

Frequency of Low Platelet Count in Third Trimester among Patients with Pre-Eclampsia

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Abstract

Objective: To determine the frequency of low platelet count among pregnant women with pre-eclampsia during the third trimester (29–41 weeks of gestation).

Methodology: This cross-sectional study was conducted at the Department of Gynecology and Obstetrics, Sheikh Zayed Hospital, Rahim Yar Khan, Pakistan, from February 2025 to July 2025. Women aged 18–45 years with pre-eclampsia and a singleton pregnancy in the third trimester were enrolled using non-probability consecutive sampling. Demographic and clinical data, including the severity of pre-eclampsia, were recorded. Platelet count was measured as part of a complete blood count using a 3-mL venous blood sample. Low platelet count was defined as $<150 \times 10^9/L$. Data were analyzed using SPSS version 26.0.

Results: Among the 167 women included in the study, the mean age was 29.8 ± 6.4 years, and the mean gestational age was 34.9 ± 3.4 weeks. Low platelet count was identified in 34 (20.4%) women. Women aged 31–45 years had higher odds of low platelet count than those aged 18–30 years (61.8% vs. 36.1%; OR: 2.9, 95% CI: 1.3–6.2; $p=0.007$). Hypertension was significantly associated with low platelet count (44.1% vs. 24.8%; OR: 2.4, 95% CI: 1.1–5.2; $p=0.026$). Severe pre-eclampsia was associated with a lower mean platelet count and a higher frequency of low platelet count ($p<0.001$).

Conclusion: Low platelet count was relatively common among women with pre-eclampsia in the third trimester and was significantly associated with severe disease. Routine platelet count assessment may therefore be an important component of the evaluation of women with pre-eclampsia, particularly those with severe disease.

Keywords: Blood pressure; platelet count; pre-eclampsia; pregnancy; proteinuria.

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Introduction

Pre-eclampsia (PE) is a multisystem hypertensive disorder of pregnancy that typically develops after 20 weeks of gestation and remains a major cause of maternal and perinatal morbidity and mortality worldwide.^{1,2} It affects approximately 2–8% of pregnancies globally, while hypertensive disorders of pregnancy contribute substantially to maternal mortality.^{3,4} Current evidence suggests that pre-eclampsia begins with abnormal placentation and inadequate remodeling of the spiral arteries, followed by placental ischemia, oxidative stress, and the release of antiangiogenic and inflammatory mediators into the maternal circulation.^{5,6} This process contributes to

widespread endothelial dysfunction and maternal organ injury involving the vascular, renal, hepatic, and haematological systems.

Among the haematological abnormalities associated with pre-eclampsia, thrombocytopenia is one of the most clinically relevant because it reflects disease progression and may indicate increased maternal risk.^{7,8} ACOG defines thrombocytopenia in pregnancy as a platelet count below $150 \times 10^9/L$ while severe thrombocytopenia is recognized as an important marker of severe maternal disease.⁹ Recent evidence supports a close relation between platelet count and both the presence and severity of pre-eclampsia. A

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systematic review and meta-analysis found that platelet count was significantly lower in women with pre-eclampsia than in normotensive pregnant women, with a pooled mean difference of -32.83 and the reduction was greater in severe disease than in mild disease.¹⁰ More recent observational work has also shown that platelet indices change with worsening disease reinforcing the role of platelet count as a simple and accessible marker of maternal involvement in pre-eclampsia.¹¹

Despite this, the reported burden of low platelet count in women with pre-eclampsia varies considerably across studies which may reflect differences in case definition, disease severity, gestational age at inclusion and population characteristics. In low and middle-income settings where laboratory resources may be limited and delayed recognition of severe disease remains a challenge, platelet count retains practical importance because it is inexpensive, routinely available, and potentially useful for risk stratification at presentation.¹² There is also limited contemporary regional evidence describing the frequency of low platelet count specifically among women with pre-eclampsia in the third trimester at tertiary care settings in Pakistan. Generating local data is important because referral patterns, disease severity at presentation and access to antenatal monitoring may differ from those in other populations. Determining the frequency of low platelet count in this group may help clinicians identify the scale of haematological involvement in pre-eclampsia and support timely monitoring and management. On these basis, the present study was conducted to determine the frequency of low platelet count in the third trimester among pregnant women with pre-eclampsia presenting to a tertiary care hospital in Rahim Yar Khan, Pakistan.

