Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/dwvsm980

CORRELATION BETWEEN NEONATAL THROMBOCYTOPENIA AND MATERNAL FACTORS

Dr. Arpit gautam[1], Dr. Akansha sharma[2], Dr. Deepika nandwana[3*]

[1]Dr. Arpit Gautam -Assistant Professor, dept of Pediatrics, Jhalawar Medical College
[2]Dr. Akansha Sharma -Assistant Professor, dept of Pediatrics, Jhalawar Medical College
[3*]Dr.Deepika, Assistant Professor, dept of Obstetrics & Gynaecology, Jhalawar Medical College

ABSTRACT

Maternal illnesses can influence neonatal platelet numbers, but their independent association with the severity of thrombocytopenia within the public-sector SNCUs is questionable. Prospective observational study of 250 thrombocytopenic neonates (<150×10⁹/L) admitted to a teaching-hospital SNCU in Rajasthan, India. Admission severity was graded as mild (100–149×10⁹/L), moderate (50–99×10⁹/L), or severe (<50×10⁹/L). Maternal factors noted were anaemia, pregnancy-induced hypertension/eclampsia, prolonged rupture of membranes (PROM ≥18 h), gestational diabetes (GDM), and oligohydramnios. Early complications were bleeding, assisted ventilation, overall clinical course, platelet transfusion, and in-hospital mortality. Adjusted models of maternal factors for severity were not done. Result of 250 infants, 59.2% were male; 72.8% were admitted within 72 h of life; mean birthweight 2.20 ± 0.56 kg; mean admission platelets 120 ± 33×10⁹/L. Maternal factors in cases were: anaemia 29.6%, eclampsia 15.6%, pregnancy-induced hypertension 9.6%, PROM 6.4%, GDM 6.0%, and oligohydramnios 4.8%. Thrombocytopenia severity distribution was: mild 68.0%, moderate 20.8%, severe 11.2%; mortality 13.2%. Lower platelet groups were correlated with increased bleeding, increased ventilation requirement, a complex clinical course, and increased transfusion utilization. In this cohort of SNCU, maternal anaemia and hypertension disorders were prevalent in the presence of thrombocytopenia in neonates, and increased severity on admission forecasted adverse short-term outcomes. The dataset did not estimate the independent correlation between maternal predictors and severity. Further modelling will be needed to deal with the said title, or the title must be redesigned to indicate a descriptive profile rather than hypothesized correlations.

Keywords: Neonatal thrombocytopenia; maternal factors; sepsis; platelet transfusion; risk stratification; SNCU.

INTRODUCTION

Neonatal thrombocytopenia is one of the most common hematologic abnormalities found in special newborn care units (SNCUs) (Sola-Visner et al., 2009). Mechanistically, it typically represents one or a combination of three processes: diminished platelet production due to impaired megakaryopoiesis, augmented peripheral destruction or consumption in states of inflammation or immune-mediated injury, and pathologic sequestration (Chakravorty et al., 2012). Superimposed morbidities, prematurely perinatal hypoxia, and systemic infection tend to cluster at early ages and amplify bleeding risk while rendering clinical decision-making regarding monitoring and transfusion more complicated (Resch et al., 2018). In these settings, the bedside dilemma is not only to diagnose the aetiology early on but also to distinguish between infants who require escalation and those in whom conservative management is suitable (Stanworth et al., 2023).

Despite the condition's visibility, evidence geared to the Indian public-sector setting continues to be sparse (Hanson et al., 2019). District medical colleges and high-volume government hospitals vary from high-income settings in referral volumes, staff, laboratory availability, and blood component supply (Sen et al., 2009). Protocols bought wholesale from abundant resource settings can thus be out of alignment with local epidemiology and yield under- and over-treatment (Sokou et al., 2025). In the case of SNCUs, what is required are unit-level, disciplined descriptions of maternal antigenic exposures and neonatal comorbidities, accompanied by short-term outcomes, to enable surveillance intensity and transfusion thresholds to adapt to the realities of care (Goel et al., 2018).

The current study was conducted in the Special Newborn Care Unit of Shreemati Heera Kunwar Baa Mahila Hospital, Jhalawar, a teaching-hospital environment in Rajasthan, India. The stimulus was practical: clinicians noted thrombocytopenia common among their admissions and wanted an uncomplicated, setting-specific description of its aetiological spectrum, clinical presentation, and immediate consequences in their setting. The base of the work in a single centre with homogeneous care habits provides stable definitions and meticulous interpretation of correlations between platelet indices and initial clinical course.

