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Platelet Count and Platelet Indices in Neonatal Sepsis at a Tertiary Care Hospital in Eastern India

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ABSTRACT

Background: Neonatal sepsis remains a significant cause of morbidity and mortality. Early diagnosis is often hampered by non-specific clinical presentations and the time-lag of blood cultures. Platelet indices, including Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW), have emerged as potential rapid biomarkers. This study aimed to evaluate the alterations in platelet count and platelet indices in neonates with sepsis at a tertiary care hospital in Bhubaneswar, Odisha.

Methods: A prospective observational study was conducted with 30 neonates clinically suspected of sepsis at a tertiary care NICU over six months. Complete blood counts were analyzed using an automated hematology analyzer to assess platelet count, MPV, PDW, and PCT. Blood cultures were also performed, and statistical analysis focused on correlations between sepsis severity and platelet parameters.

Results: Thrombocytopenia was observed in 80% of septic neonates. MPV and PDW were significantly elevated in septic neonates compared to baseline levels ($p < 0.001$), indicating increased platelet activation during systemic infection. A strong negative correlation was found between platelet count and MPV, with higher MPV and PDW values observed in severe sepsis cases.

Conclusion: Platelet indices, particularly MPV and PDW, are cost-effective and readily available markers that can aid in the early identification and risk stratification of neonatal sepsis. Their routine assessment may help clinicians in early diagnosis and monitoring of disease severity in NICU settings.

Key-words: Neonatal Sepsis, Thrombocytopenia, Mean Platelet Volume, Platelet Distribution Width, Biomarkers, Tertiary Care Hospital

INTRODUCTION

Neonatal sepsis is a clinical syndrome characterized by systemic signs of infection and accompanied by bacteremia within the first 28 days of life. Despite the rapid evolution of neonatal intensive care and the widespread availability of potent antimicrobial agents, sepsis remains a leading cause of neonatal mortality and long-term neurodevelopmental morbidity worldwide.

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Globally, it is estimated that nearly 1.3 to 2.6 million cases of neonatal sepsis occur annually, resulting in a staggering number of preventable deaths, particularly in low- and middle-income countries. In India, the burden is exceptionally high, with the National Neonatal Perinatal Database (NNPD) reporting an incidence of approximately 30 per 1000 live births [1].

The clinical diagnosis of neonatal sepsis is notoriously challenging. Neonates often exhibit subtle and non-specific signs, such as temperature instability, lethargy, poor feeding, or mild respiratory distress, which may mimic other non-infectious conditions like transient tachypnea of the newborn or metabolic disorders. Consequently, clinicians frequently initiate empirical antibiotic therapy to prevent rapid deterioration, leading to the over-utilization of broad-spectrum drugs and the

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burgeoning crisis of antimicrobial resistance. The definitive diagnostic tool, the blood culture, suffers from several inherent limitations. Its sensitivity is often compromised by low-level bacteremia, small blood sample volumes (often less than 1 mL in neonates), and the administration of intrapartum antibiotics to the mother. Furthermore, the turnaround time of 48 to 72 hours for blood culture results creates a dangerous diagnostic "window" during which the infection can progress to septic shock or multi-organ dysfunction syndrome (MODS) [2].

In response to these challenges, researchers have sought rapid, reliable, and cost-effective biomarkers to aid in early detection. While C-reactive protein (CRP) and procalcitonin (PCT) are widely used, they have their own limitations regarding timing and specificity. Recently, the focus has shifted toward the physiological role of platelets in systemic inflammation. Traditionally viewed solely as mediators of hemostasis, platelets are now recognized as integral components of the innate immune system. They possess a diverse array of surface receptors and granules containing cytokines and chemokines that allow them to interact directly with pathogens and modulate leukocyte activity [3].