Methodology

This cross-sectional study was carried out at the Department of Gynecology and Obstetrics, Sheikh Zayed Hospital, Rahim Yar Khan, Pakistan, from February 2025 to July 2025. Approval from the Institutional Ethical Committee was obtained before commencement of the study (No. 33/IRB/SZMC/SZH, dated 9 March 2024). A sample size of 167 was calculated using the OpenEpi sample size calculator, based on a reported proportion of thrombocytopenia among patients with pre-eclampsia of 19.4%,¹³ with a 95% confidence level and a 6% margin of error.

The inclusion criteria were women aged 18–45 years with a singleton pregnancy in the third trimester, between 29 and 41 weeks of gestation, who were diagnosed with pre-eclampsia. Gestational age and singleton pregnancy were confirmed by obstetric ultrasonography performed or reviewed by a consultant obstetrician. The exclusion criteria included women with a previous history of thrombocytopenia, as assessed from medical records, and those with fetal anomalies detected on the anomaly scan at 20 weeks of gestation, as assessed by a consultant obstetrician. Women with multiple pregnancies, as confirmed by a consultant obstetrician using obstetric ultrasonography, or those taking platelet-lowering drugs were also excluded.

Pre-eclampsia was defined as pregnancy beyond 20 weeks of gestation with a blood pressure $\geq 140/90$ mmHg on two separate occasions at least 4–6 hours apart and proteinuria >0.3 g in a 24-hour urine collection.¹ Non-probability consecutive sampling was used. Written informed consent was obtained from all participants.

Demographic information, including age (years), and clinical data, including gestational age, parity, gravidity, history of diabetes (yes/no), history of hypertension (yes/no), and severity of pre-eclampsia, were collected. A patient was considered diabetic if her HbA1c was $>6.4\%$ or if she had a history of taking antidiabetic medications. Hypertension was defined as a blood pressure $>130/90$ mmHg or a history of antihypertensive medication use.

Pre-eclampsia was classified as mild or severe. Mild pre-eclampsia was defined by a systolic blood pressure (SBP) of 140–160 mmHg, diastolic blood pressure (DBP) of 90–110 mmHg, urine output >500 mL/24 hours, urinary protein <5 g/24 hours, and absence of headache, visual disturbances, or epigastric pain. Severe pre-eclampsia was defined by an SBP ≥ 160 mmHg, DBP ≥ 110 mmHg, urine output ≤ 500 mL/24 hours, urinary protein ≥ 5 g/24 hours, 3+ proteinuria on dipstick testing, and the presence of headache, visual disturbances, or epigastric pain.¹

Using aseptic technique, a 3-mL blood sample was collected from each participant and sent to the institutional laboratory for analysis. A platelet count $<150 \times 10^9/L$ was considered low platelet count (LPC).¹¹

The collected data were analyzed using IBM SPSS Statistics version 26.0. Categorical variables were

presented as frequencies and percentages, whereas quantitative variables were expressed as means with standard deviations (SDs) or medians with interquartile ranges (IQRs), as appropriate. Potential effect modifiers, including age group, gestational age, gravidity, parity, diabetes status, history of hypertension, and severity of pre-eclampsia, were controlled through stratification. Post-stratification chi-square or Fisher's exact tests were used to assess the association between these variables and the outcome (LPC). Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. The mean platelet counts were compared according to the severity of pre-eclampsia using an independent-samples t-test. A p-value <0.05 was considered statistically significant.

Results

Among the 167 women included in the study, the mean age was 29.8 ± 6.4 years, and the mean gestational age was 34.9 ± 3.4 weeks. There were 61 (36.5%) primigravida women, while 67 (40.1%) were nulliparous. A rural residence was reported in 104 (62.3%) women. Diabetes mellitus and hypertension were documented in 21 (12.6%) and 48 (28.7%) women, respectively. The demographic and clinical characteristics of the study participants are presented in Table I.