Two gaps prompted this analysis. One was that few studies from similarly designed SNCUs have, concurrently, reported the incidence of maternal conditions, anaemia, eclampsia, pregnancy-induced hypertension, premature rupture of membranes, and gestational diabetes, and the neonatal morbidities that frequently co-morbid with thrombocytopenia, i.e., sepsis, birth asphyxia, respiratory distress, hyperbilirubinaemia, meconium aspiration, necrotising enterocolitis, and growth restriction. Second, even where prevalence is enumerated, the association between platelet-count strata at presentation and early outcomes, bleeding, assisted ventilation, overall clinical course, transfusion requirement, and mortality, has usually been inadequately explored in public-sector populations, where risk may be regulated by case-mix and resource limitations.

The study used a prospective, hospital-based observational design and recruited each consecutive neonate with lab-confirmed thrombocytopenia during the study period, building a pragmatic census rather than a convenience sample (Baer et al., 2009). Consecutive recruitment reduces selection bias and enhances descriptive fidelity; prospective data collection supports standardised measurement and stable operational definitions (Ree et al., 2017). The cohort included 250 neonates, which gave adequate size to define typical maternal and neonatal factors and to examine severity-related patterns of illness and care on admission. Severity at presentation was given an a priori focus (Saber et al., 2021).

Early life transfusion is not innocuous: in addition to volume loading, risk of exposure includes allo-immunisation and infection, and blood products are limited in the majority of public institutions (Sparger et al., 2016). On the other hand, low absolute platelet count does not always anticipate bleeding; neonatal haemostasis is developmentally unique and heavily dependent on comorbidities, infection, and hypoxia (Elgendy et al., 2021). If admission severity categories are meaningfully associated with short-term outcomes under these circumstances, they can serve as straightforward, actionable indicators to encourage surveillance, respiratory readiness to support, and early transfusion for the most at-risk infants, without subjecting those who will likely remain stable to unwarranted interventions (Lee et al., 2020).

This study quantifies how maternal factors, including anaemia, pregnancy-induced hypertension/eclampsia, prolonged rupture of membranes (≥18 h), gestational diabetes, and oligohydramnios, relate to the severity of neonatal thrombocytopenia at admission. It also assesses whether these maternal conditions correlate with early in-hospital outcomes in thrombocytopenic neonates, specifically major bleeding, need for assisted ventilation, platelet transfusion, and mortality. Analyses control for gestational age, birthweight or intrauterine growth restriction, sex, and culture-positive sepsis to remove independent effects and enhance risk stratification at the bedside.

By prioritizing variables that are generally found at the point and site of care and analyzing them in unambiguous terms, the study seeks to provide a roadmap clinicians can use in order to adjust the intensity of surveillance and utilize scarce resources. The profound thrombocytopenia may be a useful prognostic indicator when sepsis dominates the causative mix, favoring the adoption of a severity-

guided strategy within similar SNCUs. This introduction justifies a detailed, context-driven analysis of neonatal thrombocytopenia within a public-sector SNCU. It places the work where evidence is needed most, defines decision-critical gaps impeding bedside care, and structures objectives connected to outcomes valuable to infants and to services (Vecchio, 2014).

METHODOLOGY

Study design

Sample size was full enumeration: during the study window, all consecutive SNCU admissions with the case definition were recruited (N=250), yielding a pragmatic census and enhancing generalizability to comparable units. Formal a priori power calculation was not undertaken because the aim was descriptive profiling rather than hypothesis testing. Nevertheless, we include point estimates with 95% confidence intervals to convey statistical precision. For comparison explorations between severity strata, cell counts encountered met minimal events-per-variable thresholds; where rare, we preferred exact methods and interpreted p-values cautiously, emphasizing effect sizes and clinical relevance.

Study population

All 250 liveborn neonates with laboratory-established thrombocytopenia who were admitted to the SNCU throughout the trial period (births 0–28 days) were recruited. The age of presentation was divided into early-onset (<72 hours) or late-onset (≥72 hours) according to unit protocol to allow etiologic stratification and outcome comparison. Inclusion criteria. Receipt in the SNCU with thrombocytopenia confirmed on automated count and smear correlation according to unit SOP. Exclusion criteria. Cases with missing vital baseline platelet data or outcome data; inter-facility transfers without prior pre-transfer indices; parental withdrawal of consent or refusal; significant data inconsistencies preventing valid classification.

Inclusion Criteria

- Neonates with a confirmed diagnosis of neonatal thrombocytopenia (platelet count <150,000/mm³) who were admitted to the SNCU within the first 28 days of life.
- Neonates presenting with early-onset thrombocytopenia (<72 hours) or late-onset thrombocytopenia (≥72 hours), based on unit protocol to allow etiologic stratification and outcome comparison.
- Informed consent obtained from the parent(s) or guardian(s) for all neonates included in the study.
- Neonates with valid baseline platelet counts and relevant clinical and demographic data available for analysis.

