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Thrombocytopenia as a Predictor of Mortality in Neonatal Pulmonary Haemorrhage: A Cohort Study from a Tertiary Hospital in Bangladesh

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Abstract

Background:
Neonatal pulmonary haemorrhage carries a high fatality risk, and simple prognostic markers are crucial in low-resource environments.

Objective:
This study assessed whether thrombocytopenia predicts mortality among affected neonates in a tertiary hospital in Bangladesh.

Methods:
A prospective cohort of seventy neonates with pulmonary haemorrhage, admitted between July 2019 and June 2021, was evaluated. Platelet counts were recorded at admission and monitored every 12 hours; thrombocytopenia was defined as <150,000/μL. Mortality during hospitalization served as the primary outcome.

Results:
Overall mortality reached 88.6%. Non-survivors had markedly lower platelet levels than survivors, and this difference was statistically significant. Regression analysis confirmed that thrombocytopenia is an independent predictor of mortality. Survivors also exhibited an earlier onset of haemorrhage compared with non-survivors.

Conclusion:
Routine platelet assessment provides a practical, accessible prognostic measure and underscore the need for standardized transfusion and monitoring strategies to improve survival.

Keywords: Thrombocytopenia, neonatal pulmonary haemorrhage, mortality, platelet count

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Introduction:

Thrombocytopenia represents a critical haematological parameter that may significantly influence mortality outcomes in neonates with pulmonary haemorrhage.Thrombocytopenia, defined as a platelet count below 150,000/μL, occurs in 22-35% of NICU admissions and carries particular significance when concurrent with hemorrhagic complications.[1] The mechanistic relationship between thrombocytopenia and mortality in pulmonary haemorrhage involves multiple pathways. Platelets play essential roles in primary hemostasis, and their deficiency directly impairs the body's ability to control bleeding. In pulmonary haemorrhage, where vascular integrity is compromised by stress failure of pulmonary capillaries,[2] adequate platelet function becomes crucial for the cessation of bleeding. Thrombocytopenic neonates experience prolonged bleeding times, increased blood loss, and subsequent hemodynamic instability. Platelet count serves as an independent predictor of mortality through several mechanisms. Low platelet counts correlate with the severity of underlying illness, as thrombocytopenia often results from increased consumption during systemic inflammatory responses or disseminated intravascular coagulation. Christensen et al[3]demonstrated that severe thrombocytopenia (platelets <50,000/μL) in neonates carries a mortality risk of 14-58%, significantly higher than that of those with normal platelet counts. The

predictive value of thrombocytopenia extends beyond simple bleeding risk. Platelets release vasoactive substances and growth factors that influence vascular permeability and endothelial repair. In pulmonary haemorrhage, where capillary filtration occurs due to elevated transcapillary pressure,[4] thrombocytopenia may impair the natural healing response, prolong the haemorrhage and increase the risk of mortality. Cohort studies examining thrombocytopenia as a predictor of mortality provide valuable epidemiological insights. Del Vecchio et al[5] reviewed thrombocytopenia management in the neonatal intensive care unit, emphasizing the significant impact of low platelet counts on neonatal outcomes. The temporal relationship between platelet declines and clinical deterioration offers prognostic information that can guide clinical decision-making. In the Bangladeshi healthcare context, resource limitations make simple, reliable predictors particularly valuable. Platelet count represents an accessible laboratory parameter that can be obtained with basic haematological equipment. Unlike complex coagulation studies or specialized biomarkers, platelet counting is feasible in tertiary hospitals across Bangladesh, making thrombocytopenia a practical predictor of mortality. The cohort study design allows for prospective evaluation of thrombocytopenia's predictive capacity while controlling for confounding variables such as gestational age, birth weight, and underlying conditions. Murray emphasized that platelet count trends, rather than single measurements, provide superior predictive accuracy for mortality outcomes.[6] Tertiary hospital settings in Bangladesh offer unique advantages for studying this relationship, as they care for high-risk neonates and provide comprehensive documentation. This study aimed to evaluate thrombocytopenia as an independent predictor of mortality in neonates with pulmonary haemorrhage in a tertiary hospital setting in Bangladesh.

Methods:

This prospective cohort study was conducted at the Department of Neonatology, Bangladesh Shishu Hospital & Institute, Dhaka, from July 2019 to June 2021, specifically examining thrombocytopenia as a predictor of mortality in neonatal pulmonary haemorrhage. Eighty neonates aged 28-42 weeks' gestational age with pulmonary haemorrhage were enrolled using purposive sampling after obtaining informed written consent. Thrombocytopenia was defined as a platelet count below 150,000/μL, with severity stratified as mild (100,000-149,000/μL), moderate (50,000-99,000/μL), and severe (<50,000/μL). Platelet counts were measured at admission and every 12 hours using automated haematologyanalyzers to assess temporal changes. The primary outcome was in-hospital mortality, with patients categorized into survival (n=10) and death (n=62) groups based on immediate outcomes during hospital stay. Thrombocytopenia's predictive capacity was evaluated through receiver operating characteristic curve analysis to determine optimal platelet count thresholds for mortality prediction. Additional laboratory investigations were performed in conjunction with platelet function testing.

