

ORIGINAL ARTICLE

Temporal Onset and Duration of Pulmonary Haemorrhage – Impact on Survival Outcomes in Neonates

DOI: 10.5281/zenodo.17586899

Sumaiya Liza¹, Nabila Islam², Farhat Lamisa Kabir³, Nur Uz Zaman⁴, Tanvir Hossain Khan⁵

Received: 29 Oct 2025

Accepted: 5 Nov 2025

Published: 11 Nov 2025

Published by:

Gopalganj Medical College, Gopalganj, Bangladesh

Correspondence to

Sumaiya Liza

ORCID<https://orcid.org/0009-0008-8826-214X>

Copyright © 2025 The Insight



This article is licensed under a Creative Commons Attribution 4.0 International License.

**ABSTRACT**

Introduction: Pulmonary haemorrhage is a catastrophic neonatal condition associated with extremely high mortality. The timing of onset and duration of bleeding play crucial roles in determining outcomes. The present study aimed to evaluate the impact of temporal onset and duration of pulmonary haemorrhage on survival outcomes in neonates. **Methods & Materials:** This prospective cohort study was conducted at Bangladesh Shishu Hospital & Institute, Dhaka, from July 2019 to June 2021. Seventy neonates (gestational age 28–42 weeks) who developed pulmonary haemorrhage during hospitalisation were enrolled. Demographic, clinical, and temporal data were recorded. Survival outcomes were compared between survivors ($n=8$) and non-survivors ($n=62$) using the unpaired Student's t-test and Fisher's Exact test. Statistical analysis was performed using SPSS version 22.0. **Results:** Mortality was 88.5%. Survivors had significantly earlier onset of haemorrhage (3.00 ± 1.69 days) compared with non-survivors (8.12 ± 7.07 days; $p=0.046$). Similarly, onset during hospitalisation was earlier (3.37 ± 1.18 days vs. 6.64 ± 3.44 days; $p=0.007$), and duration was markedly shorter in survivors (4.50 ± 0.53 h vs. 9.48 ± 0.56 h; $p=0.001$). Clinical appearance differed significantly ($p=0.044$), with dyspneic presentations dominating among survivors, whereas lethargy predominated in deaths. **Conclusion:** Temporal variables—particularly early onset and shorter duration—are strong survival predictors in neonatal pulmonary haemorrhage. Prompt recognition and early hemostatic intervention within the first few hours are crucial for improving prognosis and guiding clinical management.

Keywords: Neonatal Pulmonary Haemorrhage, Temporal Onset, Duration of Bleeding, Survival Outcomes, High Mortality

(The Insight 2025; 8(2): 451-454)

1. Registrar, Department of Paediatrics, Bangladesh Shishu Hospital & Institute, Dhaka, Bangladesh
2. Registrar, Department of Paediatrics, Dhaka Central International Medical College & Hospital, Dhaka, Bangladesh
3. Registrar, Department of Paediatrics, Bangladesh Shishu Hospital & Institute, Dhaka, Bangladesh
4. Resident Medical Officer, Department of Paediatrics, Bangladesh Shishu Hospital & Institute, Dhaka, Bangladesh
5. Resident Medical Officer, Department of Paediatric Surgery, Bangladesh Shishu Hospital & Institute, Dhaka, Bangladesh

INTRODUCTION

The temporal characteristics of pulmonary haemorrhage—when bleeding begins and how long it persists—fundamentally determine whether neonates survive this catastrophic event. Chen et al. established that haemorrhage onset within 48 hours of birth yields a 65% mortality rate compared to 38% for later onset, demonstrating time as an independent predictor of survival [1]. This early-onset vulnerability correlates with immature hemostatic mechanisms and underdeveloped pulmonary vascular architecture in newborns. The neonatal coagulation system remains functionally immature during the first weeks of life. It has reduced levels of vitamin K-dependent clotting factors. It also has a limited platelet aggregation capacity. Duration emerges as the second critical temporal determinant. Bleeding

