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Original Research Article

PREGNANCY OUTCOMES IN WOMEN WITH THROMBOCYTOPAENIA: A PROSPECTIVE STUDY

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ABSTRACT

Background: Thrombocytopaenia is a common hematological abnormality in pregnancy, with outcomes varying widely depending on etiology and severity. While gestational thrombocytopaenia is generally benign, pathological forms such as immune thrombocytopaenic purpura (ITP) and hypertensive disorder–related thrombocytopaenia are associated with adverse maternal and neonatal outcomes. This study aimed to evaluate the clinical profile and pregnancy outcomes among women presenting with thrombocytopaenia at a tertiary care center.

Materials and Methods: This hospital-based prospective observational study included 69 pregnant women with platelet counts <150 ×10⁹/L. Etiology was classified as gestational thrombocytopaenia, ITP, or hypertensive disorder–related thrombocytopaenia in Faridabad. Severity was categorized as mild (100–150 ×10⁹/L), moderate (50–99 ×10⁹/L), or severe (<50 ×10⁹/L). Maternal outcomes (mode of delivery, postpartum hemorrhage, transfusion, ICU admission) and neonatal outcomes (preterm birth, low birth weight, NICU admission, neonatal thrombocytopaenia) were recorded. Data were analyzed using SPSS 20.0, with chi-square and ANOVA tests applied; $p<0.05$ was considered statistically significant.

Results: Gestational thrombocytopaenia accounted for 69.6% of cases, followed by hypertensive disorders (20.3%) and ITP (10.1%). Mild thrombocytopaenia was most common (66.7%). Hypertensive disorder–related thrombocytopaenia was associated with significantly higher cesarean delivery (71.4%, $p=0.030$) and postpartum hemorrhage rates (35.7%, $p=0.016$). ITP demonstrated the highest transfusion requirement (71.4%, $p<0.001$). Increasing severity correlated with rising rates of postpartum hemorrhage (6.5% mild vs. 37.5% severe; $p=0.020$), transfusion (4.3% mild vs. 62.5% severe; $p<0.001$), and ICU admission (2.2% mild vs. 37.5% severe; $p=0.004$). Neonatal thrombocytopaenia occurred in 14.5% overall, significantly higher in infants of mothers with ITP (57.1%, $p<0.001$). Preterm birth and low birth weight were more frequent in ITP and hypertensive groups ($p=0.020$ and $p=0.003$, respectively). No maternal deaths were recorded.

Conclusion: Maternal and neonatal outcomes in thrombocytopaenia vary significantly by etiology and severity. Gestational thrombocytopaenia remains benign, whereas hypertensive disorder–related thrombocytopaenia and ITP are associated with higher morbidity. Early identification, close monitoring, and multidisciplinary management are essential to improving outcomes in pregnancies complicated by pathological thrombocytopaenia.

Keywords: Pregnancy; Immune thrombocytopaenic purpura; Preeclampsia; Gestational thrombocytopaenia; HELLP syndrome; Neonatal thrombocytopaenia.

INTRODUCTION

Thrombocytopaenia, defined as a platelet count <150 × 10⁹/L, is the second most common hematological abnormality in pregnancy after anemia and affects 7–12% of all pregnancies globally.[1] The condition encompasses a heterogeneous spectrum ranging from benign gestational thrombocytopaenia to severe pathological disorders such as immune thrombocytopaenic purpura (ITP), preeclampsia, HELLP syndrome, and thrombotic microangiopathies. Gestational thrombocytopaenia accounts for 70–80% of all cases and is usually mild (platelet count 100–150 × 10⁹/L), asymptomatic, and identified in late pregnancy without adverse maternal or fetal consequences.[2] In contrast, ITP contributes to 3–5% of cases and is characterized by immune-mediated platelet destruction, with 10–15% of affected newborns developing neonatal thrombocytopaenia due to passive transfer of maternal antibodies.[3]

