vascular damage affecting maternal organs including the liver, kidneys, brain, and placenta and are frequently associated with hematological disturbances.[6]

Hemostatic adaptations occur in both normotensive and hypertensive pregnancies. While platelet counts in uncomplicated pregnancies typically remain within standard reference ranges, a gradual decline is often observed during the third trimester.[7] This physiological reduction is attributed to hemodilution, accelerated platelet consumption secondary to endothelial activation, and enhanced platelet aggregation driven by elevated thromboxane A2 (TXA2) levels.[8] Severe thrombocytopenia (platelet count <50,000/mm³) is rare in normal pregnancies (0.1%), but its prevalence rises significantly in pregnancy-induced hypertension (PIH).[9] The severity of thrombocytopenia in PIH correlates directly with the intensity and duration of the disease process, reflecting the extent of systemic endothelial injury and microangiopathic hemolysis.[10]

Thrombocytopenia serves as a critical clinical marker for disease progression in PIH. Quantitative platelet assessment provides robust prognostic information regarding adverse pregnancy outcomes, including HELLP syndrome, placental abruption, disseminated intravascular coagulation (DIC), intrauterine growth restriction (IUGR), and preterm birth.[11] This study aims to evaluate whether serial platelet monitoring can effectively determine the severity of preeclampsia and eclampsia and predict clinical outcomes, thereby offering a resource-efficient management strategy, particularly in low-resource healthcare settings.

2. METHODOLOGY

This prospective observational study was conducted at the Obstetrics and Gynecology Department of CMH Hospital, Multan, Pakistan, 3rd April 2024 to 4th October 2024. The study enrolled 200 pregnant women: 100 women diagnosed with PIH (study group) and 100 normotensive pregnant women (control group). Participants were selected via simple random sampling (every tenth antenatal outpatient) to minimize selection bias. Clinical criteria were used to categorize the study group into mild preeclampsia, severe preeclampsia, and eclampsia.

Patients with singleton pregnancy ≥28 weeks gestation, diagnosis of PIH (study group) or absence of hypertensive disorders (control group).

Patients with history of thromboembolic events or hemorrhagic disorders, pre-existing epilepsy, hepatic/renal dysfunction, or diabetes mellitus, chronic hypertension diagnosed before 20 weeks gestation, use of medications affecting platelet count or function were excluded.

All participants underwent standardized clinical examinations, including medical history, physical assessment, and routine antenatal investigations (blood group, hemoglobin, bleeding/clotting time, FAST, HIV, VDRL, HBsAg).

Venous blood (5 mL) was drawn from the antecubital fossa into EDTA-coated tubes. Platelet counts were performed manually using Leishman's stain. Under oil immersion microscopy (1000× magnification), platelets appeared as pale pink cytoplasmic fragments

(1–4 µm diameter). Ten consecutive fields were examined, and the total count per field was multiplied by 20,000 to determine the platelet count per cubic millimeter of blood.

Control group: Platelet counts assessed every 4 weeks from 28 weeks until delivery. Study group: Platelet counts monitored weekly from 28 weeks, with clinical management tailored to disease severity: Mild preeclampsia was Managed via outpatient visits and severe preeclampsia/eclampsia hospitalized for close surveillance until delivery.

Participants were monitored from enrollment until 6 weeks postpartum. Maternal outcomes include mode of labor onset and delivery (vaginal, cesarean), ante partum complications (imminent eclampsia, placental abruption, HELLP syndrome, preterm labor), and postpartum complications (PPH, pulmonary edema, DIC, maternal mortality).
Fetal outcomes were IUGR, appropriate for gestational age (AGA), meconium aspiration syndrome (MAS), intrauterine death (IUD), and neonatal complications (jaundice, respiratory distress syndrome [RDS], and septicemia).

Descriptive statistics were used to present demographic and clinical data. Categorical variables were expressed as percentages, while continuous variables were presented as means ± standard deviations (SD). Inferential statistics included Chi-square tests for categorical comparisons, independent sample t-tests to compare means between study and control groups, and one-way ANOVA to analyze trends in platelet counts across PIH severity levels, followed by post-hoc Tukey tests. Logistic regression was used to estimate the impact of thrombocytopenia on adverse events. A two-tailed p-value <0.05 was considered statistically significant. All analyses were performed using SPSS v23.0 (IBM Corp).