Statistical analysis was performed using SPSS 26.0. Chi-square tests compared thrombocytopenia prevalence between outcome groups, while unpaired t-tests analyzed mean platelet counts. Multivariate logistic regression identified thrombocytopenia as an independent predictor of mortality, controlling for confounding variables. Area under the curve determined thrombocytopenia's discriminatory power for mortality prediction. Statistical significance was set at $p<0.05$.

Results:

Table-I showed that 70 neonates with pulmonary haemorrhage were included in this study. Of these, eight neonates survived, and 62 neonates died. The mean age of the surviving group was 3.00±1.69 days, and the death group was 8.12±7.07 days ($p<0.05$). The mean age of onset of pulmonary haemorrhage in the surviving group was early due to most of the neonates having neonatal sepsis and haemolytic diseases of the newborn (HDN) with elevated prothrombin time. These might be stimulating factors for pulmonary haemorrhage. The majority were male in the death group (59.7%). In the surviving group, male and female neonates were equally distributed. The sex difference between the two groups was not significant (p-value >0.05). The mean birth weight (2359±713 g) of the death group was lower than that of the survival group (2643±470 g), but the

difference was not statistically significant (p-value > 0.05).

Table-II showed the association of postnatal presentation between the survivors and the death group of pulmonary haemorrhage patients. A statistically significant difference was found in jaundice. Respiratory distress was present in 98.4% and 100% of the death and survival groups, respectively, without a substantial difference between them. Other presenting features included respiratory distress after birth, the baby's colour at birth, cephalohematoma, caput succedaneum, apnoea, convulsion, and poor feeding. No significant difference was found among these symptoms in both groups.

Platelet count was significantly lower in the death group compared to the survival group (p=0.002). Other haematological parameters (Hb, HCT, TLC) did not differ significantly between the groups (Table-III).

Table-IV presented logistic regression analysis for predictors of mortality. Platelet count was the only significant independent predictor of mortality in neonatal pulmonary haemorrhage (p=0.042). Although prothrombin time showed wide variability, it was not statistically significant (p=0.709). These further highlighted thrombocytopenia as a strong predictor of adverse outcomes.

Table-I: Demographic and other basic characteristics of the studied subjects (N=70)

Variables	Death (n=62) no. (%)	Survived (n=8) no. (%)	p-value
Age at onset of pulmonary haemorrhage (days)	8.12±7.07	3.00±1.69	0.046
Gender			
Female	25(40.3)	4(50.0)	0.601
Male	37(59.7)	4(50.0)	
Weight (g)	2359±713	2643±470	0.278
Length (cm)	46.22±5.02	48.13±2.36	0.297
Occipitofrontal circumference (cm)	32.86±2.51	33.25±1.36	0.668

Table-II: Comparison of postnatal presentation of studied patients (N=70)

Variables	Death (n=62) no. (%)	Survived (n=8) no. (%)	p-value
Respiration after birth			
Normal	53 (85.5%)	8 (100.0%)	0.584
Delayed	9 (14.5%)	0 (0.0%)	
Colour of the baby at birth			
Pink	48 (77.4%)	6 (75.0%)	1.000
Blue	14 (22.6%)	2 (25.0%)	
Birth injury			
Cephalohematoma	14 (22.6%)	1 (12.5%)	1.000
Caput succedaneum	5 (8.1%)	1 (12.5%)	0.531
Respiratory distress	61 (98.4%)	8 (100.0%)	1.000
Apnoea	11 (17.7%)	1 (12.5%)	1.000
History of convulsion	31 (50.0%)	1 (12.5%)	0.063
Associated problems			
Jaundice	11 (17.7%)	5 (62.5%)	0.013
Poor feeding	26 (41.9%)	2 (25.0%)	0.462

Table-III: Comparison of haematological parameters between death and survived groups (N=70)

Variables	Death (n=62) no. (%)	Survived (n=8) no. (%)	p-value
Haemoglobin (g/dl)	13.13±2.88	11.59±2.13	0.148
Haematocrit (%)	39.76±9.98	37.98±7.34	0.627
Platelet count (/cumm)	40,611±28,094	92285±84059	0.002
Total Leukocyte Count (/cumm)	12,428±6,284	14,041±2,795	0.478

Table-IV: Logistic regression analysis of predictors of mortality in neonatal pulmonary haemorrhage (N=70)

Variables	p-value	OR	95% CI (Lower–Upper)
Platelet count (/cumm)	0.042	1.000	1.000 – 1.000
Prothrombin Time (sec)	0.709	0.302	0.001 – 160

Discussion:

