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Research Article

An Observational Study to Assess the Relationship between Retinopathy of Prematurity and Thrombocytopenia

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ABSTRACT
Objective: This study aimed to evaluate the relationship between thrombocytopenia (platelet count <150,000/µL) and the severity of Retinopathy of Prematurity (ROP) in preterm infants, focusing on disease progression and clinical outcomes.
Methods: A prospective observational study was conducted at SMS Medical College, Jaipur, involving 70 preterm infants (≤30 weeks gestation, birth weight ≤1500 g) stratified into thrombocytopenic (n=35) and non-thrombocytopenic (n=35) groups. ROP screening was performed using indirect ophthalmoscopy at baseline and follow-up visits (weeks 3, 6, 9, 12). ROP was classified per ET-ROP criteria (Type 1: treatment-required; Type 2: observation). Data on gestational age, birth weight, oxygen therapy, platelet counts, ROP stage/zone and plus disease were collected and analyzed statistically.
Results: Thrombocytopenic infants had significantly lower mean platelet counts (0.93 ± 0.35 vs. 2.34 ± 0.65 lakh/mm^3, p<0.001). Type 1 ROP was more prevalent in thrombocytopenic infants (14.29% vs. 0% at week 3, p=0.01), with earlier treatment requirements (14.29% vs. 0% by week 3, p=0.08). Thrombocytopenic infants exhibited rapid ROP progression, with Stage 1 peaking at week 6 (40%) and resolving by week 12 (p<0.0001). Zone 3 involvement increased significantly in thrombocytopenic infants by week 12 (85.71% vs. 34.29% at baseline, p<0.0001). Plus disease was transient and more frequent in thrombocytopenic infants (5.71% vs. 0% at week 3, p=0.53).
Conclusion: Thrombocytopenia is associated with more severe and rapidly progressive ROP, necessitating earlier interventions. These findings highlight the potential role of platelet counts in risk stratification and optimizing screening protocols for preterm infants. Further research is needed to elucidate the underlying mechanisms and refine clinical management strategies.

Keywords: Thrombocytopenia, Retinopathy of Prematurity, Infants.

INTRODUCTION

Retinopathy of Prematurity (ROP) is a vasoproliferative disorder of the developing retina and a leading cause of preventable childhood blindness worldwide, particularly in preterm infants with very low birth weight (VLBW) and extreme prematurity.[1] The pathogenesis of ROP is complex and multifactorial, involving abnormal retinal vascular development due to premature exposure to extrauterine conditions, fluctuations in oxygen levels, and dysregulated angiogenic signaling.[2] Despite advances in neonatal care, ROP remains a significant clinical challenge, with severe cases progressing to retinal detachment and irreversible vision loss if not detected and treated promptly.[3]

Traditionally, the primary risk factors for ROP include low gestational age (GA), low birth weight (BW), prolonged oxygen therapy, and systemic comorbidities such as sepsis and intraventricular hemorrhage.[4] However, emerging evidence suggests that hematological abnormalities, particularly thrombocytopenia (platelet count <150,000/µL), may play a crucial role in ROP pathogenesis.[5] Platelets are key regulators of angiogenesis, storing and releasing vascular endothelial growth factor (VEGF) and other mediators that influence retinal vascular development.[6] Thrombocytopenia may disrupt VEGF homeostasis, leading to uncontrolled neovascularization and worsening ROP severity.[7]

Recent studies have demonstrated an association between thrombocytopenia and severe ROP requiring treatment. For instance, Almutairi et al.[8] reported that thrombocytopenic

preterm infants had a 2.38-fold increased risk of developing Type 1 ROP, with the risk escalating to 9-fold in cases involving Zone 1 disease. Similarly, Jensen et al.[9] found that early thrombocytopenia (within the first two postnatal weeks) was a significant predictor of ROP progression, suggesting that platelet levels during critical developmental phases may influence disease outcomes.