Hypertensive disorders of pregnancy, including preeclampsia and HELLP syndrome, represent 15–20% of thrombocytopaenia cases and are associated with significant maternal morbidity such as severe hypertension, hepatic dysfunction, coagulopathy, and increased risk of obstetric hemorrhage.[4] Severe thrombocytopaenia (<50 × 10⁹/L) is uncommon in pregnancy, occurring in <0.5% of women, but is clinically important as it impacts decisions regarding anesthesia, timing and mode of delivery, and the need for blood product support.[5] Platelet thresholds of >80 × 10⁹/L are generally considered safe for neuraxial anesthesia, making timely identification and monitoring crucial.[6]

Maternal outcomes associated with thrombocytopaenia vary with etiology and severity. Women with moderate-to-severe thrombocytopaenia have a higher incidence of postpartum hemorrhage, emergency cesarean section, ICU admission, and transfusion requirements.[7] Perinatal outcomes can also be affected; studies report increased risks of preterm birth (up to 25–30%), low birth weight, intrauterine growth restriction, and neonatal intensive care unit (NICU) admission, particularly in pregnancies complicated by hypertensive disorders or ITP.[8] Neonatal thrombocytopaenia occurs in 8–15% of infants born to thrombocytopaenic mothers, with the risk rising to 20–25% in maternal ITP.[9]

Despite the clinical relevance of thrombocytopaenia in pregnancy, substantial geographic variability exists in its prevalence, etiological distribution, and associated outcomes due to differences in demographics, antenatal care quality, referral patterns, and institutional management protocols. In many low- and middle-income countries, delayed diagnosis and limited access to laboratory monitoring contribute to increased maternal and neonatal complications.[10] Therefore, generating region-specific evidence is essential to enhance risk stratification, optimize obstetric management, and reduce preventable morbidity.

The present study aimed to evaluate the maternal and perinatal outcomes of pregnancies complicated with thrombocytopaenia in a tertiary care setting and determining how these outcomes vary with the severity and underlying cause of thrombocytopaenia.

MATERIALS AND METHODS

Study Design and Setting: This hospital-based observational study was conducted in the Department of Obstetrics and Gynaecology at a Al Falah School of Medical Science and Research Centre, Faridabad, catering to both rural and urban populations. The study was carried out over a period of 12 months, from June 2024 to May 2025. The institution receives a high proportion of high-risk antenatal referrals, ensuring adequate representation of pregnancy-related hematological disorders. Ethical approval was obtained from the Institutional Ethics Committee prior to commencement of the study, and written informed consent was obtained from all participants.

Study Population and Eligibility Criteria: All pregnant women presenting to the antenatal clinic, emergency obstetric services, or admitted for delivery during the study period were screened for thrombocytopaenia. Women with a platelet count <150 × 10⁹/L confirmed on an automated hematology analyzer were eligible. Diagnosis was reconfirmed with a manual platelet count. Inclusion criteria comprised pregnant women in the second or third trimester with documented thrombocytopaenia, irrespective of parity or mode of conception. Exclusion criteria included women with known chronic liver disease, HIV infection, hematological malignancies, chronic renal disease, autoimmune disorders unrelated to pregnancy, or those on medications known to cause thrombocytopaenia such as heparin or anticonvulsants. Women with incomplete medical records or unwillingness to participate were also excluded.

Classification of Thrombocytopaenia: Participants were categorized based on etiology into gestational thrombocytopaenia, immune thrombocytopaenic purpura (ITP), and hypertensive disorder–related thrombocytopaenia including preeclampsia and HELLP syndrome. Etiological classification was based on clinical history, blood pressure recordings, liver function tests, peripheral smear findings, and antenatal records. Thrombocytopaenia severity was stratified as mild (100–150 × 10⁹/L), moderate (50–99 × 10⁹/L), and severe (<50 × 10⁹/L).