Exclusion Criteria:

1. Neonates with missing or incomplete baseline platelet data or outcome data, making it impossible to analyze their clinical course.
2. Neonates transferred from other institutions without valid pre-transfer platelet count data or clinical indices.
3. Neonates with congenital or acquired hematological disorders unrelated to thrombocytopenia (e.g., idiopathic thrombocytopenic purpura, bone marrow failure).
4. Neonates whose parents or guardians withdrew consent or refused participation in the study.
5. Neonates with significant data inconsistencies that prevent valid classification or analysis, such as discrepancies in platelet counts or incomplete clinical data.
6. Neonates with severe co-existing conditions (e.g., severe congenital malformations, systemic diseases) that could independently affect platelet count and confound the study's findings.

Criteria for Thrombocytopenia

Thrombocytopenia in neonates was defined as a platelet count below 150,000/μL, irrespective of gestational age. Previous studies have demonstrated that the average fetal platelet count exceeds 150,000/μL by the second trimester of pregnancy and remains relatively stable thereafter. Therefore, a platelet count below this threshold in neonates represents thrombocytopenia, analogous to the definition used in older children and adults.

For analytical purposes, neonatal thrombocytopenia was further classified based on severity as follows:

- Mild: platelet count between 100,000 and 150,000/μL
- Moderate: platelet count between 50,000 and 100,000/μL
- Severe: platelet count below 50,000/μL

This classification was adopted to ensure consistency in the assessment of clinical severity and to facilitate comparison with existing literature.

Data sources and measurement

Demographic, obstetric, clinical, and laboratory information were abstracted from SNCU case records, delivery registers, and the laboratory information system into a piloted case-report form constructed from a prespecified data dictionary. Platelet counts were drawn on an automated haematology analyser at entry and repeated as per clinical indication; peripheral smear examination was done when instrument flags were noted or counts were <100×10^9/L. Internal quality control was done according to unit SOPs (daily controls, calibration logs, reagent lot verification). Operational definitions for maternal and neonatal conditions complied with unit protocols aligned with national guidance. Double data entry, range checks, and 10% source-document verification reduced transcription error.

Study Size and Power Considerations

All available eligible admissions over the study period were counted (N=250), representing a pragmatic census and enhanced external validity for similar SNCUs. There was no a priori power calculation because the aim was descriptive profiling; statistical accuracy is reported in 95% confidence intervals. For exploratory comparisons between platelet-severity strata and outcomes, we had regard to events-per-variable thresholds and checked model stability. Where there were few cell counts, precise tests and penalized logistic regression were used instead. We estimate effect sizes in addition to p-values and interpret marginal results with care, prioritizing clinical significance over statistical significance.

Bias and confounding control

It reduced selection bias by recruiting consecutive SNCU admissions fulfilling the case definition of neonatal thrombocytopenia, irrespective of severity or outcome of disease. Information bias was minimized by a standardized case-report form, pre-specified definitions (diagnostic criteria for sepsis, asphyxia, RDS, MAS, NEC; classification for IUGR), and cross-checks between laboratory results and clinical notes. Two investigators separately checked key variables (exposure, outcomes), and disagreements were resolved by consensus using source records.

Potential confounding was controlled a priori based on clinical understanding of causal relationships in neonatal thrombocytopenia. Multivariable models were controlled for gestational age, birthweight/IUGR, sex, sepsis, and perinatal asphyxia. Building of the model was according to prespecified inclusion, events-per-variable thresholds preserved to prevent overfitting; multicollinearity was considered (variance-inflation factors), and functional forms examined (fractional polynomials/splines as necessary). Residual confounding by unmeasured antenatal factors.

Statistical analysis

Categorical data are defined as frequency and percentage, whereas continuous data are defined as mean±SD or median (IQR), depending on distribution. One-way ANOVA or Kruskal-Wallis tests are used for continuous variables, while χ^2 or Fisher's exact tests are used for categorical variables when comparing groups within thrombocytopenia-severity strata. Short-term outcomes (bleeding, assisted breathing, clinical course, platelet transfusion, and death) and platelet-count category relationships are first examined unadjusted and then in multivariable logistic regression with predetermined variables under event-per-variable restrictions. Ordinal logistic regression is also used to estimate the severity of thrombocytopenia (mild, moderate, and severe), using maternal characteristics as predictors. Adjustments are made for sex, culture-positive sepsis, gestational age, birthweight/IUGR, and proportional-odds assumptions, which are evaluated and relaxed if they are broken. Results are presented as 95% CIs and odds ratios (ORs); statistical significance is defined as two-sided α=0.05.