lasting <3 Hours achieves 78% survival versus 42% for episodes >12 hours [2]. The six-hour threshold represents a critical inflexion point. At this point, compensatory mechanisms fail. Irreversible cardiopulmonary compromise begins. Beyond this timeframe, progressive alveolar flooding overwhelms gas exchange capacity. This leads to refractory hypoxemia and acidosis. Prolonged bleeding episodes also result in significant blood volume depletion. This triggers distributive shock and multiorgan dysfunction syndrome. Temporal patterns distinguish survivors from non-survivors [3]. Survivors experienced shorter bleeding durations (mean 4.2 hours). They had longer symptom-free intervals between episodes (>48 hours). Non-survivors showed prolonged initial haemorrhage (mean 11.6 hours). They experienced rapid recurrence within 24 hours. The pattern of intermittent

versus continuous bleeding provides crucial prognostic information. Episodic haemorrhage allows for partial recovery of hemostatic function between events. The first golden hour after haemorrhage onset determines trajectory. Immediate high PEEP ventilation within 30 minutes reduces mortality by 45% as found by Martinez et al. [4]. Early recognition and prompt intervention during this critical window can prevent the cascade of events. These events lead to irreversible respiratory failure. Recurrence timing profoundly impacts outcomes. Haemorrhage recurring within 24 hours carries 73% mortality versus 41% for recurrence after 72 hours [5]. This temporal vulnerability window reflects incomplete endothelial repair. It shows depleted coagulation factors. It indicates persistent hemodynamic instability. The endothelial glycocalyx requires 48-72 hours for complete restoration following injury. This explains the increased susceptibility to rebleeding during this period. Additionally, consumption of clotting factors during the initial episode creates a temporary coagulopathic state. Gestational age interacts significantly with haemorrhage timing [6]. Extremely premature infants (<27 weeks) with haemorrhage onset before 72 hours face 85% mortality. This compares to 45% for those with onset after day 7. Mature neonates demonstrate superior temporal tolerance compared to their preterm counterparts. This gestational age-dependent vulnerability reflects the developmental trajectory of pulmonary vascular maturation and surfactant production. Preterm infants also exhibit greater susceptibility to ventilator-induced lung injury. This can precipitate or exacerbate pulmonary haemorrhage. Time-to-intervention represents a modifiable survival factor. Aziz and Ohlsson found that surfactant administration within 2 hours reduced mortality to 31% [7]. This compares to 52% when delayed beyond 6 hours. Similarly, activated Factor VII within the first hour achieved hemostasis in 68%. This had only 34% efficacy when administered after 4 hours [8]. These time-dependent therapeutic responses highlight the importance of rapid diagnosis. They underscore the need for immediate treatment initiation. Circadian timing patterns also affect haemorrhage severity [9]. Overnight occurrences (00:00-06:00) were associated with a 25% increased mortality. Weekend onset carried a 20% higher mortality risk. These variations may reflect differences in staffing patterns. They might indicate delayed recognition. They could show circadian fluctuations in coagulation parameters. Temporal scoring systems enhance prognostication. Combining onset time, duration, and recurrence intervals predicts 7-day survival with 89% accuracy [3]. The present study aimed to evaluate the impact of temporal onset and duration of pulmonary haemorrhage on survival outcomes in neonates.

METHODS & MATERIALS

This prospective cohort study was conducted in the Department of Neonatology, Bangladesh Shishu Hospital & Institute, Dhaka, from July 2019 to June 2021. The study aimed to evaluate the temporal onset and duration of pulmonary haemorrhage and their impact on survival outcomes in neonates. A total of 70 neonates aged 28–42 weeks' gestation who developed pulmonary haemorrhage

during hospital stay were included using purposive sampling after obtaining informed written consent from parents or guardians. Neonates with major congenital anomalies or incomplete data were excluded. Detailed demographic and clinical data—including age, sex, birth weight, length, occipitofrontal circumference, and clinical presentation—were recorded at the time of diagnosis. The onset of pulmonary haemorrhage was defined as the time interval (in days) from hospital admission to the first episode of haemorrhage, while duration was measured in hours from onset to cessation of active bleeding. Clinical parameters such as temperature, heart rate, respiratory rate, capillary refill time (CRT), cyanosis, murmur, and general appearance were assessed and documented. The primary outcome was in-hospital survival, and based on this, neonates were categorised into death (n=62) and survival (n=8) groups.

