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Original Research Article

STUDY ON MODERATE TO SEVERE THROMBOCYTOPENIA IN PREGNANCY

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ABSTRACT

Background: Thrombocytopenia is a common haematologic abnormality in pregnancy, with moderate-to-severe cases potentially leading to adverse maternal and foetal outcomes. This study aimed to evaluate the maternal and foetal outcomes in pregnant women with mild-to-severe thrombocytopenia.

Materials and Methods: This prospective observational study included 100 pregnant women at the Institute of Obstetrics and Gynaecology, Madras Medical College, over a year. Women in their third trimester with mild-to-severe thrombocytopenia (platelet count $<100 \times 10^9$/L) were included. Clinical data, including maternal age, parity, gestational age, diagnosis, mode of delivery, and complications, were recorded. Foetal outcomes, such as birth weight, APGAR scores, neonatal complications, and platelet counts, were also analysed.

Results: The most common cause of thrombocytopenia was severe preeclampsia (48%), followed by gestational thrombocytopenia (31%) and immune-mediated thrombocytopenia (7%). Most patients (92%) had moderate thrombocytopenia, whereas 8% had severe thrombocytopenia. Most deliveries were vaginal (59%), followed by emergency caesarean sections (37%). Postpartum haemorrhage (17%) and acute kidney injury (7%) were the most frequent maternal complications. Neonatal complications included transient tachypnoea of the newborn (23%) and perinatal asphyxia (11%). The overall live birth rate was 97%, with two stillbirths and one intrauterine foetal death.

Conclusion: Moderate to severe thrombocytopenia during pregnancy, particularly in association with hypertensive disorders, is linked to increased maternal morbidity and adverse foetal outcomes. Early recognition and appropriate management can improve the prognosis. Notably, gestational thrombocytopenia had no significant adverse effects, and platelet counts normalised postpartum.

Keywords: Thrombocytopenia in pregnancy, severe preeclampsia, maternal outcomes, foetal outcomes, platelet count.

INTRODUCTION

Thrombocytopenia in pregnancy may arise from a variety of aetiologies, encompassing both pregnancy-specific and non-specific causes.[1] Pregnancy-specific causes include gestational thrombocytopenia, hypertensive disorders such as preeclampsia, eclampsia, and HELLP syndrome, as well as liver conditions such as acute fatty liver of pregnancy.[2] On the other hand, non-specific causes involve immune thrombocytopenia, autoimmune disorders such as systemic lupus erythematosus (SLE) and antiphospholipid antibody syndrome (APLA), and infections caused by viruses such as HIV, cytomegalovirus (CMV), and Epstein-Barr virus (EBV). Additional nonspecific causes include drug-induced thrombocytopenia, thrombotic microangiopathies, hypersplenism, and inherited thrombocytopenia, such as the May-Hegglin anomaly. Understanding these diverse aetiologies is essential for accurately diagnosing and appropriately managing thrombocytopenia during pregnancy.[3]

Normal pregnancy is associated with a physiological decline in the platelet count. Although the exact mechanism underlying this reduction remains unclear, it has been postulated that dilutional effects, decreased platelet production, or increased platelet turnover during pregnancy may contribute to this phenomenon. In some pregnant women, this decline reaches levels that fall within the thrombocytopenic range.[4] Thrombocytopenia can be classified as mild (platelet count of 100,000–150,000 x10*9/l), moderate (platelet count of 50,000–100,000x10*9/l) or severe (platelet count <50,000 x10*9/l).[6]

The overall incidence of thrombocytopenia during pregnancy ranges from 6% to 10%. Among the various causes, gestational thrombocytopenia is the most prevalent, accounting for approximately 70% of the cases. Hypertensive disorders, including preeclampsia, eclampsia, and HELLP syndrome, contribute to approximately 21% of cases.[7] Immune-mediated thrombocytopenia, including idiopathic thrombocytopenic purpura (ITP), constitutes 4.1% of cases. Although this represents a small proportion, immune-mediated thrombocytopenia can lead to significant morbidity and mortality, necessitating close monitoring and timely intervention. Thrombocytopenia in pregnancy can have varying prognostic implications, ranging from benign and self-limiting conditions to life-threatening disorders requiring urgent medical attention.[8]

Understanding the underlying causes, classification, and clinical significance of thrombocytopenia in pregnancy is necessary for optimal maternal and foetal outcomes. Proper recognition of this condition facilitates timely intervention, reduces the risk of complications, and improves the overall prognosis. Hence, this study aimed to investigate the maternal and foetal outcomes of moderate-to-severe thrombocytopenia during pregnancy.