Table I: Characteristics of women. (n=167)

Characteristics		N (%)
Age group (years)	18-30	98 (58.7%)
	31-45	69 (41.3%)
Gestational age (weeks)	29-34	79 (47.3%)
	35-41	88 (52.7%)
Gravida	Primigravida	61 (36.5%)
	Multigravida	106 (63.5%)
Parity	Nulliparous	67 (40.1%)
	Parous	100 (59.9%)
Residence	Urban	63 (37.7%)
	Rural	104 (62.3%)
Diabetes mellitus		21 (12.6%)
Hypertension		48 (28.7%)

Table II: Association of low platelet count with characteristics of females. (n=167)

Characteristics	Low platelet count (n=34)	No low platelet count (n=133)	OR with 95% CI	P-value
Age group (years)	18-30	85 (63.2%)	Reference	0.007
	31-45	48 (36.8%)	2.9 (1.3-6.2)	
Gestational age (weeks)	29-34	62 (46.6%)	Reference	0.724
	35-41	71 (53.4%)	0.9 (0.4-1.9)	
Gravida	Primigravida	51 (38.3%)	Reference	0.334
	Multigravida	82 (61.7%)	1.5 (0.7-3.4)	
Parity	Nulliparous	56 (42.1%)	Reference	0.300
	Parous	77 (57.9%)	1.5 (0.7-3.4)	
Residence	Urban	53 (39.8%)	Reference	0.262
	Rural	80 (60.2%)	1.6 (0.7-3.6)	
Diabetes mellitus	5 (14.7%)	16 (12.0%)	1.3 (0.4-3.7)	0.675
Hypertension	15 (44.1%)	33 (24.8%)	2.4 (1.1-5.2)	0.026

LPC was identified in 34 (20.4%) women. Women aged 31–45 years had higher odds of LPC than those aged 18–30 years (61.8% vs. 36.1%; OR: 2.9, 95% CI: 1.3–6.2; p=0.007). Hypertension was more common among women with LPC than among those without LPC (44.1% vs. 24.8%; OR: 2.4, 95% CI: 1.1–5.2; p=0.026). The association between LPC and the demographic and clinical characteristics of the study participants is presented in Table II.

The severity of pre-eclampsia was significantly associated with a lower mean platelet count (p<0.001), as shown in Figure I. Similarly, LPC was significantly more frequent among women with severe pre-eclampsia than among those with mild pre-eclampsia (37.5% vs. 9.7%; p<0.001). The detailed distribution is presented in Figure II.

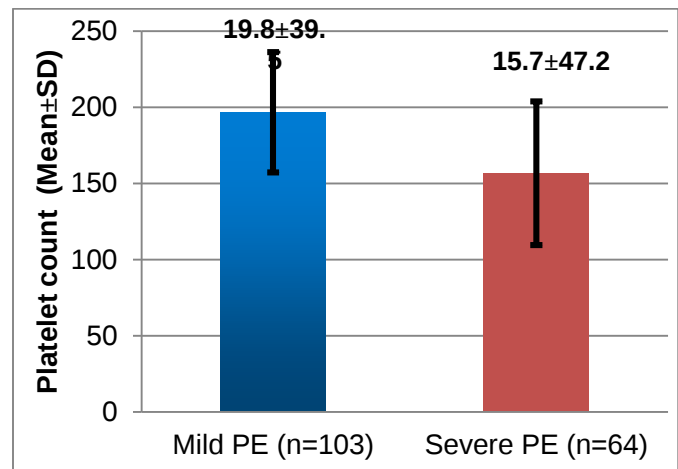


Figure I. Association of mean platelet count with severity of PE.

