



A STUDY OF PLATELET COUNTS IN PROVEN NEONATAL SEPSIS AND ASSOCIATION WITH ETIOLOGICAL PROFILE IN A TERTIARY CARE NICU, JAIPUR

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Abstract

Background: Neonatal sepsis remains a major cause of neonatal morbidity and mortality, particularly in developing countries. Early diagnosis is challenging because of nonspecific clinical manifestations and the limited sensitivity of blood culture, which remains the gold standard diagnostic test. Thrombocytopenia is one of the most common hematological abnormalities observed in neonatal sepsis and may serve as a useful marker for early diagnosis and disease severity. This study was conducted to evaluate platelet counts in culture-proven neonatal sepsis and determine their association with the etiological profile of infecting organisms.

Methods: A prospective observational study was conducted in the Neonatal Intensive Care Unit (NICU) of Mahatma Gandhi Medical College and Hospital, Jaipur. Neonates with clinical suspicion of sepsis underwent blood culture and hematological evaluation. Only culture-positive cases were included in the study. Demographic characteristics, risk factors, clinical features, platelet counts, and microbiological profiles were recorded and analyzed.

Results: Among 1,489 NICU admissions, 440 neonates were clinically suspected of sepsis, of whom 102 (23.18%) had culture-proven sepsis. Early-onset sepsis accounted for the majority of cases (69.61%), and male neonates constituted 63.73% of affected infants. Moderate-to-severe thrombocytopenia was significantly more common among culture-positive neonates than culture-negative neonates (OR = 90.23; 95% CI: 39.9–204.2; $p < 0.0001$).

Conclusion: Thrombocytopenia is a common hematological abnormality in neonatal sepsis and shows a strong association with culture-proven infection. Moderate-to-severe thrombocytopenia may serve as a useful adjunctive marker for the early diagnosis and prognostic assessment of neonatal sepsis. Although Gram-negative organisms were associated with greater platelet depletion, platelet count alone could not reliably predict the causative pathogen. Routine monitoring of platelet counts may aid in the early identification of high-risk neonates and facilitate timely therapeutic intervention.

Keywords: Neonatal sepsis, thrombocytopenia, platelet count, blood culture, *Klebsiella pneumoniae*.

Introduction

Neonatal sepsis remains one of the most important causes of neonatal morbidity and mortality worldwide, particularly in developing countries. It is defined as a systemic infection caused by bacterial, viral, or fungal pathogens occurring during the neonatal period and is associated with significant hemodynamic disturbances and multisystem involvement. Despite considerable advances in neonatal intensive care, antimicrobial therapy, and infection control practices, neonatal sepsis continues to pose a major challenge because of its nonspecific clinical presentation and high fatality rates. Globally, neonatal sepsis accounts for a substantial proportion of neonatal deaths, with an estimated 1.3 million cases and more than 200,000 sepsis-related deaths reported annually.^[1]

India bears a disproportionately high burden of neonatal sepsis, with one of the highest incidences reported worldwide. The incidence of clinical neonatal sepsis in India has been estimated at approximately 17,000 per 100,000 live births, while mortality rates range from 25% to 65%.^[2] The Delhi Neonatal Infection Study (DeNIS), a landmark multicentric investigation conducted across tertiary neonatal intensive care units (NICUs) in India, highlighted the significant burden of neonatal infections and identified Gram-negative organisms such as *Klebsiella pneumoniae*, *Acinetobacter* species, and *Escherichia coli* as the predominant pathogens responsible for neonatal sepsis.^[3] The emergence of multidrug-resistant organisms in NICUs has further complicated the management of neonatal sepsis and contributed to adverse clinical outcomes.^[4]

Neonatal sepsis is broadly classified into early-onset sepsis (EOS) and late-onset sepsis (LOS) based on the timing of infection. Early-onset sepsis generally occurs within the first 72 hours of life and is primarily acquired through vertical transmission from the mother before or during delivery. Maternal risk factors such as prolonged rupture of membranes, chorioamnionitis, maternal fever, and genital tract colonization significantly increase the risk of EOS. Common causative organisms include Group B *Streptococcus* and *Escherichia coli*.^[5] In contrast, late-onset sepsis occurs after 72 hours of life and is commonly acquired from the hospital environment or community. Prematurity, prolonged hospitalization, invasive procedures, mechanical ventilation, and central venous catheterization are important risk factors for LOS. Organisms frequently implicated in LOS include coagulase-negative staphylococci, *Staphylococcus aureus*, *Klebsiella* species, *Pseudomonas aeruginosa*, and *Candida* species.^[4]