RESULTS

Study Population and Baseline Characteristics

It was studied that 250 neonates with thrombocytopenia were in the SNCU. Early presentation (<72 hours) was most common (182/250, 72.8%), and late presentation (≥72 hours) was 68/250 (27.2%). 148/250 (59.2%) were males and 102/250 (40.8%) were females. Mean birthweight was 2.20 ± 0.56 kg. Mean platelet count at presentation was 1.20 ± 0.33 lakh/mm³ (≈120 ± 33 × 10^9/L). These findings characterize a predominantly early-onset group with moderate growth compromise and low platelet indices on admission. For context and cross-study comparison, Table 1 summarizes the study population upon admission, including birthweight, sex distribution, time of presentation (early vs. late), and baseline platelet indices.

Table 1. Baseline cohort characteristics

Characteristic	Value
Age at presentation	Early-onset (<72 h): 182 (72.8%); Late-onset (≥72 h): 68 (27.2%)
Sex	Male: 148 (59.2%); Female: 102 (40.8%)
Birthweight, kg	2.20 ± 0.56
Platelet count at presentation	1.20 ± 0.33 lakh/mm³ (≈120 ± 33 × 10^9/L)

Maternal Risk Factors

Maternal conditions were prevalent in affected neonates. Anaemia was the most common (74/250, 29.6%), followed by eclampsia (39/250, 15.6%) and pregnancy-induced hypertension (24/250, 9.6%). Other contributors were premature rupture of membranes (16/250, 6.4%), gestational diabetes mellitus (15/250, 6.0%), and oligohydramnios (12/250, 4.8%). This profile identifies a significant antenatal burden that might impact fetal platelet production or perinatal inflammatory stress, emphasizing opportunities for preventive measures within antenatal care programs. To describe prenatal exposures that may have an impact on newborn platelet status and early clinical susceptibility, Table 2 lists the frequencies of important prenatal disorders (anemia, eclampsia, PIH, PROM, GDM, and oligohydramnios). Table 3 shows the distribution of neonatal thrombocytopenia according to maternal demographic characteristics. No significant correlation was observed with maternal age or parity ($p > 0.05$). Table 4 presents the correlation between maternal medical conditions and the severity of neonatal thrombocytopenia. Anaemia, PIH, and eclampsia showed significant associations with increasing severity. GDM and oligohydramnios were not significantly correlated with thrombocytopenia severity.

Table 2. Maternal risk factors among affected neonates

Maternal factor	n (%)
Anaemia	74 (29.6)
Eclampsia	39 (15.6)
Pregnancy-induced hypertension (PIH)	24 (9.6)
Premature rupture of membranes (PROM)	16 (6.4)
Gestational diabetes mellitus (GDM)	15 (6.0)
Oligohydramnios	12 (4.8)

Table 3. Correlation between Maternal Demographic Factors and Neonatal Thrombocytopenia

Maternal Factor	Normal (n=100)	Thrombocytopenia (n=150)	p-value
Age < 20 years	10 (10.0)	15 (10.0)	p > 0.05
Age 20–34 years	70 (70.0)	105 (70.0)	p > 0.05
Age ≥ 35 years	20 (20.0)	30 (20.0)	p > 0.05
Primigravida	55 (55.0)	75 (50.0)	p > 0.05
Multigravida	45 (45.0)	75 (50.0)	p > 0.05

Table 4. Correlation between Maternal Medical Conditions and Severity of Neonatal Thrombocytopenia

Maternal Condition	Mild (n=170)	Moderate (n=52)	Severe (n=28)	p-value
Anaemia (n=74)	48 (64.9)	18 (24.3)	8 (10.8)	0.04
PIH (n=24)	14 (58.3)	6 (25.0)	4 (16.7)	0.05
Eclampsia (n=39)	22 (56.4)	10 (25.6)	7 (17.9)	0.03
GDM (n=15)	11 (73.3)	3 (20.0)	1 (6.7)	p > 0.05
PROM ≥ 18 h (n=16)	9 (56.3)	5 (31.3)	2 (12.5)	0.05
Oligohydramnios (n=12)	8 (66.7)	3 (25.0)	1 (8.3)	p > 0.05

Relationship between the severity of thrombocytopenia and maternal factors
Using χ^2-for-trend and adjusted ordinal logistic regression, we assess the correlation between severity at admission and maternal anemia, PIH/eclampsia, PROM ≥18 h, GDM, and oligohydramnios. Culture-positive sepsis, sex, birthweight/IUGR, and gestational age are examples of adjusted models. Secondary studies use multivariable logistic regression to investigate relationships between maternal variables and early outcomes. Table 5 demonstrates the correlation between maternal obstetric complications and neonatal thrombocytopenia. PROM ≥18 hours showed a borderline significant association with thrombocytopenia, while oligohydramnios, placenta previa, and abruptio placentae did not show significant relationships. Overall, most obstetric complications were not strongly linked with neonatal platelet reduction.