Despite these findings, the exact mechanisms linking thrombocytopenia to ROP remain unclear, and clinical data on platelet thresholds, timing of thrombocytopenia, and its impact on treatment response are limited.[10] this study aimed to evaluate the relationship between thrombocytopenia and ROP severity in preterm infants at SMS Medical College, Jaipur, by assessing platelet counts, ROP progression, and treatment requirements. By elucidating this association, we hope to improve risk stratification, optimize screening protocols, and guide therapeutic decisions to mitigate vision-threatening complications in this vulnerable population.

MATERIALS AND METHOD

Study Design: Prospective observational study.

Setting: Departments of Ophthalmology and Paediatrics, SMS Medical College, Jaipur.

Inclusion Criteria

- Preterm infants (≤30 week's gestation, birth weight ≤1500 g) deemed at risk for ROP (e.g., oxygen therapy, sepsis).

Exclusion Criteria

- Lack of final ROP outcome documentation or death before ophthalmic examination.

Sample Size: 35 neonates per group (thrombocytopenic/non-thrombocytopenic), accounting for 10% dropout.

Grouping

Neonates stratified by platelet count at birth/first visit:

- Thrombocytopenic (<1.50 lakh/mm³).
- Non-thrombocytopenic (≥1.50 lakh/mm³).

ROP Screening

Fundus examination using indirect ophthalmoscopy (2.2D lens) at baseline and follow-ups (3, 6, 9, 12 weeks).

Classification

ROP staged per ET-ROP Criteria

- **Type 1 (Treatment-required):** Zone I any stage with plus disease, Zone I stage 3, or Zone II stage 2/3 with plus disease.
- **Type 2 (Observation):** Zone II stage 1/2 without plus disease.

Data Collection

Gestational age, birth weight, oxygen therapy duration, platelet counts, ROP zone/stage and plus disease.

Statistical Analysis

Mean ± SD, percentages, and significance testing ($p < 0.05$ considered significant).

RESULTS

Table 1. Demographic and Clinical Characteristics Comparison

Parameter	Thrombocytopenic (n=35)	Non-Thrombocytopenic (n=35)	p-value
Gestational Age			
≤27 weeks	4 (11.43%)	3 (8.57%)	0.29
>27 weeks	31 (88.57%)	32 (91.43%)	
Mean ± SD (weeks)	28.94 ± 1.21	29.22 ± 1.00	
Gender			
Male	20 (57.14%)	17 (48.57%)	0.47
Female	15 (42.86%)	18 (51.43%)	
Postnatal Age			
8-28 days	13 (37.14%)	17 (48.57%)	0.5
29-90 days	22 (62.86%)	18 (51.43%)	
Mean ± SD (days)	32.17 ± 12.17	35.31 ± 25.24	
Birth Weight			
≤1000g	13 (37.14%)	4 (11.43%)	0.14
1001-1500g	21 (60.00%)	30 (85.71%)	
>1500g	1 (2.86%)	1 (2.86%)	
Mean ± SD (grams)	1174.14 ± 259.03	1253.91 ± 184.50	
Oxygen Therapy			
1-7 days	11 (31.43%)	15 (42.86%)	0.32
8-14 days	16 (45.71%)	15 (42.86%)	

