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Article

Correlation Between Neonatal Sepsis and Hyperbilirubinemia in Hospitalized Newborns

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Abstract

Background: *Neonatal sepsis and hyperbilirubinemia are leading causes of morbidity in NICUs, especially in low-resource settings. This study aimed to evaluate the correlation between laboratory-confirmed sepsis and elevated serum bilirubin in hospitalized neonates.*

Methods: *A retrospective observational study was conducted on 90 neonates admitted to the NICU at Bangladesh Shishu Hospital and Institute between January 2023 and December 2024. Data on sepsis markers, bilirubin levels, phototherapy, and clinical outcomes were analyzed using descriptive statistics, correlation analysis, and logistic regression.*

Results: *Among the 90 neonates, 73.33% had sepsis, 42.22% developed jaundice, and 67.78% required phototherapy. Septic neonates had significantly higher mean serum bilirubin levels (10.5 ± 2.7 mg/dL) compared to non-septic neonates (8.2 ± 2.1 mg/dL; $p = 0.036$). Bilirubin showed significant positive correlations with CRP ($r = 0.38$; $p = 0.002$) and blood culture positivity ($r = 0.44$; $p = 0.001$). Sepsis independently predicted hyperbilirubinemia (AOR = 2.81; $p = 0.045$). Among culture-positive cases, Pseudomonas and Acinetobacter infections were associated with the highest bilirubin levels. Jaundiced neonates also exhibited significantly higher rates of RDS, hypoglycemia, and feeding intolerance.*

Conclusion: *Sepsis significantly contributes to hyperbilirubinemia in neonates. Early recognition, bilirubin monitoring, and infection-specific management may improve outcomes in this vulnerable population*

Introduction

Neonatal sepsis and hyperbilirubinemia are two of the most prevalent, life-threatening, yet potentially reversible conditions encountered in the first 28 days of life. Together, they account for a substantial share of neonatal morbidity globally. According to the World Health Organization, approximately 5 million neonatal deaths occur each year, with sepsis and meningitis contributing to a significant proportion of these fatalities[1]. Simultaneously, neonatal jaundice, marked by elevated serum bilirubin levels, is observed in up to 60% of term and 80% of preterm infants worldwide[2]. Though often physiological and self-limiting, hyperbilirubinemia, if unmonitored or untreated, can lead to kernicterus, a preventable cause of cerebral palsy, sensorineural hearing loss, and other neurodevelopmental impairments[3]. These two conditions, while distinct in etiology, often intersect in clinical settings, especially within resource-constrained neonatal intensive care units (NICUs), complicating early detection and targeted management strategies. Neonatal sepsis is broadly defined as a systemic inflammatory response to infection, confirmed by clinical signs and supported by laboratory parameters such as elevated C-reactive protein (CRP), abnormal white cell counts, and positive blood cultures[4]. It is typically categorized into early-onset sepsis (EOS), occurring within the first 72 hours of life, and late-onset sepsis (LOS), occurring thereafter. Globally, the incidence of neonatal sepsis ranges between 1 to 8 per 1,000 live births, with disproportionately higher rates in low- and middle-income countries (LMICs), including South Asia and sub-Saharan Africa[5]. A recent systematic review identified prematurity, prolonged rupture of membranes (PROM), invasive procedures such as catheterization and mechanical ventilation, and maternal genitourinary infections as key risk factors for sepsis[6]. These same risk factors are also prevalent in Bangladeshi NICUs, underscoring the urgency for regional data to inform care standards. Hyperbilirubinemia in neonates results from an imbalance between bilirubin production and clearance. Physiologically, neonatal red blood cells have a shorter lifespan and higher turnover, contributing to increased bilirubin production. At the same time, hepatic immaturity impairs the conjugation and excretion of bilirubin. According to American Academy of Pediatrics (AAP) guidelines, serum bilirubin levels exceeding the 95th percentile on an age-