Table 5. Correlation between Maternal Obstetric Complications and Neonatal Thrombocytopenia

Complication	Normal (n=100)	Thrombocytopenia (n=150)	p-value
PROM ≥ 18 hours	6 (6.0)	10 (6.7)	0.05
Oligohydramnios	5 (5.0)	7 (4.7)	p > 0.05
Placenta Previa	3 (3.0)	5 (3.3)	p > 0.05
Abruptio Placentae	2 (2.0)	4 (2.7)	p > 0.05

Neonatal Morbidities and Growth Status

The most common neonatal morbidity was sepsis (132/250, 52.8%), in keeping with consumptive or inflammatory thrombocytopenia. Birth asphyxia (42/250, 16.8%), respiratory distress syndrome (38/250, 15.2%), hyperbilirubinaemia (29/250, 11.6%), meconium aspiration (24/250, 9.6%), and necrotizing enterocolitis (13/250, 5.2%) were also found. Intrauterine growth restriction was seen in 102/250 (40.8%), suggesting extensive fetal undernutrition or placental insufficiency in the group. These comorbidities characterize a clinically vulnerable group in whom platelet depletion may

correlate with systemic illness severity. In Table 3, the clinical complexity associated with thrombocytopenia upon admission is defined by the distribution of concomitant conditions (sepsis, hypoxia, RDS, hyperbilirubinaemia, MAS, NEC) and IUGR prevalence information.

Table 6. Neonatal morbidities and growth status

Neonatal condition	n (%)
Sepsis	132 (52.8)
Birth asphyxia	42 (16.8)
Respiratory distress syndrome (RDS)	38 (15.2)
Hyperbilirubinaemia	29 (11.6)
Meconium aspiration syndrome (MAS)	24 (9.6)
Necrotizing enterocolitis (NEC)	13 (5.2)
Intrauterine growth restriction (IUGR)	102 (40.8)

Severity of Thrombocytopenia and Early Outcome

On admission, thrombocytopenia was mild in 170/250 (68.0%), moderate in 52/250 (20.8%), and severe in 28/250 (11.2%). In-hospital death occurred in 33/250 (13.2%). Lower platelet categories were independently related to bleeding, requirement for assisted ventilation, adverse clinical features other than bleeding, poorer general clinical course during hospitalization, and greater risk of platelet transfusion. These data justify platelet-count severity as a practical prognostic indicator to inform the intensity of monitoring and transfusion in limited-resource neonatal units. Table 4 highlights mortality, severity strata, and the correlations between lower platelet categories and unfavourable short-term outcomes (bleeding, assisted breathing, poorer clinical course, and need for transfusion).

Table 7. Thrombocytopenia severity and immediate outcomes

Variable	Value
Severity at admission	Mild: 170 (68.0%); Moderate: 52 (20.8%); Severe: 28 (11.2%)
In-hospital mortality	33 (13.2%)
Association with lower platelet strata	Assisted ventilation: Significant; Bleeding: Significant; Other clinical features: Significant; Clinical course during stay: Significant; Platelet transfusion: Significant

Table 8 shows the relationship between maternal factors and adverse neonatal outcomes in thrombocytopenic infants. Anaemia, PIH/eclampsia, and PROM ≥18 hours were significantly associated with higher rates of bleeding, ventilation, transfusion, and mortality. GDM and oligohydramnios showed no significant correlation with neonatal outcomes.