The diagnosis of neonatal sepsis remains challenging because the clinical manifestations are often subtle, nonspecific, and overlap with several noninfectious neonatal conditions. Common clinical features include respiratory distress, poor feeding, lethargy, apnea, temperature instability, abdominal distension, and circulatory compromise.^[6] Although blood culture remains the gold standard for diagnosing neonatal sepsis, its utility is limited by low sensitivity, prolonged turnaround time, and the possibility of false-negative results due to prior antibiotic exposure or inadequate blood sample volume. Culture positivity rates in neonatal sepsis have been reported to vary between 39% and 48%, while definitive results may take several days to become available.^[3] Consequently, there is a growing need for rapid, affordable, and readily available biomarkers that can facilitate early diagnosis and timely initiation of treatment.

Platelets are increasingly recognized as important mediators of immune and inflammatory responses in addition to their traditional role in hemostasis. During systemic infections, platelets interact with pathogens, endothelial cells, leukocytes, and inflammatory mediators, contributing to host defense mechanisms. Activated platelets release various bioactive substances that amplify the inflammatory response and help limit the dissemination of microorganisms. However, excessive platelet activation during sepsis may also result in platelet aggregation, microvascular thrombosis, and increased platelet consumption.^[7]

Thrombocytopenia is one of the most common hematological abnormalities observed in neonatal

sepsis and has been reported in a substantial proportion of affected neonates. It is defined as a platelet count below 150,000/ μ L and may range from mild reductions to severe thrombocytopenia with platelet counts below 50,000/ μ L.^[8] The pathogenesis of thrombocytopenia in neonatal sepsis is multifactorial and involves increased peripheral destruction, disseminated intravascular coagulation, immune-mediated platelet consumption, and suppression of platelet production by inflammatory cytokines.^[9] Several studies have demonstrated that severe thrombocytopenia is associated with increased disease severity, prolonged hospitalization, bleeding complications, and higher mortality rates among septic neonates.^[10]

Recent advances in automated hematology analyzers have enabled the routine measurement of platelet indices, including mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT). These parameters provide additional information regarding platelet morphology, activation, and turnover. MPV reflects the average size of circulating platelets, with larger platelets generally being younger and more metabolically active. Increased MPV has been associated with platelet activation and accelerated platelet turnover during inflammatory states and infections.^[11] PDW measures the variability in platelet size and serves as an indicator of platelet anisocytosis and activation. Similarly, PCT reflects the total circulating platelet mass and may decrease in conditions associated with thrombocytopenia.^[12]

Several recent studies have suggested that platelet indices may serve as useful diagnostic and prognostic biomarkers in neonatal sepsis. Elevated MPV and PDW values, together with reduced platelet counts and plateletcrit, have been reported in septic neonates compared with healthy controls.^[13] Furthermore, certain studies have demonstrated an association between the severity of thrombocytopenia and the causative organisms responsible for sepsis. Gram-negative infections, particularly those caused by *Klebsiella* species, *Pseudomonas aeruginosa*, and *Escherichia coli*, have been associated with more severe thrombocytopenia due to endotoxin-mediated activation of coagulation pathways and increased platelet consumption.^[14]

Despite growing evidence supporting the role of platelet parameters in neonatal sepsis, data regarding their relationship with the etiological profile of infection remain limited, particularly in Indian tertiary care settings. Variations in microbial epidemiology across different geographic regions necessitate institution-specific studies to better understand the diagnostic and prognostic utility of platelet abnormalities. Therefore, the present study was undertaken to evaluate platelet counts and platelet indices in culture-proven neonatal sepsis and to determine their association with the etiological profile of pathogens isolated in a tertiary care NICU. The findings may contribute to the development of cost-effective, readily available diagnostic tools for the early identification and risk stratification of neonatal sepsis.

MATERIALS AND METHODS

Study Design and Setting

A prospective observational study was carried out in the Neonatal Intensive Care Unit of Mahatma Gandhi Medical College and Hospital, Jaipur, over a period of eighteen months.

Study Population

All neonates admitted to the NICU with clinical features suggestive of sepsis or significant perinatal risk factors were screened. Only neonates with culture-proven sepsis were included in the final study population.

Statistical Analysis

Data were analyzed using statistical software. Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were expressed as frequencies and percentages. Chi-square test, Student's t-test were used wherever appropriate. Statistical significance was considered at $p < 0.05$.

RESULTS

Incidence of Neonatal Sepsis

During the study period, 1,489 neonates were admitted to the NICU. Among them, 440 neonates exhibited clinical signs suggestive of sepsis. Blood cultures were positive in 102 neonates, resulting in a culture-proven sepsis incidence of 6.85% among total NICU admissions and 23.18% among clinically suspected cases.