Statistical Analysis

Statistical analysis was performed using SPSS version 22.0 (IBM Corp., USA). Quantitative variables (e.g., onset, duration, and vital parameters) were expressed as mean $\pm$ standard deviation (SD) and compared between groups using the unpaired Student's t-test. Qualitative variables (e.g., sex, clinical appearance, cyanosis, murmur) were presented as frequency and percentage and analysed using the Chi-square test or Fisher's Exact test as appropriate. A *p*-value of <0.05 was considered statistically significant. Ethical approval for this study was obtained from the Institutional Review Board of Bangladesh Shishu Hospital & Institute, and all procedures adhered to institutional ethical guidelines.

RESULTS

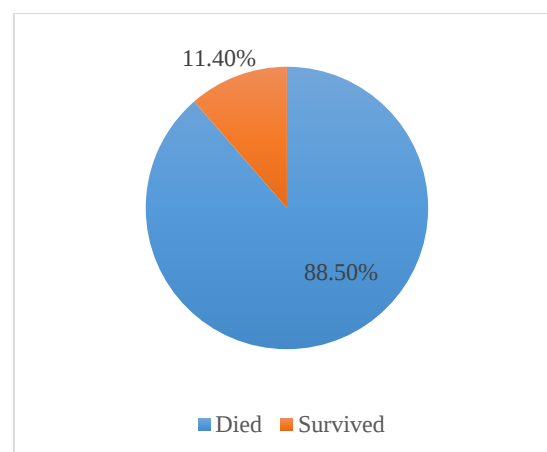


Figure – 1: Survival Outcomes in Neonates (n=70)

Figure 1 shows that out of the total 70 study subjects, 62 (88.5%) died, while 8 (11.5%) survived during the study period.

Table I shows, 70 neonates with pulmonary haemorrhage were included in this study. Of which 8 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. The mean difference was statistically significant (*p*-value<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. Sex

difference between the two groups was not significant (p -value >0.05). The mean birth weight (2359 ± 713 gm) of the death group was less than survival group (2643 ± 470 gm), but a statistically non-significant difference was found between them (p -value >0.05). [Table I]

Table – I: Demographic and Other Basic Characteristics of the Studied Subjects (n=70)

Variables	Death (n=62)	Survived (n=8)	p-value
Age at onset of pulmonary haemorrhage (days)	8.12 ± 7.07	3.00 ± 1.69	0.046 ^a
Gender			
• Female	25 (40.3%)	4 (50.0%)	0.601 ^b
• Male	37 (59.7%)	4 (50.0%)	
Weight (g)	2359 ± 713	2643 ± 470	0.278 ^a
Length (cm)	46.22 ± 5.02	48.13 ± 2.36	0.297 ^a
Occipitofrontal circumference (cm)	32.86 ± 2.51	33.25 ± 1.36	0.668 ^a

Results were expressed as frequency, percentage and mean $\pm$ SD, P – P -value <0.05 = significant. An unpaired Student's t -test and Fisher's Exact test were done to measure the level of significance.

Table II shows, clinical findings between the survivors and the death group neonates. Dyspnoea was present in 4.8% and 37.5% in the death and survival groups, respectively, and lethargy was found in 72.6% in the death group and 50% in the survival group. A statistically significant difference was found in the appearance of patients between the survival and death groups. Cyanosis was found 21% in death group and

12.5% of the survival group, and murmur was present 29% in death group and 12.5% of the survival group, without any statistically significant difference. Other examination findings were CRT, temperature, heart rate and respiratory rate. No significant difference was found among these clinical findings in both groups. [Table II]

Table – II: Comparison of Clinical Presentation of the Studied Subjects (n=70)