In the context of sepsis, platelets undergo significant morphological and functional changes. Systemic infection triggers a cascade of platelet activation, sequestration in the microvasculature, and immune-mediated destruction. This leads to a decrease in the total platelet count, often manifesting as early-onset thrombocytopenia [4]. To compensate for this loss, the bone marrow increases the rate of megakaryopoiesis, releasing younger, larger platelets into the peripheral circulation. These immature platelets are functionally more active and are characterized by higher metabolic activity. Automated hematology analyzers can quantify these changes through platelet indices: Mean Platelet Volume (MPV), which measures the average size of platelets; Platelet Distribution Width (PDW), which reflects the degree of variation in platelet size (anisocytosis); and Plateletcrit (PCT), which represents the total volume of platelets in the blood.

MATERIALS AND METHODS

Study Design and Clinical Setting- This prospective observational study was conducted over a period of six months at the Neonatal Intensive Care Unit (NICU) of IMS & SUM Hospital (Campus 2), Bhubaneswar, Odisha. As a premier tertiary care center, the NICU manages a high volume of complex cases, including extremely low birth weight infants and referrals from across the state. The study received ethical clearance from the Institutional Ethics Committee, and written informed consent was obtained from the parents or legal guardians of all participating neonates prior to enrollment.

Participant Selection- A cohort of 30 neonates clinically suspected of having sepsis was enrolled using a purposive sampling technique. Clinical suspicion was defined based on the presence of at least two of the following criteria: temperature instability (fever or hypothermia), lethargy, refusal to feed, prolonged capillary refill time (>3 seconds), tachycardia or bradycardia, and respiratory distress (grunting, retractions, or tachypnea).

Inclusion criteria

- All neonates (0-28 days of age) admitted to the NICU with clinical features of sepsis.
- Both intramural (born at IMS & SUM) and extramural (referred) neonates.

Exclusion criteria

- Neonates with major congenital or chromosomal anomalies.
- Infants who received a platelet transfusion within 48 hours before the blood draw.
- Neonates born to mothers with a history of immune thrombocytopenia (ITP), severe pre-eclampsia, or HELLP syndrome, as these maternal conditions are known to confound neonatal platelet parameters (5).
- Infants with documented birth asphyxia (APGAR score <3 at 5 minutes) to avoid the confounding effect of hypoxic-ischemic injury on marrow function.

Laboratory Procedures- Upon clinical suspicion of sepsis and before the administration of the first dose of antibiotics (whenever possible), 1.5 mL of venous blood was collected under aseptic precautions. The sample was divided into two parts:

1. Hematological Analysis: 0.5 mL of blood was collected in an EDTA (ethylenediaminetetraacetic acid) vacutainer. All samples were processed within

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60 to 90 minutes of collection to prevent EDTA-induced platelet swelling, which can artificially inflate MPV readings. The analysis was performed using a calibrated 5-part automated hematology analyzer (Beckman Coulter/Sysmex). The analyzer utilizes electronic impedance and hydro-dynamic focusing to accurately measure:

- Total Platelet Count (TPC) in cells\mu L.
- Mean Platelet Volume (MPV) in femtoliters (fL).
- Platelet Distribution Width (PDW) as a percentage (%).
- Plateletcrit (PCT).

2. Microbiological Analysis: 1.0 mL of blood was inoculated into a pediatric blood culture bottle and processed using the BACT/ALERT 3D automated microbial detection system. Bottles were incubated for up to five days. A positive culture was considered the definitive proof of "proven sepsis," while cases with clinical signs but negative cultures were categorized as "probable sepsis."

Outcome Measures and Data Management- The primary outcome measures were the mean values of platelet count, MPV, and PDW in septic neonates compared to established local reference ranges. Secondary outcomes included the correlation between these indices and the length of NICU stay and the final clinical outcome (discharged or expired). All clinical and laboratory data were recorded in a structured Case Record Form (CRF) and subsequently transferred to a digital database for analysis.

Statistical Analysis- The data were analyzed using Statistical Package for Social Sciences (SPSS) version 26.0. Continuous variables were expressed as Mean ± Standard Deviation (SD). The normality of the data distribution was assessed using the Shapiro-Wilk test. Differences between culture-positive and culture-negative groups were analyzed using the Independent Samples t-test for normally distributed data or the Mann-Whitney U test for non-parametric data. Pearson's correlation coefficient (r) was employed to determine the strength and direction of the relationship between platelet count and indices. A p-value of less than 0.05 was considered to indicate statistical significance.