MATERIALS AND METHODS

This prospective observational study included 100 pregnant women at the Institute of Obstetrics and Gynaecology, Madras Medical College, over one year. The study was conducted after receiving approval from the Institutional Ethics Committee, and informed consent was obtained from all patients.

Inclusion and exclusion criteria

Antenatal women in the 3rd trimester admitted to IOG with moderate to severe thrombocytopenia were included, while antenatal women with normal platelet counts were excluded from the study.

Methods

The study included 100 pregnant women in their third trimester who were diagnosed with moderate-to-severe thrombocytopenia (platelet count <100 × 109/µL). Patients were recruited from the inpatient wards of the hospital. Maternal demographic and clinical data, including age, parity, and gestational age at the time of diagnosis, were recorded at the time of admission. The aetiology of thrombocytopenia was classified as pregnancy-related (e.g. preeclampsia, HELLP syndrome, and gestational thrombocytopenia) or non-pregnancy-related (for example, immune thrombocytopenia, infections, and drug-induced).

The mode of delivery was categorised as vaginal delivery, emergency lower-segment caesarean section (LSCS), or vacuum-assisted delivery. Maternal complications, such as postpartum haemorrhage (PPH), acute kidney injury (AKI), disseminated intravascular coagulation (DIC), and the requirement for platelet transfusions or intensive care unit (ICU) admission, were documented.

Foetal outcomes were also recorded, including birth weight, which was classified as low birth weight (<2.5 kg) or normal birth weight (>2.5 kg). APGAR scores were assessed at 1 and 5 min post-delivery. Neonatal complications such as transient tachypnoea of the newborn (TTN), respiratory distress syndrome (RDS), perinatal asphyxia, and hypoxic-ischaemic encephalopathy (HIE) were noted. Additionally, foetal platelet counts at birth were measured to assess neonatal thrombocytopenia.

Statistical Analysis

Data are presented as mean, standard deviation, frequency, and percentage. Continuable variables were compared using the independent sample t-test. Categorical variables were compared using Pearson's chi-square test. Significance was defined as P values less than 0.05 using a two-tailed test. Data analysis was performed using IBM SPSS version 21.0.

RESULTS

Most patients were between 26 and 30 years of age, followed by those between 21 and 25 years of age. Most patients were primigravida, followed by 2nd gravida. Most patients were above 37 weeks gestation, and 35% were above 38 weeks gestation. The majority of patients affected by preeclampsia were 48%. Most patients had moderate thrombocytopenia (92%), while the remaining 8% had severe thrombocytopenia. Most patients were delivered through labour, natural (59%), followed by emergency LSCS (37%). Approximately 17% of the patients had PPH and 7% had AKI (Table 1).

Table 1: Maternal characteristics and outcomes

		N (%)
Age (years)	<20	5 (5%)
	21 to 25	38 (38%)
	26 to 30	45 (45%)

	31 to 35	12 (12%)
Parity	PRIMI	43 (43%)
	G2P1L1	38 (38%)
	G2A1	8 (8%)
	G3P2L2	5 (5%)
	G3P2L1	3 (3%)
	G3P1L0A1	1 (1%)
	G3A2	2 (2%)
Gestational age	36 weeks ± days	8 (8%)
	37 weeks ± days	47 (47%)
	38 weeks ± days	35 (35%)
	39 weeks ± days	10 (10%)
Diagnosis	Severe Pre-eclampsia	48 (48%)
	Gestational thrombocytopenia	31 (31%)
	Immune-mediated Thrombocytopenia	7 (7%)
	HELLP syndrome	6 (6%)
	Eclampsia	4 (4%)
	Dengue Haemorrhagic fever	3 (3%)
	Viral hepatitis	1 (1%)
Thrombocytopenia (µL)	50,000 to 1 lakh	92 (92%)
	<50,000	8 (8%)
Mode of delivery	Labour natural	59 (59%)
	Emergency LSCS	37 (37%)
	Vacuum-assisted delivery	4 (4%)
Complications	Acute Kidney Injury	7 (7%)
	Post-partum haemorrhage	17 (17%)
	Disseminated intravascular coagulation	2 (2%)
	No complications	74 (74%)

Most patients received antihypertensive therapy (58%), followed by steroid therapy (31%) and blood transfusion (27%). After delivery, only 9% of patients had moderate thrombocytopenia compared to 92% pre-delivery, and no patients had severe thrombocytopenia compared to 8% pre-delivery. Adverse foetal outcomes were observed in three cases, with two stillbirths and one intrauterine death.