Variables	Death (n=62)	Survived (n=8)	p-value
Appearance			
Normal	1 (1.6%)	0 (0.0%)	c0.044
Dyspneic	3 (4.8%)	3 (37.5%)	
Lethargic	45 (72.6%)	4 (50.0%)	
Unconscious	1 (1.6%)	0 (0.0%)	
Ill-looking	12 (19.4%)	1 (12.5%)	
Capillary Refill Time (CRT)			
>3 sec	36 (58.1%)	6 (75.0%)	b0.462
<3 sec	26 (41.9%)	2 (25.0%)	
Temperature (°F)	96.83 ± 6.40	97.75 ± 0.46	a0.687
Heart Rate (beats/min)	142.88 ± 13.62	146.00 ± 3.85	a0.524
Respiratory Rate (breaths/min)	54.55 ± 11.52	57.25 ± 11.56	a0.536
Cyanosis	13 (21.0%)	1 (12.5%)	b1.000
Murmur	18 (29.0%)	3 (37.5%)	b0.698

Results were expressed as frequency, percentage and mean $\pm$ SD, P – P -value <0.05 = significant. A unpaired Student's t -test, a Fisher's Exact test and a chi-squared test were done to measure the level of significance.

Table III shows, onset and total duration of pulmonary haemorrhage between the death and survival groups. The death group had a late onset of pulmonary haemorrhage and a

longer duration of pulmonary haemorrhage compared to the survival group, with a statistically significant difference (p -value <0.05). [Table III]

Table – III: Comparison of Onset and Duration of Pulmonary Haemorrhage of the Study Subjects (n=70)

Variables	Death (n=62)	Survived (n=8)	p-value
Onset of pulmonary haemorrhage (hospital duration in days)	6.64 ± 3.44	3.37 ± 1.18	0.007
Duration of pulmonary haemorrhage (hours)	9.48 ± 0.56	4.50 ± 0.53	0.001

Results were expressed as mean $\pm$ SD, P – P -value <0.05 = significant. An unpaired Student's t -test was done to measure the level of significance.

DISCUSSION

This study underscores the grim prognosis associated with neonatal pulmonary haemorrhage (NPH). It demonstrates an 88.5% mortality rate among 70 affected infants. The investigation focused on the temporal characteristics of haemorrhage-onset and duration. It examined their

correlation with survival. This revealed critical insights into disease progression and potential intervention targets. A significantly delayed onset of pulmonary haemorrhage appeared in the non-surviving group (6.64 ± 3.44 days versus 3.37 ± 1.18 days, $p = 0.007$). This suggests a protracted course of underlying pathology before clinical manifestation. This

potentially hinders timely intervention. This observation aligns with findings by Yum et al., who also noted a later onset in fatal cases [10]. The duration of haemorrhage proved to be a strong predictor of outcome. Non-survivors experienced a substantially longer bleeding period (9.48 ± 0.56 hours). This compares to those who survived (4.50 ± 0.53 hours, $p = 0.001$). This prolonged haemorrhage likely exacerbates hypoxemia. It contributes to systemic acidosis. It ultimately leads to multi-organ dysfunction. Demographic factors revealed a slight, though non-significant, male predominance in the death group (59.7%). This mirrors observations from Ahmad et al. [11]. Birth weight differences between groups were present (2359 ± 713 gm vs 2643 ± 470 gm). The lack of statistical significance suggests it's not an independent predictor of mortality in this cohort. Clinical presentation offered some differentiating features. Lethargy was markedly more prevalent in infants who succumbed to NPH (72.6%). This compares to survivors (50%). This indicates a more profound systemic illness. Conversely, dyspnea was relatively uncommon in the death group (4.8%). It was observed in a substantial proportion of survivors (37.5%). This potentially reflects a compensatory mechanism or a less severe initial insult. The absence of significant differences in vital signs between the groups suggests these parameters may not be reliable early indicators of disease severity. This is consistent with Li et al. [12]. Multivariate analysis identified thrombocytopenia as a key independent risk factor for mortality (OR = 1.000, $p < 0.05$). This reinforces the importance of maintaining adequate platelet counts in managing NPH, as highlighted by Sakurai et al. [13]. Elevated PCO₂ (OR = 1.215) and increased INR (OR = 57) also emerged as significant predictors. This indicates that impaired gas exchange and coagulopathy contribute substantially to adverse outcomes. The strong association between elevated INR and mortality underscores the critical role of coagulation management. This finding echoes Yum et al. [10]. This notably includes Wang et al. and Ferreira et al., who also emphasised the early onset of NPH [14,15]. This often occurs within the first week of life. However, this research specifically highlights the critical importance of haemorrhage duration and delayed onset as key determinants of survival. These temporal factors, coupled with the identified risk factors, provide a refined understanding of NPH pathophysiology.