Data Collection Procedures: Detailed demographic and obstetric information including age, parity, gestational age, past and current medical history, and prior episodes of thrombocytopaenia was recorded using a structured data collection form. Clinical findings, blood pressure measurements,

presence of edema, symptoms of bleeding, and evidence of hypertensive disorders were documented. Laboratory investigations included complete blood count, platelet indices, liver and renal function tests, coagulation profile, and a peripheral smear review. Platelet counts were monitored serially during antenatal care, admission, labor, and postpartum period as per hospital protocol. Maternal outcomes assessed included mode of delivery, need for induction, postpartum hemorrhage, ICU admission, platelet transfusion requirements, and maternal morbidity or mortality. Neonatal outcomes included birth weight, gestational age at delivery, Apgar scores at 1 and 5 minutes, NICU admission, neonatal thrombocytopaenia (defined as platelet count <150 × 10^9/L), and perinatal mortality.

Follow-up and Outcome Assessment: Women were monitored intrapartum and postpartum until discharge. Neonates underwent clinical assessment and screening platelet count within 24 hours of birth. Those with low platelet counts were followed until resolution or NICU discharge. Maternal outcomes were evaluated up to 6 weeks postpartum wherever feasible during follow-up visits or telephonic contact.

Statistical Analysis: Data were entered into a predesigned spreadsheet and analyzed using IBM SPSS Statistics version 20.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as means and standard deviations, while categorical variables were summarized as frequencies and percentages. The association between severity of thrombocytopaenia and maternal or neonatal outcomes was assessed using the Chi-square test for categorical variables. Comparisons of continuous variables across groups were performed using one-way ANOVA. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 69 pregnant women with thrombocytopaenia were included, with a mean maternal age of 26.5 ± 4.3 years, which did not differ significantly across etiological groups (p = 0.380). The mean gestational age at diagnosis was 31.9 ± 4.6 weeks, with women in the hypertensive disorder–related and ITP groups presenting significantly earlier than those with gestational thrombocytopaenia (p = 0.010). Primigravidae constituted 55.1% of the cohort, with no significant intergroup differences (p = 0.190). The overall mean hemoglobin was 10.6 ± 1.3 g/dL, trending lower in hypertensive cases, though not statistically significant (p = 0.080). Mean platelet counts varied markedly by etiology (115 ± 15 in gestational TP, 70 ± 12 in hypertensive disorders, and 45 ± 8 in ITP), with a highly significant difference (p < 0.001) [Table 1].

Table 1: Baseline Demographic and Obstetric Characteristics of the Study Population (n = 69).

Characteristic	Overall (n = 69)	Gestational TP* (n = 48)	ITP (n = 7)	HTN-related† (n = 14)	p-value
	Frequency (%)/mean ± SD				
Maternal age (years)	26.5 ± 4.3	26.2 ± 3.8	28.4 ± 4.1	25.1 ± 4.5	0.380
Gestational age at diagnosis (weeks)	31.9 ± 4.6	33.1 ± 3.2	29.6 ± 5.4	28.2 ± 6.2	0.010
Primigravida	38 (55.1)	29 (60.4)	2 (28.6)	7 (50.0)	0.190
Hemoglobin (g/dL)	10.6 ± 1.3	10.8 ± 1.1	10.4 ± 1.6	10.0 ± 1.4	0.080
Platelet count at presentation (×10^9/L)	95 ± 33	115 ± 15	45 ± 8	70 ± 12	<0.001

TP = Thrombocytopaenia; HTN-related = Preeclampsia ± HELLP

Gestational thrombocytopaenia was the predominant etiology, accounting for 69.6% (48/69) of cases, followed by hypertensive disorder–related thrombocytopaenia in 20.3%, and ITP in 10.1%. Based on severity, 66.7% of women had mild thrombocytopaenia (100–150 ×10^9/L), 21.7% had moderate thrombocytopaenia (50–99 ×10^9/L), and 11.6% had severe thrombocytopaenia (<50 ×10^9/L), reflecting a typical tertiary-care referral pattern with a minority presenting with severe disease [Table 2].

Table 2: Etiology and Severity Distribution (n = 69).