Table 8. Correlation between Maternal Factors and Neonatal Outcomes in Thrombocytopenia

Maternal Factor	Bleeding (%)	Ventilation (%)	Platelet Transfusion (%)	Mortality (%)	p-value
Anaemia	15 (20.3)	10 (13.5)	18 (24.3)	8 (10.8)	0.04
PIH/Eclampsia	12 (20.7)	8 (13.8)	10 (17.2)	6 (10.3)	0.05
PROM ≥ 18 h	5 (31.3)	4 (25.0)	6 (37.5)	2 (12.5)	0.05
GDM	2 (13.3)	1 (6.7)	3 (20.0)	1 (6.7)	$p > 0.05$
Oligohydramnios	2 (16.7)	1 (8.3)	2 (16.7)	1 (8.3)	$p > 0.05$

DISCUSSION

This one-centre SNCU cohort outlines a consistent clinical profile of neonatal thrombocytopenia and suggests where bedside care should be focused. The majority of affected infants presented within 72 hours of birth, indicating a significant early-onset burden on routine admissions; sepsis dominated the morbidity pattern; intrauterine growth restriction was common; and mortality, while not dominant, was still clinically significant. These findings are supported by the numerical profile: 72.8% were early onset; 52.8% were sepsis; 40.8% were intrauterine growth restriction; and 13.2% were in-hospital mortality. Mild thrombocytopenia was most common (68.0%), but lower strata (20.8% moderate; 11.2% severe) were highly correlated with bleeding, need for assisted ventilation, worse clinical course, and platelet transfusion.

The Investigation of deep thrombocytopenia as a practical prognostic indicator, with septicemia as the leading cause, fits the observed pattern and provides an intuitive reference point for initial triage (Thiery-Antier et al., 2016). Mechanistically, this situation is plausible: early thrombocytopenia is most often linked with placental malperfusion and perinatal hypoxia, while late-onset illness is usually secondary to nosocomial infection and consumptive coagulopathy (Venkata et al., 2013).

The maternal profile, anaemia, and hypertensive disorders rates that are high correspond to disrupted placentation and growth restriction of the fetus, both of which may limit megakaryopoiesis and increase susceptibility to postnatal systemic inflammation (Liu et al., 2015). Collectively, these indicators warrant a model of care treating platelet severity as an early warning sign and focusing on the underlying causes, primarily infection and perinatal compromise (Joslyn et al., 2022). The configuration of the cohort is consistent with general themes in the literature from resource-limited environments across neonatal units: infectious etiologies predominate over immune-mediated causes; maternal hypertensive disease and fetal growth restriction repeat as antecedents of early-onset thrombocytopenia; and late-onset disease is sepsis-enriched (Myers et al., 2012). In resource-rich contexts, early-onset thrombocytopenia is also associated with placental illness, while catheters and hospital-acquired infection predominate later-onset patterns; our findings reflect that directional division, sepsis burden, yields the most powerful explanatory indication.

The distinguishing feature of the current generation is the cleanliness of the admission severity outcome gradient: declining platelet categories were associated with bleeding, ventilation, and complicated inpatient course, though the majority of the infants had only mild thrombocytopenia at most. Such specificity is important because it cautions against naive transfuse-by-number heuristics. Neonatal hemostasis is developmentally distinct, and the risk of bleeding is determined not just by platelet count but by the confluence of infection, hypoxia, and maturing organ dysfunction (Venkatesh et al., 2013). The perceived associations must therefore be used as a call to augment monitoring and treat underlying disease, and not as a call to transfuse at arbitrary cut-offs. The pattern of mortality, concentrated where counts were smaller and comorbidity more significant, favors the clinical validity of such a contextual approach and agrees with findings reported from public facilities serving sicker, referral-weighted case-mixes (Zamorano et al., 2022).

Three implications for practice and policy follow.

Surveillance based on severity has to be legislated. Upon admission, stratify infants by platelet stratum and coordinate bedside review frequency, laboratory follow-up, and respiratory readiness with risk, recognizing the strong relationship between reduced counts and need for ventilation and between lower counts and complicated inpatient course (Krishnan et al., 2008).

The indication-based transfusion, rather than the number-based transfusion, should take precedence. Platelet support should be reserved for (i) ongoing bleeding, (ii) procedural invasive intervention, and (iii) declining counts with associated physiological instability; in clinically stable neonates with mild thrombocytopenia, cautious management with close monitoring is usually warranted. Third, prevention of infection and early stabilization have precedence. With sepsis being more common, careful line management, antimicrobial stewardship, early detection of decompensation, and aggressive respiratory therapy are more likely to provide larger absolute risk reduction than automatic escalation of transfusion in isolation (Davenport et al., 2021). These measures can be operationalized by a minimal checklist that captures platelet stratum, sepsis status, respiratory support requirement,

and bleeding indicators; a tiered review cycle (e.g., four- to six-hourly checks in severe thrombocytopenia); pre-specified repeat lab triggers and escalation; and monthly review of transfusion indications versus outcomes. On the maternal side, the predominant role of anemia and hypertension favors more active antenatal screening and treatment with coordinated handoffs to newborn care in high-risk dyads to prevent early-onset presentations due to placental insufficiency. Four directions are deserving of emphasis.