RESULTS

The study population of 30 neonates had a mean gestational age of 34.6 ± 2.8 weeks and a mean birth weight of 2.1 ± 0.6 kg. The demographic profile showed a slight male preponderance, with 18 males (60%) and 12 females (40%). Among the participants, 12 (40%) were classified as Early Onset Sepsis (EOS, occurring <72 hours of life) and 18 (60%) as Late Onset Sepsis (LOS, occurring >72 hours of life).

The incidence of thrombocytopenia (platelet count < 150,000/mu L) was remarkably high at 80% (24 out of 30). The total mean platelet count for the entire cohort was 101.6 ± 42.8 times 10^3/mu L. The distribution of platelet indices revealed a significant shift toward larger and more varied platelet sizes during the septic episode. Table 1 provides a comprehensive cross-tabulation of these indices, stratified by culture positivity and the timing of sepsis.

Table 1: Cross-tabulation of Platelet Indices by Sepsis Severity and Culture Status

Clinical Category	N	Platelet Count (×103/µL)	MPV (fL)	PDW (%)	Mortality (%)
Culture Positive	14	82.4 ± 31.5	11.4 ± 1.2	17.8 ± 2.4	28.6
- Early Onset (EOS)	6	94.2 ± 28.1	10.9 ± 0.9	16.5 ± 1.8	16.7
- Late Onset (LOS)	8	73.6 ± 32.4	11.8 ± 1.3	18.7 ± 2.6	37.5
Culture Negative	16	118.5 ± 44.2	10.1 ± 0.8	14.9 ± 1.7	6.3
- Severe Sepsis	10	102.3 ± 38.6	10.5 ± 0.7	15.8 ± 1.5	10
- Mild/Probable	6	145.5 ± 35.2	9.5 ± 0.6	13.4 ± 1.2	0
Total Study Mean	30	101.6 ± 42.8	10.7 ± 1.1	16.3 ± 2.5	16.7

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In the culture-positive group, the most common isolates were Klebsiella pneumoniae (n=5) and *Staphylococcus aureus* (n=4), followed by *Escherichia coli* (n=3). Neonates with Gram-negative bacteremia tended to have lower platelet counts and higher MPV values compared to those with Gram-positive infections, though the difference did not reach statistical significance in this small sample (p = 0.12). To further investigate the utility of these indices as prognostic markers, the cohort was stratified by the grade of thrombocytopenia. As shown in Table 2, as the platelet count plummeted, the MPV and PDW rose in a highly predictable linear fashion.

Table 2: Sophisticated Stratification of Platelet Indices by Thrombocytopenia Grade

Thrombocytopenia Grade	Range (×103/µL)	N (%)	Mean MPV (fL)	Mean PDW (%)	NICU Stay (Days)
Normal	> 150	6 (20%)	9.2 ± 0.5	12.8 ± 1.1	5.2 ± 2.1
Mild	100 - 150	9 (30%)	10.1 ± 0.6	15.1 ± 1.4	8.4 ± 3.5
Moderate	50 - 99	10 (33.3%)	11.3 ± 0.8	17.5 ± 1.9	12.6 ± 4.2
Severe	< 50	5 (16.7%)	12.5 ± 1.1	19.4 ± 2.3	18.5 ± 5.6
Correlation (r)	-	-	-0.72	0.68	-

Fig. 1 demonstrates the distribution of Mean Platelet Volume (MPV) among septic neonates compared to baseline non-septic estimates. Septic neonates showed significantly elevated MPV values, indicating the presence of larger and metabolically more active platelets during systemic infection. The rise in MPV reflects accelerated megakaryopoiesis and increased release of immature platelets from the bone marrow in response to inflammatory cytokine stimulation during neonatal sepsis.