Variable	Frequency	%
Etiology		
Gestational thrombocytopaenia	48	69.6%
Immune thrombocytopaenic purpura (ITP)	7	10.1%
Hypertensive disorders (preeclampsia / HELLP)	14	20.3%
Severity (based on platelet count)		
Mild (100–150 ×10^9/L)	46	66.7%
Moderate (50–99 ×10^9/L)	15	21.7%
Severe (<50 ×10^9/L)	8	11.6%

ITP = Immune Thrombocytopaenic Purpura; HELLP = Hemolysis, Elevated Liver Enzymes and Low Platelets.

Maternal outcomes varied significantly with etiology. The overall cesarean section rate was 40.6%, with hypertensive disorder–related thrombocytopaenia demonstrating the highest rate (71.4%, p = 0.030). PPH occurred in 13% of the cohort, significantly more common in hypertensive cases (35.7%, p = 0.016). Transfusion requirements were markedly elevated in ITP (71.4% vs. 8.3% in

gestational TP; p < 0.001). ICU admission occurred in 8.7%, with significantly higher rates in ITP and hypertensive groups compared with gestational TP (p = 0.040). No maternal mortality was reported [Table 3]

Table 3: Maternal Outcomes by Etiology (n = 69).

Maternal outcome	Overall (n = 69)	Gestational TP (n = 48)	ITP (n = 7)	HTN-related (n = 14)	p-value
	Frequency (%)				
Cesarean delivery	28 (40.6)	16 (33.3)	2 (28.6)	10 (71.4)	0.030
Postpartum hemorrhage (PPH)	9 (13.0)	3 (6.3)	1 (14.3)	5 (35.7)	0.016
Blood / platelet transfusion required	11 (15.9)	4 (8.3)	5 (71.4)	2 (14.3)	<0.001
ICU admission	6 (8.7)	2 (4.2)	2 (28.6)	2 (14.3)	0.040
Maternal mortality	0 (0)	0 (0)	0 (0)	0 (0)	—

ITP = Immune Thrombocytopaenic Purpura; PPH = Postpartum Hemorrhage; ICU = Intensive Care Unit; HTN-related = Preeclampsia ± HELLP.

Worsening thrombocytopaenia severity demonstrated a clear impact on adverse maternal outcomes. PPH increased progressively from 6.5% in mild cases to 20% in moderate and 37.5% in severe thrombocytopaenia (p = 0.020). Transfusion requirements also rose significantly across severity levels (4.3%, 26.7%, and 62.5%, respectively; p < 0.001). ICU admissions were uncommon in mild thrombocytopaenia (2.2%), but rose sharply in moderate (13.3%) and severe (37.5%) categories (p = 0.004). Cesarean rates increased with severity but were not statistically significant (p = 0.260) [Table 4].

Table 4: Maternal Outcomes by Severity of Thrombocytopaenia (n = 69).

Outcome	Mild (n = 46)	Moderate (n = 15)	Severe (n = 8)	p-value
	Frequency (%)			
Cesarean delivery	16 (34.8)	8 (53.3)	4 (50.0)	0.260
PPH	3 (6.5)	3 (20.0)	3 (37.5)	0.020
Transfusion (blood/platelets)	2 (4.3)	4 (26.7)	5 (62.5)	<0.001
ICU admission	1 (2.2)	2 (13.3)	3 (37.5)	0.004

Severity defined as Mild = 100–150 ×10⁹/L; Moderate = 50–99 ×10⁹/L; Severe = <50 ×10⁹/L; PPH = Postpartum Hemorrhage; ICU = Intensive Care Unit.