specific nomogram necessitate clinical intervention, usually through phototherapy or, in severe cases, exchange transfusion[7]. If untreated, severe hyperbilirubinemia can progress to bilirubin-induced neurological dysfunction (BIND) and kernicterus, with devastating lifelong consequences[8,9]. There exists a strong biological rationale linking sepsis and hyperbilirubinemia in neonates. Inflammatory cytokines such as TNF-α and IL-1β, which are released during sepsis, impair hepatic conjugation and biliary excretion of bilirubin[10]. Sepsis-induced hepatocellular dysfunction and cholestasis further reduce the liver's capacity to process bilirubin[11]. Additionally, sepsis can trigger hemolysis—either directly through bacterial toxins or indirectly via immune mechanisms—further increasing bilirubin production[12]. Moreover, alterations in albumin binding due to acidosis or competition from inflammatory molecules exacerbate the risk of bilirubin crossing the blood-brain barrier[9]. Notably, hyperbilirubinemia may itself mask early signs of sepsis, such as lethargy or temperature instability, resulting in delayed recognition and initiation of antibiotics[11]. Although international studies have explored the co-occurrence of sepsis and elevated bilirubin, they are limited in number and often methodologically constrained. Das et al. (2016), in a single-center study of 41 neonates, reported significantly elevated levels of conjugated and delta bilirubin in septic compared to non-septic neonates[1]. Similarly, a retrospective Ethiopian cohort study involving 328 NICU neonates found that sepsis was the leading comorbid factor (66.9%) among those with hyperbilirubinemia[2]. However, these studies are primarily descriptive, lack multivariable adjustment, and are often constrained by small sample sizes. Hansen et al. even proposed that physiological hyperbilirubinemia might have protective antibacterial effects, although this hypothesis remains speculative and unsupported by clinical outcomes[13]. Critically, there remains a stark absence of published data from Bangladeshi NICUs evaluating the quantitative association between laboratory-confirmed sepsis and serum bilirubin levels. No known studies have investigated whether septic neonates are at higher risk of requiring phototherapy or progressing to bilirubin-related complications within this population. Given the high burden of multidrug-resistant Gram-negative infections (e.g., Klebsiella, Acinetobacter) in Bangladeshi hospitals, and frequent delays in diagnostic turnaround and therapeutic intervention, this data gap is not merely academic but has tangible consequences for clinical prioritization and resource allocation. This study aims to fill this important knowledge gap by evaluating the correlation between neonatal sepsis and hyperbilirubinemia in a cohort of NICU-admitted neonates in Bangladesh. By analyzing serum bilirubin levels, phototherapy requirements, and sepsis biomarkers in a defined hospital population, the study seeks to generate actionable insights that can inform integrated screening and intervention strategies for high-risk neonates in similar LMIC settings.

Methods

This retrospective observational study was conducted in the Neonatal Intensive Care Unit (NICU) of Bangladesh Shishu Hospital and Institute, Dhaka, Bangladesh, from January 2023 to December 2024. We screened 124 neonates aged 0–28 days admitted to the NICU during this period. Inclusion criteria required complete medical records documenting:[1] sepsis workup (blood culture, C-reactive protein [CRP], complete blood count),[2] ≥2 bilirubin measurements within 72 hours of admission, and[3] phototherapy details if applicable. Exclusion criteria comprised neonates with major congenital anomalies (e.g., cardiac/neural tube defects), hemolytic disorders (Rh incompatibility/G6PD deficiency), or incomplete data. After exclusions (n=34; 27 for incomplete records, 7 for hemolysis), 90 neonates were enrolled. Data were extracted from electronic health records by two independent pediatricians, with discrepancies resolved by a third reviewer. Variables included demographics (gestational age, sex), sepsis markers (culture results, CRP, WBC), peak bilirubin within 24 hours of admission, phototherapy use, and complications (RDS, IVH, NEC, MODS, hypoglycemia, feeding intolerance). Statistical analysis was conducted using SPSS v27.0. Descriptive statistics (frequencies, mean ± SD) summarized baseline characteristics. Group comparisons employed chi-square/Fisher's exact tests for categorical variables and independent t-tests for continuous data. Pearson's correlation assessed bilirubin-sepsis marker relationships. Multivariable logistic regression (adjusted for gestational age and CRP) identified hyperbilirubinemia predictors. Significance was set at $p < 0.05$.