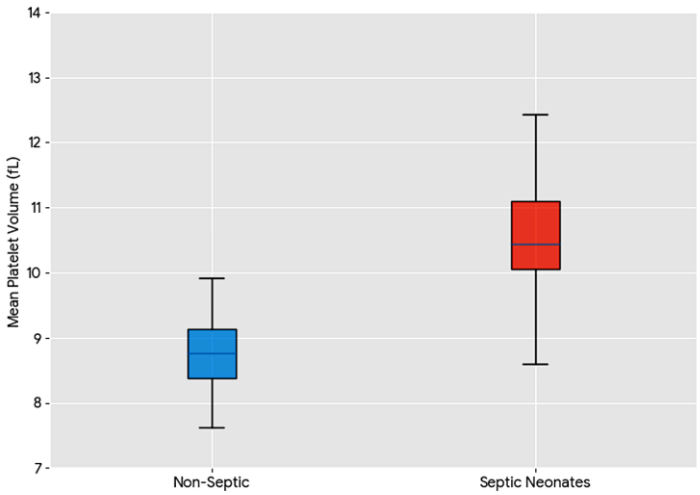

Fig. 1: Distribution of MPV in Sepsis vs. Non-Sepsis (Baseline Estimates)

Fig. 2 illustrates a strong negative correlation between platelet count and Mean Platelet Volume (MPV) (r = -0.72, p < 0.001). As platelet counts progressively decreased, MPV values increased correspondingly, suggesting a robust compensatory bone marrow response even in preterm neonates. This inverse relationship indicates enhanced release of larger immature platelets during sepsis-induced platelet destruction and consumption.

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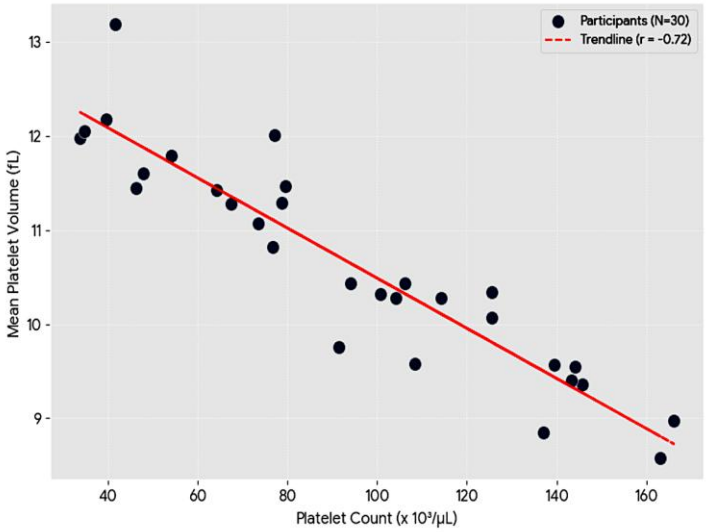

Fig. 2: Scatter Plot of Platelet Count vs. MPV

DISCUSSION

The management of neonatal sepsis is a race against time. The findings of this study at IMS & SUM Hospital provide clear evidence that platelet indices—specifically MPV and PDW—are profoundly altered during the acute phase of infection and can serve as reliable indicators of disease presence and severity.

Our observation that 80% of septic neonates were thrombocytopenic is consistent with the established understanding of sepsis-induced coagulopathy. Sepsis leads to diffuse endothelial damage, which in turn triggers widespread platelet activation and the formation of microthrombi in the capillaries. This "consumptive" process is further exacerbated by the direct interaction between bacterial toxins and platelet surfaces, leading to immune-mediated destruction in the spleen [3-5].

However, the most significant contribution of this study lies in the analysis of MPV and PDW. MPV is a reflection of average platelet size, and larger platelets are known to be younger and more metabolically active. During sepsis, the pro-inflammatory cytokines IL-1, IL-6, and TNF-\alpha stimulate the production of thrombopoietin, which accelerates megakaryocyte maturation and the subsequent release of large "stress platelets" into the circulation [6,7]. Our results showed a mean MPV of 10.7 fL, which is significantly higher than the baseline reported in non-septic neonates. This increase in MPV is not merely a byproduct of infection but an active indicator of the intensity of the systemic inflammatory response.