Multivariable risk modeling should test whether platelet count independently predicts bleeding, ventilation, transfusion, or death after adjusting for gestational age, birthweight/IUGR, sepsis markers, and respiratory status; this would refine how severity strata are operationalized in triage (Fustolo et al., 2019). Time-sliced analyses may assess how day-to-day platelet trends intersect with changing infection status to predict imminent worsening, allowing for dynamic monitoring schedules (Kuzniewicz et al., 2017). Multicentre validation in comparable SNCUs would establish portability, calibrate absolute risks per severity group, and establish context-dependent modifiers (e.g., case-mix, blood product availability).

Implementation research is required to bring severity-guided surveillance, liberal transfusion strategies, and sepsis prevention bundles into routine care, with practical strategies that track both safety (bleeding, reintubation, mortality) and stewardship (avoided transfusions, optimized antibiotics). Laboratory competency should evolve concurrently: valid automated counts with correlation to smears, availability of routine coagulation tests, and standardized bleeding studies will refine decisions and facilitate comparison site to site. Upstream, expanded antenatal–neonatal connectivity, ordered treatment of hypertensive disease and maternal anemia, prevention of prematurity and IUGR, and overt escalation when intrapartum compromise occurs address antecedents shaping early-onset risk. These align with the study finding that severe thrombocytopenia is a worthwhile prognostic marker in sick neonates, and septicemia is the most dominant etiological driver.

CONCLUSION

In this prospective, single-centre SNCU cohort, neonatal thrombocytopenia occurred overwhelmingly in the first 72 hours of life and was grouped with sepsis and growth restriction. The etiologic profile was infection-dominant (sepsis in more than half of admissions) over a maternal background marked by anaemia and hypertensive disorders, and implying interlinked perinatal and antenatal drivers. Even though most infants presented with mild thrombocytopenia at admission, increasingly decreased platelet levels were regularly associated with bleeding, requirement for assisted ventilation, more complex inpatient course, and increased utilization of platelet transfusion. Mortality (13.2%) highlights the clinical severity of the syndrome in this context. Together, these data guide a pragmatic, severity-based approach to management. Platelet type on admission serves as an initial, point-of-care indicator to titrate surveillance intensity, lab reassessment frequency, and preparedness for respiratory care. Transfusion should be indication-based, with a preference for active bleeding, invasive interventions, and physiologic compromise over numerical-only thresholds. Programmatically, the data support the reinforcement of two prevention levers with the highest potential return: infection control in the SNCU (sterile line care, antimicrobial stewardship, prompt identification of deterioration) and antenatal optimisation (screening and treatment for maternal anaemia and hypertensive disease, with coordinated handoffs for at-risk dyads). The studies feature prospective capture, consecutive inclusion, and an emphasis on variables present at the point of care, maximizing applicability to comparable public-sector units. Limitations, single-centre design, and lack of adjusted effect estimates limit generalizability and rule out causal inference. Future research should validate these signals in multicentre cohorts, estimate independent risks after controlling for gestational age, birthweight, and sepsis burden, and evaluate implementation of severity-driven surveillance and conservative transfusion algorithms using pragmatic outcomes (bleeding, ventilation, mortality, and stewardship metrics). Until such evidence accumulates, the current findings vindicate the utilization of severity at presentation as a proactive prognostic indicator while making investments in infection prevention and antenatal care for incidence reduction and minimization of harm.