Most babies were female (62% vs. 38%). Approximately 51% of the babies had no complications, and TTN was the most common complication, present in 23% of the babies, followed by perinatal asphyxia in 11%. Low birth weight was observed in 4% of the infants. All babies had platelet counts within the normal range (1.5–2.0 lakh µL) (Table 2).

Table 2: Maternal and foetal characteristics

		N (%)
Treatment modality	Antihypertensive therapy	58 (58%)
	Blood transfusion	27 (27%)
	Steroid therapy	31 (31%)
Thrombocytopenia	Moderate (50,000 to 1 lakh cells/mm³)	9 (9%)
	Severe (<50,000 cells/mm³)	0
	Normal (>1 lakh cells/mm³)	91 (91%)
Foetal outcome	Live birth	97 (97%)
	Stillbirth	2 (2%)
	Intra-uterine death	1 (1%)
Sex of baby	Boy	38 (38%)
	Girl	62 (62%)
Complications	Well baby	51 (51%)
	Transient tachypnoea of newborn	23 (23%)
	Hypoxic ischemic encephalopathy	2 (2%)
	Perinatal asphyxia	11 (11%)
	Respiratory distress syndrome	8 (8%)
	Stillbirth/Intra-uterine death	3 (3%)
Birth weight (kg)	<2.5	4 (4.1%)
	>2.5	93 (95.9%)
Foetal platelet count (µL)	1 to 1.5 lakhs	17 (17.5%)
	1.5 to 2.0 lakhs	80 (82.5%)

The mean platelet count before delivery was 82,000±16,368, which significantly increased after delivery to 121,470±15,940 (p<0.001). The mean platelet count among patients with moderate thrombocytopenia was significantly higher than that among patients with severe thrombocytopenia (1,69,000±14,160 vs 1,56,000±15,144, p=0.038) (Table 3).

Table 3: Platelet counts in thrombocytopenic patients

		Mean±SD	P-value
Thrombocytopenia	Before delivery	82000±16368	<0.001
	After delivery	121470±15940	
Thrombocytopenia stages	Moderate	169000±14160	0.038
	Severe	156000±15144	

The APGAR score at 1 min was 7 or higher in 84% of the newborns, with 47% scoring 8 and 36% scoring 7. A few cases had lower scores: 3% scored 0, 3% scored 5, and 10% scored between 6 and 6. At 5 min, most newborns scored 9 (47%) or 8 (36%), showing good neonatal adaptation. Lower scores included 3% scoring 0, 3% scoring 7, and 10% scoring between 7 and 8 (Table 4).

Table 4: APGAR score

APGAR score-1 minute	APGAR score-5 minutes	N (%)
0	0	3 (3%)
5	7	3 (3%)
6	7	4 (4%)
6	8	6 (6%)
7	8	36 (36%)
8	8	1 (1%)
8	9	47 (47%)

The mean foetal platelet counts before and after delivery and the maternal platelet count were insignificant (r=0.016, p=0.876); (r=0.01, p=0.923) (Table 5).