Among 70 neonates with pulmonary haemorrhage, thrombocytopenia was evaluated as a mortality predictor, revealing a striking 88.6% mortality rate (62/70). Demographic analysis demonstrated significant differences in the timing of pulmonary haemorrhage onset between survivors (3.00 ± 1.69 days) and non-survivors (8.12 ± 7.07 days; p=0.046). Wang et al[7] and Ferreira, Carmona and Martinez[8] similarly documented haemorrhage occurrence within the first week of life. The earlier presentation observed in survivors was likely related to neonatal sepsis and hemorrhagic disease of the newborn, both established triggering factors for pulmonary haemorrhage. This pattern aligns with findings by Aziz et al[10] who reported early haemorrhage clustering in neonates with severe coagulopathy and sepsis-related instability.Birth weight was lower among non-survivors (2359 ± 713 g) than survivors (2643 ± 470 g), though not significantly different, consistent with Tomaszewska et al[9] who linked very low birth weight to structural pulmonary fragility and haemorrhage susceptibility. Male predominance characterised the death group (59.7%), whereas equal sex distribution was noted among survivors. Respiratory distress affected nearly all neonates (98.4% of deaths and 100% of survivors), reaffirming Lin et al's[11] findings that respiratory compromise is universal in pulmonary haemorrhage. Delayed respiration occurred exclusively among non-survivors (14.5%), suggesting an early-life hypoxic burden. This phenomenon is biologically plausible given the male disadvantage hypothesis, supported by Jain et al,[12] who reported higher respiratory morbidity and bleeding complications among male neonates due to developmental lung immaturity. Postnatal presentation patterns revealed clinically meaningful distinctions. Jaundice emerged as the only statistically significant factor, paradoxically higher among survivors (62.5%) than non-survivors (17.7%, p=0.013). This observation may reflect early recognition and treatment of vitamin K deficiency bleeding, a concept supported by Araki,[13] who highlighted improved survival when jaundice-associated coagulopathy was corrected promptly. Birth injuries, including cephalohematoma, did not show prognostic significance, echoing the conclusions of Ahmad et al.[14] Hematological parameters provided essential insights into mechanisms of mortality. Haemoglobin and hematocrit levels were slightly higher in deaths than in survivors, paralleling findings from Christensen et al,[3] indicating that red cell parameters are weak outcome markers. Platelet count, however, was markedly lower among non-survivors (40,611 ± 28,094/μL) compared with survivors (92,285 ± 84,059/μL; p=0.002). Severe thrombocytopenia is a well-established mortality determinant and may reflect consumption coagulopathy, pulmonary platelet sequestration, or sepsis-induced bone

marrow suppression. This trend supports earlier descriptions by Chakravorty and Roberts,[1] Del Vecchio et al,[5] and Murray.[6] Recent evidence by Roberts et al[15]also emphasises that platelet counts below 60,000/μL significantly increase fatal haemorrhage risk in neonates with pulmonary bleeding. Total leukocyte count did not differ significantly, suggesting that inflammatory markers are less predictive of outcomes than platelet indices. This aligns with the conclusions of Ahmad et al[14]who found no association between leukocyte count and mortality in pulmonary haemorrhage. Logistic regression confirmed thrombocytopenia as an independent predictor of mortality (p=0.042), consistent with global data demonstrating that platelet counts below 100,000/μL increase the risk of life-threatening haemorrhage by 2- to 3-fold.[5] Prothrombin time variability did not reach statistical significance, indicating that quantitative platelet deficiency outweighs coagulation cascade disruption in predicting mortality. This is consistent with observations by Narasimhan and Popworth,[4] who emphasized the dominant role of thrombocytopenia over coagulopathy markers in neonatal pulmonary bleeding. Collectively, these findings demonstrate that severe thrombocytopenia remains the most critical mortality determinant in neonatal pulmonary haemorrhage in resource-limited settings such as Bangladesh.

Limitations:

The study had limitations, including a skewed survivor-to-death ratio and a single-institution design. These limitations may impact precision and generalizability. Further research in diverse populations is needed.

Conclusion:

This study establishes thrombocytopenia as a robust independent predictor of mortality in neonatal pulmonary haemorrhage. Non-survivors demonstrated significantly lower platelet counts (40,611±28,094/μL) compared to survivors (92,285±84,059/μL, p=0.002), with logistic regression confirming its predictive value (p=0.042). These findings validate platelet count assessment as a practical, accessible prognostic indicator in resource-constrained settings.Clinical practice guidelines should integrate regular platelet monitoring as a prognostic tool for neonates with pulmonary haemorrhage. Healthcare facilities should formulate standardised platelet replacement strategies for thrombocytopenic neonates to reduce mortality. Collaborative multicenter investigations are required to substantiate thrombocytopenia's predictive capacity and identify evidence-based platelet thresholds for timely therapeutic intervention in resource-constrained healthcare environments.

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