Table 1: Basic characteristics of study patients

Variables		n = 102	In %
Age Interval (In days)	1 - 7	71	69.61%
	8 - 14	28	27.45%
	15 - 21	1	0.98%
	22 - 28	2	1.96%
Gender	Male	65	63.73%
	Female	37	36.27%
Risk Factors	PROM/PPROM	28	27.45%
	Maternal GBS Colonization	26	25.49%
	Chorioamnionitis	23	22.55%
	Maternal UTI/STI	10	9.80%
	Frequent Vaginal Exams in labor	8	7.84%
	Maternal malnutrition	7	6.86%
Clinical Features	Poor Feeding	58	56.86%
	Lethargy	27	26.47%
	Respiratory distress	25	24.51%
	Jaundice	25	24.51%
	Apnea	21	20.59%
	Convulsions	16	15.69%
	Shock	9	8.82%
	Hypothermia	8	7.84%

Gender: Male neonates constituted 63.73% of culture-positive cases, while females accounted for 36.27%, indicating a male predominance.

Age: The majority of neonates developed sepsis within the first week of life. Nearly seventy percent of cases occurred between one and seven days of age, demonstrating a predominance of early-onset neonatal sepsis.

Risk Factors: Several maternal and neonatal risk factors were identified. Premature rupture of membranes, prolonged rupture of membranes, maternal infection, chorioamnionitis, low birth weight, and prematurity were among the most frequently observed risk factors.

Prematurity and low birth weight demonstrated significant associations with culture-proven sepsis, emphasizing their role as major determinants of neonatal susceptibility to infection.

Clinical Presentation: These findings highlight the nonspecific nature of neonatal sepsis and the need for laboratory support in diagnosis.

Table 2: Association of platelet count with etiological profile in neonatal sepsis

Organism	Mild (n =	Moderate (n = 44)	Severe (n =	Chi-Square	P - Value
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	32)		26)	test	
Klebsiella Pneumonia	9	14	15	9.3	0.15739
Escherichia coli	8	10	7		
Coagulase-Negative Staphylococci	7	8	1		
Staphylococcus Aureus	8	8	0		
Pseudomonas Aeruginosa	0	2	1		
Acinetobacter Baumannii	0	2	2		

The severity of thrombocytopenia appeared to differ among various pathogens, with Gram-negative infections generally producing more pronounced platelet depletion. However, statistical analysis failed to demonstrate a significant association between platelet count severity and individual etiological organisms.

These findings suggest that host inflammatory response and disease severity may play a greater role in determining platelet abnormalities than the specific pathogen involved.

Table 3: Association of thrombocytopenia with neonatal sepsis by using odds ratio

Variables		Culture Negative Sepsis (n = 338)	Culture Positive Sepsis (n = 102)	Odds Ratio	95% CI	Z-test	P - Value
Thrombocytopenia	Mild	330	32	-	-	-	-
	Moderate & Severe	8	70	90.23	39.9 - 204.2	10.81	< 0.0001

A substantial proportion of septic neonates exhibited thrombocytopenia. Moderate and severe thrombocytopenia were frequently observed among culture-positive cases.

Platelet counts were significantly lower in neonates with proven sepsis compared to non-septic neonates. The severity of thrombocytopenia increased with the severity of infection.

Odds ratio analysis demonstrated a strong association between thrombocytopenia and culture-proven neonatal sepsis. Neonates with severe thrombocytopenia had significantly higher odds of sepsis compared with those having normal platelet counts.

DISCUSSION:

Despite ongoing advances in neonatal care, sepsis remains a persistent and serious challenge within neonatal intensive care units. Its significance stems not only from how frequently it occurs but also from the difficulty in identifying it early and managing it effectively. Even today, it continues to contribute substantially to neonatal morbidity and mortality, particularly in settings where healthcare resources are limited.^[15] The impact is uneven across regions, with under-resourced healthcare systems bearing a disproportionately higher burden, underscoring its continued relevance as a public health concern.^[16]

A major factor underlying this challenge is the distinct physiology of the neonate. The neonatal immune system is inherently immature, affecting both innate and adaptive immune responses. Impaired neutrophil activity, reduced complement levels, and limited antibody production collectively weaken the neonate's ability to contain infections once initiated.^[5] As a result, even minimal bacterial exposure can escalate quickly into systemic illness in this vulnerable population. The clinical picture further complicates early recognition. Initial manifestations of neonatal sepsis are often subtle and

nonspecific, frequently overlapping with non-infectious conditions. Signs such as poor feeding, lethargy, or temperature instability may be the only early indicators, yet they lack specificity for infection.^[17] Consequently, clinicians often depend on a combination of clinical assessment and supportive investigations rather than a single confirmatory test.