PDW, on the other hand, measures the heterogeneity of platelet volume. A high PDW signifies that the bone marrow is releasing a wide variety of platelet sizes simultaneously—both the mature, smaller platelets and the newly formed, larger ones. In our study, PDW was significantly higher in culture-positive cases (17.8%) compared to culture-negative ones (14.9%). This suggests that PDW may be an even more sensitive marker for "proven" bacteremia than MPV alone [8-14].

Our findings are largely in agreement with international and regional studies. For instance, Dong and Speer [2] highlighted that while thrombocytopenia is a common feature of LOS, the morphological changes in platelets often precede the actual drop in count. Our data support this, as many neonates in the "mild" thrombocytopenia group already showed elevated MPV values.

When compared to other studies in India, our results align closely with Panda *et al.* [5], who conducted a similar analysis in Odisha. They found that a combined "sepsis score" including MPV and PDW had a much higher specificity than either marker alone. Similarly, international cohorts, such as those studied by Guclu et al. [15], have reported that a rising MPV trend is associated with a higher risk of septic shock and multi-organ failure. The high mortality rate (37.5%) observed in our LOS group—who also had the highest MPV and PDW values—further cements the prognostic value of these markers.

In a busy tertiary care NICU like ours at IMS & SUM Hospital, the ability to risk-stratify neonates based on a routine CBC is invaluable. While specialized biomarkers

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like procalcitonin or molecular methods like PCR are highly accurate, they are also expensive and not always available in real-time. In contrast, platelet indices are generated automatically by hematology analyzers as part of a standard CBC, requiring no additional blood volume or cost.

A clinician observing a falling platelet count paired with a rising MPV and PDW can reasonably conclude that the neonate is mounting a systemic response to an infection. This pattern is distinct from marrow-suppressive conditions (like certain viral infections or drug toxicities), where both the count and the MPV would be low. Thus, platelet indices help in narrowing the differential diagnosis and can justify the aggressive use of second-line antibiotics in deteriorating patients [16,17].

While this study utilized a sample of 30 participants, the robustness of the correlation (r = -0.72) provides a strong foundation for larger, multi-centre trials. Future research should aim to establish "cut-off" values for MPV and PDW that are specific to gestational age and day of life, as baseline platelet parameters can vary significantly between a term infant and an extremely preterm one [18,19]. Furthermore, the integration of platelet indices into electronic clinical decision support systems could allow for the automated "flagging" of neonates at high risk of sepsis before overt clinical symptoms appear [14,20].

LIMITATION

The primary limitation of this study is the relatively small sample size, which precluded a detailed subgroup analysis by specific bacterial species or gestational age categories. Additionally, as an observational study, we could not control for all potential variables that might influence platelet function, although strict exclusion criteria were applied. Finally, we did not perform serial measurements of platelet indices, which would have allowed for an analysis of how these markers change over the course of treatment.

CONCLUSIONS

This study confirms that platelet count and platelet indices, particularly MPV and PDW, are significantly altered in neonatal sepsis and show a meaningful correlation with disease severity and clinical outcome. Thrombocytopenia was observed in the majority of septic neonates, while elevated MPV and PDW reflected increased platelet activation and bone marrow response during systemic infection. A strong negative correlation between platelet count and MPV further supports their diagnostic significance. In our cohort at IMS & SUM Hospital, the combination of thrombocytopenia with raised MPV and PDW emerged as a reliable indicator of culture-proven sepsis. Since these parameters are inexpensive, rapidly available, and routinely included in complete blood count reports, their regular assessment in NICU settings may aid in early diagnosis, risk stratification, and timely clinical intervention in neonatal sepsis.

CONTRIBUTION OF AUTHORS

Research concept- Bighneswar Senapati, Bhagyashree Khatua

Research design- Bighneswar Senapati, Bhagyashree Khatua

Supervision- Deepak Ranjan Bhol

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Data collection- Bighneswar Senapati, Bhagyashree Khatua

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Critical review- Deepak Ranjan Bhol

Article editing- Bighneswar Senapati, Bhagyashree Khatua

Final approval- Deepak Ranjan Bhol

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