Discussion

This study found that low platelet count was present in 20.4% of women with pre-eclampsia in the third trimester. This finding indicates that nearly one in five women with pre-eclampsia in late pregnancy had thrombocytopenia at presentation, highlighting the

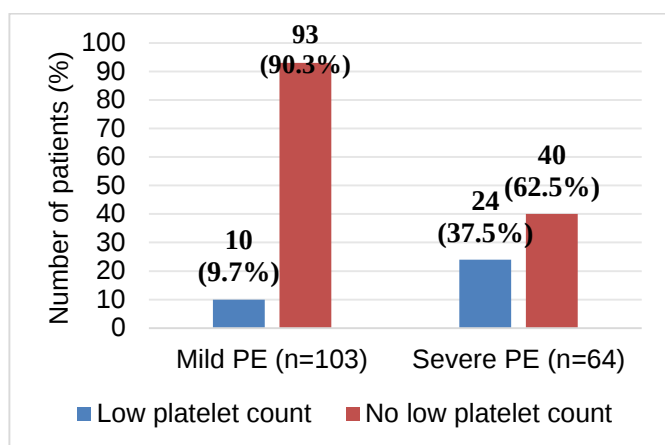


Figure II. Association of low platelet count with severity of PE.

clinical relevance of routine platelet assessment in such patients. A review noted that thrombocytopenia burden was higher when pregnancy specific pathological states like PE were present and when the presentation is in 3rd trimester which was the period in which thrombocytopenia was most often detected.¹⁴ The present estimate is also lower than reports drawn from cohorts enriched with hypertensive disease or mixed obstetric thrombocytopenia populations which is understandable because the present study excluded women with prior thrombocytopenia, multiple gestation, fetal anomalies, and exposure to platelet lowering drugs.¹⁵ The differences may be related to variation in study design, spectrum of disease severity, and eligibility criteria.

An important finding of this study was the close relation between platelet count and severity of pre-eclampsia. Severe pre-eclampsia had a substantially lower mean platelet count than mild pre-eclampsia, and the proportion with low platelet count was almost four times higher. A prospective study of women with PE reported a mean platelet count of $141.3 \pm 42.47 \times 10^3/\mu\text{L}$ in PE compared with $174.32 \pm 27.24 \times 10^3/\mu\text{L}$ in normotensive pregnancy and within the PE group the count fell further to $99.42 \pm 29.69 \times 10^3/\mu\text{L}$ in women with severe features compared with $149.18 \pm 39.89 \times 10^3/\mu\text{L}$ in those without severe features.¹⁶ A meta-analysis reported LPC to be significantly more prevalent among PE women.¹⁰ Platelet consumption in PE is thought to reflect widespread endothelial injury, platelet activation, microvascular deposition, and accelerated peripheral turnover.¹⁷ An observational study of platelet indices in PE noted that platelet count was lower in PE pregnancies while markers such as mean platelet volume were increased reinforcing the view that

thrombocytopenia is part of an activated and consumed platelet state rather than an isolated laboratory abnormality.¹⁸ This pathophysiological explanation provides a basis for the lower platelet counts seen in severe pre-eclampsia in the present study.

Platelet count a low cost and readily available marker that can contribute to triage in hospitals where more advanced biomarkers are not easily accessible. A routine complete blood count is inexpensive, familiar, and quickly actionable. LPC in women with PE could therefore prompt closer surveillance for disease progression, expedited review of coagulation status where indicated, and careful planning for delivery and anaesthesia.^{19,20} This may be particularly relevant in tertiary care settings receiving women with advanced disease or limited antenatal follow-up. The association between relatively older maternal age and LPC in this study aligns with recent literature of PE.²¹ Hypertension was another factor associated with LPC. Evidence identified higher SBP and DBP, and pre pregnancy diabetes as factors associated with PE.^{22,23}

Regarding limitations, the cross-sectional design limits causal inference and does not permit evaluation of whether platelet count declines over time before the onset of severe features. Maternal and fetal outcomes and their linkage with LPC were not assessed. Future studies should examine whether low platelet count predicts adverse maternal and neonatal outcomes, including postpartum haemorrhage, need for transfusion, preterm birth, and perinatal morbidity.

Conclusion

Low platelet count was a relatively common finding among women with pre-eclampsia in the third trimester and was significantly associated with severe disease. These findings support the importance of routine platelet count assessment as part of the evaluation of women with pre-eclampsia. Women with severe pre-eclampsia, older age, or coexisting hypertension may require closer laboratory monitoring.

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