15-21 days	8 (22.86%)	5 (14.29%)	
Mean ± SD (days)	10.2 ± 5.31	8.97 ± 5.17	

The study compared baseline characteristics between thrombocytopenic (n=35) and non-thrombocytopenic (n=35) neonates. Gestational age distribution was similar between groups (Table 1), with 11.43% of thrombocytopenic versus 8.57% of non-thrombocytopenic infants born ≤27 weeks (mean GA 28.94±1.21 vs 29.22±1.00 weeks, p=0.29). Gender distribution (Table 2) showed a slight male predominance in thrombocytopenic infants (57.14% vs 48.57%), though not statistically significant (p=0.47). Postnatal age at evaluation (Table 3) revealed 62.86% of thrombocytopenic infants were assessed between 29-90 days compared to 51.43% of controls, with comparable mean ages (32.17±12.17 vs 35.31±25.24 days, p=0.5). Birth weight distribution (Table 4) showed thrombocytopenic infants were more likely to be ≤1000g (37.14% vs 11.43%) with a lower mean weight (1174.14±259.03g vs 1253.91±184.50g, p=0.14). Oxygen therapy duration (Table 5) was longer in thrombocytopenic infants (mean 10.2±5.31 vs 8.97±5.17 days), with 22.86% requiring 15-21 days of support versus 14.29% of controls (p=0.32). While none of these baseline differences reached statistical significance, thrombocytopenic infants consistently trended toward higher-risk profiles across all demographic and clinical parameters.

Table 2. Comparison of Mean Platelet Count between Groups

Group	Mean Platelet Count (lakh/mm³) ± SD	p-value
Thrombocytopenic	0.93 ± 0.35	**<0.001**
Non-thrombocytopenic	2.34 ± 0.65	

The thrombocytopenic group demonstrated significantly lower mean platelet counts (0.93 ± 0.35 lakh/mm³) compared to non-thrombocytopenic infants (2.34 ± 0.65 lakh/mm³), with this difference being highly statistically significant ($p < 0.001$). This clear separation in platelet levels between the two groups validated our study's group stratification and established the foundation for subsequent ROP comparisons.

Table 3. Type of ROP at Follow-Up Visits

Visit	Type 1 ROP (Thrombocytopenic)	Type 1 ROP (Non-thrombocytopenic)	p-value
Week 1	2.86%	0%	**0.01**
Week 3	14.29%	0%	
Week 6	14.29%	5.71%	

Thrombocytopenic neonates showed markedly higher rates of Type 1 (treatment-requiring) ROP throughout follow-up. While only 2.86% of thrombocytopenic infants had Type 1 ROP at week 1, this increased to 14.29% by week 3 and remained stable thereafter. In contrast, non-thrombocytopenic infants had no Type 1 cases until week 6 (5.71%). The between-group differences were statistically significant ($p = 0.01$), highlighting thrombocytopenia's association with more severe ROP forms.

Table 4. Treatment Requirements

Visit	Treatment Required (Thrombocytopenic)	Treatment Required (Non-thrombocytopenic)	p-value
Week 1	2.86%	0%	0.9
Week 3	14.29%	0%	0.08
Week 9	11.43% (Laser)	5.71% (Laser)	0.39

Treatment needs differed notably between groups. Thrombocytopenic infants required earlier interventions, with 14.29% needing treatment by week 3 compared to 0% in controls. Anti-VEGF therapy was administered more frequently and earlier in thrombocytopenic cases, while laser treatment appeared comparable between groups by week 9 (11.43% vs 5.71%). Though some time points showed borderline significance ($p = 0.08$

at week 3), the overall trend suggested thrombocytopenic infants required more aggressive management.

Table 5. Retinal Zone Progression (Thrombocytopenic Group)

Visit	Zone 1 (%)	Zone 2 (%)	Zone 3 (%)	p-value
Week 1	2.86	62.86	34.29	**<0.0001**
Week 12	0	14.29	85.71	

The thrombocytopenic group exhibited a distinct pattern of retinal involvement progression. Initial examinations showed 62.86% Zone 2 and 34.29% Zone 3 involvement. By week 12, Zone 3 predominated (85.71%) while Zone 1 completely resolved. This significant shift ($p < 0.0001$) suggested thrombocytopenia might influence the spatial progression of ROP, with disease migrating peripherally over time.

Table 6. Stage-Wise ROP Progression

Stage	Week 1 (%)	Week 6 (%)	Week 12 (%)	p-value
Stage 1	14.29	40.00	0	**<0.0001**
Stage 2	51.43	14.29	0	

Stage-wise analysis revealed dynamic changes in thrombocytopenic infants. Stage 1 ROP increased from 14.29% at baseline to 40% by week 6 before resolving, while Stage 2 decreased from 51.43% to 0% by week 12 ($p < 0.0001$). This rapid escalation and subsequent resolution pattern differed from the more stable course in non-thrombocytopenic infants.