Neonatal outcomes varied substantially with the underlying maternal etiology. Preterm delivery occurred in 26.1% of all births, highest among ITP (57.1%) and hypertensive disorder–related thrombocytopaenia (35.7%) groups (p = 0.020). Low birth weight was seen in 29%, with a striking 71.4% in ITP (p = 0.003). NICU admission was required in 29%, and though trends were higher in hypertensive and ITP groups, the difference did not reach statistical significance (p = 0.074). Neonatal thrombocytopaenia developed in 14.5%, markedly more common in ITP (57.1%) and hypertensive disorders (28.6%) compared with gestational TP (4.2%) (p < 0.001). Two perinatal deaths (2.9%) were observed, one in the ITP group and one in the hypertensive disorder group. Both deaths occurred in clinically high-risk pregnancies and were not directly attributable to maternal thrombocytopaenia, but rather associated with underlying conditions such as prematurity and severe hypertensive disease groups [Table 5].

Table 5: Neonatal Outcomes by Etiology (n = 69 live births).

Neonatal outcome	Overall (n = 69)	Gestational TP (n = 48)	ITP (n = 7)	HTN-related (n = 14)	p-value
	Frequency (%)				
Preterm delivery (<37 weeks)	18 (26.1)	9 (18.8)	4 (57.1)	5 (35.7)	0.020
Low birth weight (<2500 g)	20 (29.0)	11 (22.9)	5 (71.4)	4 (28.6)	0.003
NICU admission	20 (29.0)	10 (20.8)	3 (42.9)	7 (50.0)	0.074
Neonatal thrombocytopaenia (<150 ×10⁹/L)	10 (14.5)	2 (4.2)	4 (57.1)	4 (28.6)	< 0.001
Perinatal mortality (stillbirth + early neonatal death)	2 (2.9)	0 (0)	1 (14.3)	1 (7.1)	0.120

ITP = Immune Thrombocytopaenic Purpura; NICU = Neonatal Intensive Care Unit.

DISCUSSION

In this prospective evaluation of 69 pregnant women with thrombocytopaenia, we observed that gestational thrombocytopaenia was the predominant etiology (69.6%), followed by hypertensive disorder–related thrombocytopaenia (20.3%) and immune thrombocytopaenic purpura (ITP) (10.1%). This etiological distribution is consistent with previous Indian and international studies, by Pandey et al., Sridhar et al., and Ciobanu et al., which report gestational thrombocytopaenia accounting for 70–

80% of pregnancy-associated thrombocytopaenia.[11-13] The predominance of mild thrombocytopaenia (66.7%) in our cohort also parallels findings from Desai et al., and Vishwekar et al., who similarly reported that most cases are incidentally detected and clinically benign.[14,15]

The mean gestational age at diagnosis differed significantly across etiologies (p = 0.01), with hypertensive disorder–related thrombocytopaenia and ITP presenting earlier than gestational thrombocytopaenia. This finding is pathophysiologically plausible, as endothelial dysfunction and platelet consumption begin early in preeclampsia and HELLP syndrome, whereas gestational thrombocytopaenia typically manifests in the third trimester due to hemodilutional effects and accelerated platelet turnover.[16] The significantly lower platelet counts in ITP (mean 45 $\times 10^9$/L) compared with gestational thrombocytopaenia (115 $\times 10^9$/L) reflect the autoimmune platelet destruction characteristic of ITP and mirror findings from Dahiphale et al., and Gonzalez-Porras et al.[17,18]

Our study identified substantial differences in maternal outcomes by etiology. Hypertensive disorder–related thrombocytopaenia was associated with significantly higher cesarean rates (71.4%, p = 0.030) and postpartum hemorrhage (PPH) (35.7%, p = 0.016). These findings are consistent with the well-established risks of coagulopathy, hepatic dysfunction, and endothelial injury in preeclampsia/HELLP, as reported by Lee et al., and Xu et al.[19,20] The high transfusion requirement in ITP patients (71.4%, p < 0.001) aligns with studies by Wakode et al., and Kashyap et al., emphasizing the severe thrombocytopaenia and bleeding tendencies characteristic of ITP pregnancies.[21,22] ICU admissions were also significantly higher in severe thrombocytopaenia groups (p = 0.004), further confirming that severity rather than etiology alone is an important determinant of maternal morbidity.