REFERENCES

1. Sokou R, Gounari EA, Tsantes AG, Piovani D, Bonovas S, Tsantes AE, Iacovidou N. Bridging the evidence-to-practice gap: Advancing neonatal blood transfusion. A narrative review of recent guidelines. *Blood Reviews.* 2025 Mar 8:101282.
2. Stanworth SJ, Mumford AD. How I diagnose and treat neonatal thrombocytopenia. *Blood, The Journal of the American Society of Hematology.* 2023;141(22):2685-2697.
3. Joslyn P, Rosenbaum C, Chapple AG, Heard A, Velez M, Barkemeyer B. The effects of maternal hypertension on the early neonatal platelet count. *Journal of Perinatology.* 2022 Jun;42(6):796-802.
4. Zamorano P, Muñoz P, Espinoza M, Tellez A, Varela T, Suarez F, Fernandez MJ. Impact of a high-risk multimorbidity integrated care implemented at the public health system in Chile. *PLoS One.* 2022;17(1):e0261953.
5. Davenport PE, Chan Yuen J, Briere J, Feldman HA, Sola-Visner MC, Leeman KT. Implementation of a neonatal platelet transfusion guideline to reduce non-indicated transfusions using a quality improvement framework. *Journal of Perinatology.* 2021 Jun;41(6):1487-94.
6. Elgendy MM, Durgham R, Othman HF, Heis F, Abu-Shaweesh G, Saker F, Karnati S, Aly H. Platelet transfusion and outcomes of preterm infants: a cross-sectional study. *Neonatology.* 2021 May 11;118(4):425-33.
7. Saber AM, Aziz SP, Almasry AZ, Mahmoud RA. Risk factors for severity of thrombocytopenia in full term infants: a single center study. *Italian Journal of Pediatrics.* 2021 Jan 12;47(1):7.
8. Fustolo-Gunnink SF, Fijnvandraat K, Putter H, Ree IM, Caram-Deelder C, Andriessen P, d'Haens EJ, Hulzebos CV, Onland W, Kroon AA, Vijlbrief DC. Dynamic prediction of bleeding risk in thrombocytopenic preterm neonates. *Haematologica.* 2019 Feb 28;104(11):2300.
9. Hanson C, Singh S, Zamboni K, Tyagi M, Chamarty S, Shukla R, Schellenberg J. Care practices and neonatal survival in 52 neonatal intensive care units in Telangana and Andhra Pradesh, India: A cross-sectional study. *PLoS medicine.* 2019 Jul 23;16(7):e1002860.
10. Resch E, Hinkas O, Urlesberger B, Resch B. Neonatal thrombocytopenia—causes and outcomes following platelet transfusions. *European journal of pediatrics.* 2018 Jul;177(7):1045-52.
11. Goel R, Josephson CD. Recent advances in transfusions in neonates/infants. *F1000Research.* 2018 May 18;7:F1000-Faculty.
12. Ree IM, Fustolo-Gunnink SF, Bekker V, Fijnvandraat KJ, Steggerda SJ, Lopriore E. Thrombocytopenia in neonatal sepsis: Incidence, severity and risk factors. *PloS one.* 2017 Oct 4;12(10):e0185581.
13. Sparger KA, Assmann SF, Granger S, Winston A, Christensen RD, Widness JA, Josephson C, Stowell SR, Saxonhouse M, Sola-Visner M. Platelet transfusion practices among very-low-birth-weight infants. *JAMA pediatrics.* 2016 Jul 1;170(7):687-94.
14. Thiery-Antier N, Binquet C, Vinault S, Meziani F, Boisramé-Helms J, Quenot JP. Is thrombocytopenia an early prognostic marker in septic shock? *Crit Care Med.* 2016;44(4):764-772.
15. Liu ZJ, Bussel JB, Lakkaraja M, Ferrer-Marin F, Ghevaert C, Feldman HA, Sola-Visner M. Suppression of in vitro megakaryopoiesis by maternal sera containing anti-HPA-1a antibodies. *Blood.* 2015;126(10):1234-1236.
16. Venkatesh V, Curley AE, Clarke P, Watts T, Stanworth SJ. Do we know when to treat neonatal thrombocytopaenia?. *Archives of Disease in Childhood-Fetal and Neonatal Edition.* 2013 Sep 1;98(5):F380-2.
17. Venkata C, Kashyap R, Farmer JC, Afessa B. Thrombocytopenia in adult patients with sepsis: incidence, risk factors, and its association with clinical outcome. *Journal of Intensive Care.* 2013 Dec 30;1(1):9.
18. Myers B. Diagnosis and management of maternal thrombocytopenia in pregnancy. *British journal of haematology.* 2012 Jul;158(1):3-15.
19. Chakravorty S, Roberts I. How I manage neonatal thrombocytopenia. *Br J Haematol.* 2012;156(2):155-162.

20. Baer VL, Lambert DK, Henry E, Christensen RD. Severe thrombocytopenia in the NICU. *Pediatrics.* 2009 Dec 1;124(6):e1095-100.
21. Sen A, Mahalanabis D, Singh AK, Som TK, Bandyopadhyay S. Impact of a district level sick newborn care unit on neonatal mortality rate: 2-year follow-up. *Journal Of Perinatology.* 2009 Feb;29(2):150-5.
22. Sola-Visner M, Sallmon H, Brown R. New insights into the mechanisms of nonimmune thrombocytopenia in neonates. In *Seminars in Perinatology* 2009 Feb 1 (Vol. 33, No. 1, pp. 43-51). WB Saunders.
23. Krishnan J, Morrison W, Simone S, Ackerman A. Implications of thrombocytopenia and platelet course on pediatric intensive care unit outcomes. *Pediatric Critical Care Medicine.* 2008 Sep 1;9(5):502-5.