Table 7. Plus Disease Trends

Visit	Plus Disease (Thrombocytopenic)	Plus Disease (Non-thrombocytopenic)	p-value
Week 1	2.86%	0%	0.9
Week 3	5.71%	0%	0.53

Plus disease occurrence was uncommon but showed a trend toward higher prevalence in thrombocytopenic infants (5.71% at week 3 vs 0% in controls). However, these differences did not reach statistical significance ($p = 0.53$), possibly due to low event rates. All cases resolved by week 6, suggesting plus disease in thrombocytopenic infants, when present, may be transient.

DISCUSSION

Understanding the role of thrombocytopenia in ROP could have clinical implications, including improved risk stratification and timing of intervention. This work aims on an observational study to assess the relationship between retinopathy of prematurity and thrombocytopenia.

Thrombocytopenic and non-thrombocytopenic groups had comparable gestational ages (28.94 vs. 29.22 weeks, $p=0.29$), with marginally more thrombocytopenic infants born ≤27 weeks (11.43% vs. 8.57%). This aligns with studies showing ROP severity inversely correlates with gestational age.[11,12] Notably, Borroni et al.[13] reported 75% of ROP cases occurred in infants born ≤24 weeks (OR 2.01, $p=0.008$), while Cao et al.[14] found no GA differences in sepsis-matched cohorts (27.0 vs. 26.5 weeks, $p=0.335$).

The study found no significant gender-based differences in ROP risk between thrombocytopenic and non-thrombocytopenic neonates. While males showed a slight predominance in both groups (57.1% in thrombocytopenic vs. 48.6% in non-thrombocytopenic, $p=0.47$), this difference was not statistically significant. These findings are consistent with previous research showing similar non-significant trends, including a gradual increase in male representation with ROP severity (50% in no-ROP to 64% in type 1 ROP, $p=0.72$),[15] comparable gender distribution between ROP and non-ROP cases (45% vs 46% males, OR=0.96, $p=0.85$),[16] and non-significant patterns in sepsis cohorts (52.9% vs 37.3% males post-matching, $p=0.11$).[17] Collectively, these results suggest that gender does not appear to be a significant factor in

ROP development or progression among preterm infants.

The study found comparable postnatal age distributions between thrombocytopenic (mean 32.2 days) and non-thrombocytopenic neonates (mean 35.3 days, p=0.5). However, research suggests the timing of thrombocytopenia significantly impacts ROP progression. Korkmaz et al.[18] identified postnatal days 29-45 (PMA 30-35 weeks) as a critical period where thrombocytopenia predicts Type 1 ROP/APROP development. Cakir et al.[19] further demonstrated thrombocytopenia during days 21-35 increases severe ROP risk (aOR 2.97, p=0.006), with early-onset cases (weeks 2-4) showing strongest associations. These findings align with Seliniotaki et al.[20] observation that ROP-affected infants require extended follow-up (9.56 vs 8.08 weeks, p=0.003), suggesting platelet monitoring during specific postnatal windows may improve risk prediction.

Thrombocytopenic neonates showed a higher proportion of extremely low birth weight infants (≤1000g: 37.1% vs 11.4%) and lower mean birth weight (1174g vs 1254g, p=0.14). This aligns with established evidence linking lower birth weights to ROP severity. Ling et al.[21] demonstrated significantly decreasing birth weights across ROP severity groups (1163g in no-ROP vs 846g in type 1 ROP, p=0.002), while Borroni et al.[13] found 68% of ROP cases occurred in infants ≤750g (OR 1.73, p=0.007). Notably, Cao et al.[14] reported birth weight didn't influence sepsis outcomes (976g vs 1005g post-matching, p=0.403), suggesting its specific relevance for ROP risk stratification rather than general neonatal outcomes.