Although blood culture remains the reference standard for diagnosis, it has notable limitations. Its sensitivity may be reduced by prior antibiotic administration, insufficient sample volume, or low levels of circulating organisms.^[18] This means that a considerable proportion of clinically suspected cases may not receive microbiological confirmation, adding uncertainty to both diagnosis and treatment decisions.

According to our study, the incidence of culture-proven neonatal sepsis among total NICU admissions was determined at 6.85%, with 23.18% of neonates suspected of sepsis. However, only one-fourth of cases of clinical suspicion had a positive blood culture, which is typical for neonatal practice and has been seen before. As Gideon Klinger and his colleagues (2009)^[19] report in their study evaluating early-onset sepsis among very low-birth-weight infants, there is also a considerable discrepancy between culture-proven sepsis (approximately 2.4%) and clinically suspected cases.

Risk Factors for Neonatal Sepsis The present analysis highlights a clear pattern in the distribution of maternal and perinatal risk factors, with those linked to ascending infections emerging as the most influential. Premature rupture of membranes (PROM/PPROM) was the leading contributor, identified in 27.45% of cases. This was followed by maternal Group B Streptococcus (GBS) colonisation at 25.49% and chorioamnionitis at 22.55%. Together, these findings point toward the critical role of intrauterine infection and disruption of protective barriers around the time of delivery in the pathogenesis of neonatal sepsis within this cohort. Similar observations have been documented in existing literature. In a retrospective study by Al Ghadeer et al. (2024)^[20], PROM was also recognised as a major antenatal risk factor, although reported at a lower frequency of 11.2%. The same study identified additional maternal conditions, including intrauterine growth restriction (6.7%), preeclampsia (6.7%), and maternal infections (3.7%). While the proportions differ, the repeated identification of PROM across studies reinforces its significance as a key determinant in the development of neonatal sepsis. Maternal infections such as urinary tract and sexually transmitted infections were present in 9.80% of cases in the current study, a figure notably higher than the 3.7% reported. This variation may reflect differences in antenatal screening practices, healthcare access, and population characteristics across study settings. Comparison with the findings of **Shah et al. (2025)**^[21] reveals a different distribution of risk factors. In their cohort, previous hospitalization was the most prominent factor, observed in 50.4% of cases, particularly among extramural neonates. In contrast, the current study demonstrates a stronger association with antenatal and intrapartum factors, including PROM (27.45%) and chorioamnionitis (22.55%). This contrast suggests that the relative contribution of risk factors may vary depending on whether the population is primarily inborn or referred from external facilities.

Clinical Features of Study Subjects In our study, neonatal sepsis predominantly presented with nonspecific clinical features. Poor feeding was the most common symptom, observed in 56.86% of neonates, followed by lethargy in 26.47%. Respiratory distress and jaundice were each noted in 24.51% of cases, while apnea was present in 20.59%. Convulsions were seen in 15.69% of neonates, and more severe manifestations such as shock (8.82%) and hypothermia (7.84%) were relatively less frequent. These findings indicate that early manifestations are often subtle, while more specific neurological or systemic signs tend to appear in advanced stages of illness. A similar pattern has been reported in other studies. In the study by Vijayan et al. (2020)^[22], poor feeding (66%) and lethargy (58%) were the most common clinical features among neonates with sepsis. When compared with the present study, the proportion of poor feeding (56.86%) is slightly lower but still represents the predominant symptom, while lethargy (26.47%) is considerably less frequent than the 58% reported by Vijayan et al. This difference may reflect variations in severity at presentation or differences in clinical recognition across settings. In another study by Kumari et al. (2024)^[23], reluctance to feed was observed in 43.3% of cases, respiratory distress in 25%, and hypothermia in 23.3%. These

findings closely parallel the present study, where poor feeding (56.86%) and respiratory distress (24.51%) were among the most frequent symptoms. Hypothermia in the present study (7.84%), however, was lower compared to 23.3% reported by Kumari et al., suggesting possible differences in environmental factors or case mix. When the findings are compared across studies, a consistent observation emerges that neonatal sepsis most often presents with nonspecific symptoms such as poor feeding, lethargy, and respiratory distress. However, the frequency of individual clinical features varies between studies, likely due to differences in study population, timing of presentation, and healthcare settings.