3. RESULTS

Among the 100 women with PIH, platelet counts exhibited a significant progressive decline as disease severity escalated (Table 1). In the mild preeclampsia group (n=47), 70.2% of patients maintained platelet levels above 2 $\times 10^5/mm^3$. Conversely, severe preeclampsia cases (n=38) predominantly registered counts between 1–1.5 $\times 10^5/mm^3$ (39.5%), while 60.0% of eclampsia patients (n=15) fell into this lower range. Severe thrombocytopenia (<1 $\times 10^5/mm^3$) was observed in 7% of the total PIH cohort but was most prevalent in the eclampsia subgroup (33.3%). In the normotensive control group, 65% of participants had platelet counts above 2 $\times 10^5/mm^3$, and none exhibited severe thrombocytopenia (Table 2).

Table 1: Platelet count distribution in mild preeclampsia, severe preeclampsia, and eclampsia

PLT count ($\times 10^5/mm^3$)	Mild preeclampsia n (%)	Severe preeclampsia n (%)	Eclampsia n (%)
< 1	0 (0%)	2 (5.3%)	5 (33.3%)
1–1.5	4 (8.5%)	15 (39.5%)	9 (60.0%)
1.5–2	10 (21.3%)	13 (34.2%)	1 (6.7%)
> 2	33 (70.2%)	8 (21.1%)	0 (0%)
Total	47 (100%)	38 (100%)	15 (100%)

Table 2: Platelet count distribution in controls

Platelet count ($\times 10^5/mm^3$)	Controls (n)	Percentage (%)
< 1	0	0%
1–1.5	0	0%
1.5–2	35	35%
> 2	65	65%
Total	100	100%

Maternal complications occurred in 56% of PIH cases, with preterm labor (32%) and imminent eclampsia (24%) being the most

frequent. Placental abruption and DIC were exclusively observed in severe preeclampsia and eclampsia. Fetal complications affected 61% of cases, predominantly IUGR (20%), IUD (11%), and low birth weight (LBW) (10%). (Tables 3 & 4)

Table 3: Maternal complications across PIH categories

Group	Preterm Labour	Abruption	DIC	PPH	Maternal Death
Mild Preeclampsi	6	0	0	0	0
Severe Preeclampsia	15	4	0	1	0
Eclampsia	11	1	2	0	1

Table 4: Fetal complications across PIH categories

Group	IUGR	IUD	RDS	LBW
Mild Preeclampsia	2	0	0	1
Severe Preeclampsia	17	4	5	7
Eclampsia	1	7	2	2
Total	20	11	7	10

4. DISCUSSION

Thrombocytopenia represents the most characteristic hematological abnormality in pregnancy-induced hypertension (PIH), with its incidence and severity increasing as the disease progresses. This study corroborates existing evidence that thrombocytopenia is highly prevalent in PIH, particularly in severe preeclampsia and eclampsia, where endothelial dysfunction and microangiopathic processes drive accelerated platelet consumption.[10,11] The observed progressive decline in mean platelet counts from 2.09 $\times 10^5/mm^3$ in mild preeclampsia to 0.96 $\times 10^5/mm^3$ in eclampsia aligns with the pathophysiological continuum of PIH, wherein systemic inflammation and placental ischemia exacerbate platelet aggregation and turnover.[12]

Consistent with prior studies, nulliparity emerged as a notable demographic factor, with 58% of PIH cases occurring in nulliparous women.[13,14] This parallels findings by Kumar et al. (61%) and Sajith et al. (53.8%), suggesting that first pregnancies may lack the immunotolerance mechanisms acquired in subsequent gestations.[15,16] However, in this cohort, all 100 normotensive controls were also nulliparous, which warrants caution in interpreting parity as an independent risk factor in this specific population.

The mean maternal age in the PIH group was 26.7 ± 4.5 years, reflecting a trend toward delayed childbearing in urban settings, contrasting with rural norms where first-time mothers are typically younger.[17] This urban demographic shift necessitates area-specific risk assessment protocols.

The inverse relationship between platelet count and PIH severity observed in this study is consistent with research by Jambhulkar et al and Poluri et al, who reported severe thrombocytopenia in eclampsia patients.[18,19] In the present study, 33.3% of eclampsia patients presented with platelet counts below 1 $\times 10^5/mm^3$. The lowest recorded platelet level (0.51 $\times 10^5/mm^3$) was observed in an eclampsia patient who developed pulmonary edema and succumbed, a finding consistent with the established correlation between profound thrombocytopenia, DIC, and multi-organ failure.[20]

5. CONCLUSION

Platelet count serves as a highly effective, economical, and accessible biomarker for predicting the progression of PIH and anticipating adverse maternal and fetal outcomes. Routine antenatal platelet monitoring should be integrated into the standard management protocols for hypertensive pregnancies to facilitate early intervention and mitigate severe complications.

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