Thrombocytopenic neonates showed non-significantly longer oxygen therapy durations (10.2 vs 8.97 days, p=0.32), with nearly 60% more requiring extended support (22.9% vs 14.3% for 15-21 days). This trend aligns with established ROP risk literature demonstrating significantly prolonged oxygen requirements in affected infants (14.4 vs 2.7 days, p<0.001)[22] and particularly in progressive cases (68.3 vs 29.8 days, p=0.0001).[23] However, the relationship appears confounded by comorbidities, as evidenced by reduced significance in matched cohorts (35 vs 23 days, p=0.12).[24] These findings collectively reinforce oxygen duration as a modifiable ROP risk factor while emphasizing the need for comprehensive clinical assessment.

The study revealed significantly lower platelet counts in thrombocytopenic neonates (0.93 vs 2.34 lakh, p<0.001). While some studies report similar reductions in ROP cases (218.34 vs 300.72, p=0.001),[25] these differences often become non-significant after adjusting for comorbidities (p=0.689).[26] Notably, Rastogi et al.[27] found platelet decline (>30%) more predictive than absolute counts (p<0.001), while Jensen et al.[9] identified severe thrombocytopenia as particularly relevant for aggressive zone 1 ROP (OR 6.69). These findings suggest platelet dynamics may be more clinically informative than isolated measurements for ROP risk assessment.

Initial examination revealed Zone 2 as the most commonly affected area in both groups (62.9% thrombocytopenic vs 80.0% non-thrombocytopenic), while thrombocytopenic neonates showed greater early Zone 3 involvement (34.3% vs 17.1%). Both groups demonstrated progressive peripheral migration, with Zone 3 predominating by week 12 (85.7% vs 94.3%, p>0.05). These findings align with existing literature showing: Stronger associations between thrombocytopenia and Zone 1 ROP (OR 9.00) versus Zone 2 disease.[9] Higher thrombocytopenia prevalence in posterior Zone I cases (83.3% vs 64.7%, p=0.073).[28] Characteristic peripheral disease progression (Zone 2: -77.3%; Zone 3: +149.9%).[29]

Thrombocytopenic neonates demonstrated more rapid ROP progression, with Stage 3 appearing by week 3 exclusively in this group. Stage 1 disease peaked earlier (40% at week 6, +179.97%) followed by complete resolution by week 12 (p<0.0001). These findings align with Jensen et al.[9] observations that early thrombocytopenia (24-34 weeks PMA) significantly predicts Type 1 ROP development (100% vs 21-44% in controls, p=0.001-0.10), while Hengartner et al.[30] reported most cases stabilize at moderate stages (Stage 2:51.1%, Stage 3:47.8%).

Plus disease showed transient occurrence in both groups, appearing in 2.86-5.71% of cases with no significant between-group differences (p>0.05). While thrombocytopenic infants demonstrated earlier plus disease presentation (week 3 vs week 6 in controls), all cases resolved by week 9. This clinical pattern aligns with research demonstrating: Strong associations between severe thrombocytopenia (~90×10^9/L) and plus disease/AP-ROP (p<0.001).[31] The predictive value of platelet indices (e.g., PDW) over absolute counts for severe ROP (p<0.05).[32] Frequent

thrombocytopenia ($<100\times10^9$/L) in AP-ROP cases (>80%).[33]

CONCLUSION

The study demonstrates a significant association between thrombocytopenia and severe, rapidly progressive ROP in preterm infants, with thrombocytopenic infants exhibiting higher rates of Type 1 ROP, earlier treatment needs, and distinct disease progression patterns. These findings suggest that platelet counts may serve as a valuable biomarker for risk stratification, enabling timely interventions to mitigate vision-threatening complications. Future research should explore the mechanistic links between thrombocytopenia and ROP pathogenesis to refine clinical guidelines and improve outcomes in this vulnerable population.

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