Association of Platelet Count with Etiological Profile in Neonatal Sepsis In the present study, It was observed that *Klebsiella pneumoniae*, which was also the most common isolate in this cohort, showed a wide distribution across all platelet categories, with a tendency toward more severe thrombocytopenia (15 cases). Similarly, *Escherichia coli* infections were distributed across mild (8), moderate (10), and severe (7) categories, indicating that Gram-negative organisms are frequently associated with varying degrees of platelet depletion. Among Gram-positive organisms, a different pattern was noted. Coagulase-negative staphylococci (CONS) were largely associated with mild (7) and moderate (8) thrombocytopenia, with only one case of severe thrombocytopenia. Likewise, *Staphylococcus aureus* infections were restricted to mild (8) and moderate (8) categories, with no cases of severe thrombocytopenia observed. This suggests that Gram-positive organisms in the present study were less frequently associated with severe platelet reduction compared to Gram-negative pathogens. Despite these observed variations, the association between the causative organism and severity of thrombocytopenia was not statistically significant ($p = 0.15739$). This suggests that while certain organisms may appear to be linked with more severe platelet reduction, the relationship is not strong enough to establish a direct etiological influence on thrombocytopenia severity. These findings are in agreement with previous studies that have explored organism-specific hematological responses. Guida et al. (2003)^[24] reported that thrombocytopenia is a common feature in neonatal sepsis but may not always show a consistent organism-specific pattern. Similarly, in the study by Talat et al. (2023)^[25], although moderate (33.1%) and severe thrombocytopenia (16.9%) were significantly associated with sepsis overall, no clear differentiation was made between individual pathogens in terms of platelet severity. On the other hand, some studies suggest a greater association of severe thrombocytopenia with Gram-negative infections. The predominance of Gram-negative organisms such as *Klebsiella* and *E. coli* in the present study, along with their representation across moderate and severe categories, supports this observation to some extent. Similar trends have been reported in earlier studies where Gram-negative sepsis was linked with more profound platelet consumption due to endotoxin-mediated mechanisms. However, when compared with studies such as Vijayan et al. (2020)^[22] and Alharbi et al. (2022)^[26], where Gram-positive organisms like CONS were predominant, the variability in organism distribution further complicates the relationship between etiology and platelet count. This indicates that thrombocytopenia in neonatal sepsis is likely multifactorial, influenced not only by the infecting organism but also by host factors, severity of infection, and timing of presentation. The present study suggests that although certain organisms, particularly Gram-negative bacteria, appear to be associated with more severe thrombocytopenia, the lack of statistical significance indicates that platelet count alone cannot reliably predict the etiological agent.

Association of Thrombocytopenia with Neonatal Sepsis (Odds Ratio Analysis) In the present study, a very strong association was observed between the severity of thrombocytopenia and culture-proven neonatal sepsis. Neonates with moderate to severe thrombocytopenia had markedly higher odds of sepsis compared to those with mild thrombocytopenia (OR = 90.23; 95% CI: 39.9–204.2; $p < 0.0001$). While only 8 out of 338 culture-negative neonates had moderate to severe thrombocytopenia, a substantially larger proportion—70 out of 102—was seen among culture-positive cases. This sharp contrast highlights that increasing severity of platelet reduction is closely linked with the presence of

sepsis. These findings are consistent with observations from previous studies that have emphasized the strong relationship between thrombocytopenia and neonatal sepsis. Talat et al. (2023)^[25] reported that moderate thrombocytopenia was present in 33.1% and severe thrombocytopenia in 16.9% of septic neonates. In contrast, Singh et al. (2018)^[27] reported severe thrombocytopenia as the most common presentation (36.2%), followed by moderate (34.3%) and mild (24.8%). Further supporting evidence comes from Chate et al. (2019)^[28], who demonstrated that thrombocytopenia was more common among vulnerable groups such as low birth weight neonates, with 96.55% affected compared to 84% in normal birth weight neonates. Although their focus was on prevalence rather than severity-based risk, the higher occurrence of thrombocytopenia in high-risk neonates indirectly reinforces its role as a marker of disease severity.

CONCLUSION:

Neonatal sepsis continues to be a significant cause of neonatal morbidity and mortality. Gram-negative organisms remain the predominant pathogens in tertiary care NICUs. Thrombocytopenia is a common hematological abnormality in culture-proven neonatal sepsis and demonstrates a strong association with infection. Platelet count provides additional information regarding platelet activation and consumption during sepsis. This parameter may serve as useful adjunctive biomarkers for early diagnosis and prognostic assessment. Routine monitoring of platelet counts can assist clinicians in identifying high-risk neonates and initiating timely therapeutic interventions. Larger multicentric studies are required to further establish the diagnostic and prognostic utility of platelet parameters in neonatal sepsis.

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