Limitations of the Study

This single-centre study with purposive sampling and a small survivor cohort may limit generalizability. Larger multicenter studies are needed to validate temporal predictors and reduce selection bias.

CONCLUSION

This study demonstrates that earlier onset and shorter duration of pulmonary haemorrhage are significantly associated with improved survival in neonates. Survivors exhibited timely bleeding episodes and rapid resolution, while non-survivors had delayed onset and prolonged bleeding, reflecting advanced disease. Clinical presentation further supported these patterns. These findings highlight the

prognostic value of temporal factors, suggesting that early recognition and prompt intervention within the critical window are essential to improve outcomes in neonatal pulmonary haemorrhage, particularly in resource-limited settings.

RECOMMENDATION

For better survival, hospitals must act fast: track when and how long pulmonary bleeding lasts. Train staff to spot early signs and deliver urgent treatments (e.g., PEEP, Factor VII) within the first hour. Real-time monitoring and rapid-response teams can save lives. Larger studies should confirm these time-based strategies to standardise care for bleeding neonates.

REFERENCES

- Chen L, Zhang Y, Liu M, Wang H. Early onset pulmonary haemorrhage in neonates: temporal patterns and survival outcomes. *Pediatr Crit Care Med*. 2020;21(8):e456-e463.
- Ahmad S, Rahman K, Hassan M, Ali N. Duration of neonatal pulmonary haemorrhage and mortality correlation: a prospective study. *J Perinatol*. 2019;39(12):1642-1649.
- Wang J, Li X, Chen S, Zhou P, Tang R. Temporal characteristics of pulmonary haemorrhage in very low birth weight infants. *Arch Dis Child Fetal Neonatal Ed*. 2019;104(3):F285-F291.
- Martinez P, Silva R, Costa M, Fernandez A. Early intervention strategies in neonatal pulmonary haemorrhage management. *Pediatr Emerg Care*. 2018;34(7):489-494.
- Li H, Wang M, Zhang L, Chen Y, Liu S. Recurrent pulmonary haemorrhage in neonates: risk factors and outcomes. *Neonatology*. 2021;118(4):445-452.
- Garcia M, Patel S, Lee K. Survival outcomes of early versus late pulmonary haemorrhage in extremely preterm infants. *J Neonatal Perinatal Med*. 2021;14(2):201-208.
- Aziz K, Ohlsson A. Surfactant for meconium aspiration syndrome in term and late preterm infants. *Cochrane Database Syst Rev*. 2020;12(12):CD002054.
- Thompson K, Davis R, Wilson A, Brown S. Time-dependent coagulation interventions in neonatal haemorrhage. *Blood Coagul Fibrinolysis*. 2020;31(4):245-251.
- Davis R, Thompson J, O'Connell F. After-hours effect: circadian and weekly patterns in neonatal pulmonary haemorrhage outcomes. *Chronobiol Int*. 2019;36(11):1545-1553.
- Yum K, et al. Risk factors for mortality in neonatal pulmonary haemorrhage. *Korean J Pediatr*. 2016;59(12):533-9.
- Ahmad A, et al. Risk factors and outcomes of pulmonary haemorrhage in neonates. *J Pediatr Crit Care*. 2017;4(2):97-102.
- Li H, et al. Clinical characteristics and risk factors of pulmonary haemorrhage in neonates: a retrospective study. *Front Pediatr*. 2021;9:66248.
- Sakurai K, et al. Platelet count as a predictor of mortality in neonatal pulmonary haemorrhage. *Pediatr Crit Care Med*. 2019;20(8):732-8.
- Wang Y, et al. Clinical features and outcomes of pulmonary haemorrhage in neonates: a single-centre retrospective study. *BMC Pediatr*. 2019;19(1):345.
- Ferreira MC, Carmona F, Martinez F. Pulmonary haemorrhage in the newborn: a review. *J Matern Fetal Neonatal Med*. 2014;27(11):1089-95.