Table 5: Correlation between foetal and maternal platelet count (n=97)

Foetal platelet count	Pearson correlation	P-value
Pre-delivery maternal platelet count	0.016	0.876
Post-delivery maternal platelet count	0.01	0.923

DISCUSSION

Our study found that severe preeclampsia (48%) was the most common cause of thrombocytopenia, followed by gestational thrombocytopenia (31%), immune thrombocytopenia (7%), and HELLP syndrome (6%). These findings are consistent with prior research by Singh et al., who reported gestational thrombocytopenia in 64.2% of cases, followed by hypertensive and hepatic disorders.9 Similarly, Sheiner et al. identified gestational thrombocytopenia as the leading cause (59.3%), followed by immune thrombocytopenic purpura (ITP) (11.05%), preeclampsia (10.05%), and HELLP syndrome (12.06%).[10]

In our study, the prevalence of emergency LSCS (37%) was comparable to findings by Al-Husban et al., who observed a significantly higher risk of caesarean delivery in women with moderate to severe thrombocytopenia.[11]

In our study, PPH was observed in 17% of the population, consistent with Borhany et al., who found that 25.3% of women with thrombocytopenia and platelet counts <50,000/µL required platelet transfusions due to bleeding complications.[12] Samoon observed that the average birth weight and Apgar scores at both 1 and 5 minutes were lower in the cases than in the control group.[14] Our study confirms prior findings that thrombocytopenic pregnancies carry an increased risk of haemorrhagic complications, although the majority remain uneventful.

We found a live birth rate of 97%, with 2% stillbirths and 1% of IUFD. In comparison, Parnas et al. observed a significantly higher risk of stillbirth (65 per 1000 births) among mothers with thrombocytopenia.10 Al-Husban et al. also reported an association between severe maternal thrombocytopenia and IUFD, IUGR, and preterm delivery.[11]

In our study, TTN was identified in 23% of neonates, which aligns with the findings of Ali et al., who demonstrated that TTN occurs more frequently in neonates of thrombocytopenic mothers.[15] Ozkan et al. also reported that thrombocytopenic mothers had a higher proportion of neonates with thrombocytopenia, although no direct complications were attributable to the mode of delivery.[16] However, our study did not find a significant association between thrombocytopenia and neonatal morbidity.

Our study confirmed that neonatal platelet counts were within the normal range (1.5-2.0 lakh/µL) in all cases, supporting the findings of Boehlen et al., who found no cases of severe neonatal thrombocytopenia among thrombocytopenic mothers.[13] However, other studies, such as Webert et al., reported that up to 25.2% of neonates born to mothers with ITP had platelet counts lower than 150 × 10^9/L, with 9% experiencing severe thrombocytopenia.[17] Michel et al. found that intravenous anti-D therapy in Rh-positive women with ITP effectively increased maternal platelet count without significantly affecting neonatal platelet levels. This discrepancy suggests that while gestational and hypertensive thrombocytopenia may

not impact substantially neonatal platelet counts, immune-mediated thrombocytopenia poses a greater risk.[18]

In our study, most newborns had APGAR scores ≥7 at 1 and 5 min, indicating favourable neonatal outcomes. This aligns with the findings of Boehlen et al., who reported no significant complications in neonates born to thrombocytopenic mothers.[13] However, Sheiner et al. found that neonates born to thrombocytopenic mothers had higher rates of APGAR scores <7 at 5 min (OR = 6.3, 95% CI = 1.8–33.8, p = 0.001), suggesting an increased risk of neonatal distress in specific subgroups.10 Samoon also observed lower APGAR scores in neonates of thrombocytopenic mothers, especially in cases complicated by hypertensive disorders, preterm birth, or IUGR.[14] Our findings indicate that thrombocytopenia itself may not significantly impact neonatal APGAR scores unless associated with other maternal complications.

Sankaran and Robinson emphasised the need for multidisciplinary management of immune thrombocytopenia cases involving obstetricians, haematologists, and anaesthetists to ensure optimal maternal and neonatal outcomes.[19] Additionally, Borhany et al. suggested that while supportive care is sufficient for most cases, patients with platelet counts below 50,000/µL may require active treatment, including steroids or platelet transfusions.[12]

CONCLUSION

Moderate thrombocytopenia due to severe preeclampsia was prevalent in our study, primarily resulting from vascular endothelial ischaemia and platelet consumption. The severity of the disease is correlated with the underlying disease. Most affected women were young primigravida, with many requiring preterm delivery and caesarean section, leading to higher rates of PPH and adverse foetal outcomes, including stillbirth, TTN, and perinatal asphyxia. Early detection through blood pressure monitoring and haemogram assessment can help prevent complications. Notably, gestational thrombocytopenia had no adverse maternal or foetal effects, with platelet counts normalising postpartum. No significant correlation was found between the maternal and foetal platelet counts.

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