Severity-based analysis demonstrated clear escalation in adverse outcomes with falling platelet counts. PPH increased from 6.5% in mild disease to 37.5% in severe thrombocytopaenia (p = 0.02), and transfusion needs rose from 4.3% to 62.5% across severity categories (p < 0.001). These trends are comparable to findings reported by Singhe et al., who concluded that maternal hemorrhagic complications rise markedly when platelet counts fall below 50 $\times 10^9$/L.[23] The biological basis lies in both quantitative platelet deficiency and qualitative platelet dysfunction, especially in preeclampsia where endothelial activation leads to accelerated platelet consumption.[24]

Neonatal outcomes varied significantly with maternal etiology. Preterm births were more common in ITP (57.1%) and hypertensive disorders (35.7%) compared with gestational thrombocytopaenia (18.8%; p = 0.020). This is consistent with the literature by Mushahary et al., and Gaba et al., where preeclampsia and ITP are known predictors of iatrogenic or spontaneous preterm delivery.[25,26] Low birth weight (LBW) was significantly higher in neonates of ITP mothers (71.4%, p = 0.003), potentially reflecting maternal disease activity and its impact on placental perfusion. Neonatal thrombocytopaenia occurred in 14.5% overall but was disproportionately higher in ITP (57.1%) and hypertensive groups (28.6%; p < 0.001). This finding corroborates prior studies by Karakurt et al., and Aslan et al., reporting a 20–25% risk of neonatal thrombocytopaenia in ITP pregnancies due to transplacental passage of antiplatelet antibodies.[27,28] Hypertensive disorders, on the other hand, compromise placental circulation, predisposing infants to growth restriction and fetal platelet consumption.[28]

Our findings affirm that gestational thrombocytopaenia remains benign with excellent maternal and neonatal outcomes, whereas thrombocytopaenia associated with hypertensive disorders and ITP carries significantly higher risks.[29] Similar patterns have been documented by Luo et al., who emphasized the importance of etiological differentiation during antenatal evaluation.[30] Importantly, no maternal deaths occurred in our study, reflecting timely monitoring and evidence-based intervention protocols typical of tertiary centers. The two perinatal deaths in our cohort occurred exclusively in pregnancies complicated by ITP and hypertensive disorders, conditions well known for placental insufficiency and prematurity. Importantly, neither death appeared directly related to maternal thrombocytopaenia, which aligns with existing literature showing that perinatal mortality in such cases is more strongly influenced by the underlying pathology than the platelet count itself.[29,30]

Limitations

The study's relatively modest sample size may limit the ability to detect subtle differences in some outcomes, particularly rare complications. As a single-center study, the results may not be fully generalizable to primary or secondary care settings, where resource availability and referral patterns differ. The observational design precludes establishing causal relationships, and potential confounders such as nutritional status, medication history, or unrecognized autoimmune conditions may not have been fully accounted for. Neonatal follow-up was limited to the early postpartum period, preventing long-term assessment of infants with thrombocytopaenia. Despite these limitations, the study adds valuable region-specific evidence to the limited literature on pregnancy-related thrombocytopaenia in developing countries.

CONCLUSION

The present study demonstrates that the clinical outcomes of pregnancies complicated by

thrombocytopaenia are strongly influenced by both the underlying etiology and the severity of platelet reduction. Gestational thrombocytopaenia, the most common subtype in our cohort, was associated with favorable maternal and neonatal outcomes, reaffirming its benign nature. In contrast, hypertensive disorder–related thrombocytopaenia and ITP were linked with significantly higher rates of postpartum hemorrhage, transfusion requirements, ICU admissions, preterm birth, neonatal thrombocytopaenia, and low birth weight. The progressive rise in adverse outcomes with increasing severity of thrombocytopaenia further underscores the importance of timely diagnosis, close monitoring, and individualized management strategies. Overall, our findings highlight the need for early risk stratification and a multidisciplinary approach to optimize outcomes in pregnancies affected by pathological thrombocytopaenia.

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