Results

Table 1: Baseline characteristics of the study population (N=90)

Variable	Frequency (n)	Percentage
Sex: Male	54	60.00%
Sex: Female	36	40.00%
Preterm (<37 weeks)	69	76.67%
Term (≥37 weeks)	21	23.33%
Birth Weight (grams) (Mean ± SD)	**1880 ± 420**	
Sepsis Present	66	73.33%
Sepsis Absent	24	26.67%
Jaundice Diagnosed	38	42.22%
No Jaundice	52	57.78%
Phototherapy Required	61	67.78%
Phototherapy Not Required	29	32.22%

Table 1 represents the baseline characteristics of the study population. Among the 90 neonates included in the study, 60% were male and 40% were female. A majority (76.67%) were preterm, while 23.33% were term. The mean birth weight was 1880 ± 420 grams. Sepsis was diagnosed in 73.33% of

neonates. Jaundice was observed in 42.22% of the cases, and 67.78% of neonates required phototherapy during the course of admission.

Table 2: Hyperbilirubinemia in Sepsis vs No Sepsis (N=90)

Parameter	Sepsis (n = 66)	No Sepsis (n = 24)	p-value
Mean Serum Bilirubin within 24 hours (mg/dL)	10.5 ± 2.7	8.2 ± 2.1	0.036
Jaundice Present	29 (43.94%)	10 (41.67%)	0.411
Phototherapy Required	51 (77.27%)	10 (41.67%)	0.48

- Percentages are calculated on the number of Sepsis present/absent

Table 2 denotes the hyperbilirubinemia in patients with sepsis and non-sepsis. Neonates with sepsis exhibited significantly higher mean serum bilirubin levels within 24 hours of admission compared to those without sepsis (10.5 ± 2.7 mg/dL vs. 8.2 ± 2.1 mg/dL; p = 0.036). The proportion of neonates with jaundice was comparable between septic and non-septic groups (43.94% vs. 41.67%; p = 0.411), although phototherapy was more frequently required in septic neonates (77.27% vs. 41.67%; p = 0.48), though this difference did not reach statistical significance.

Table 3: Correlation Between Bilirubin and Sepsis Markers (N=90)

Marker	Correlation Coefficient (r)	p-value
CRP (mg/L)	0.38	0.002
WBC Count	0.21	0.071
Positive Blood Culture	0.44	0.001

Table 3 signifies the correlation between bilirubin and sepsis markers. A moderate positive correlation was observed between serum bilirubin levels and CRP (r = 0.38, p = 0.002), while a stronger correlation was noted with blood culture positivity (r = 0.44, p = 0.001). No statistically significant correlation was found between bilirubin and total WBC count (r = 0.21, p = 0.071).

Table 4 represents the logistic regression of the predictors of hyperbilirubinemia. Logistic regression analysis revealed that sepsis was an independent predictor of hyperbilirubinemia (Adjusted OR = 2.81; 95% CI: 1.02–7.65; p = 0.045). Other variables such as prematurity, low birth weight (<1500 g), and elevated CRP (>10 mg/L) showed positive associations with hyperbilirubinemia.

Table 4: Logistic Regression - Predictors of Hyperbilirubinemia (N=90)

Variable	Adjusted OR	95% CI	p-value
Sepsis (yes vs no)	2.81	1.02–7.65	0.045
Preterm status	1.65	0.78–3.51	0.192
Birth Weight (<1500g)	2.13	0.91–5.04	0.081
CRP >10 mg/L	1.92	0.85–4.33	0.12

Table 5 demonstrates the blood culture positivity versus jaundice. Jaundice was significantly more prevalent among neonates with positive blood cultures compared to those with negative cultures (84.8% vs. 17.5%; p < 0.001), indicating a strong association between confirmed infection and bilirubin elevation.

Table 5: Blood Culture Positivity vs Jaundice (N=90)

Blood Culture Result	Jaundice Present (n-38)	Jaundice Absent (n-52)	p-value
Positive (n=33)	28	5	<0.001
Negative (n=57)	10	47	

Table 6 illustrates bilirubin levels addressing the organism. Among neonates with positive blood cultures, the highest mean serum bilirubin levels at 24 hours were observed in those infected with Pseudomonas (11.1 mg/dL), followed by Acinetobacter (10.9 mg/dL), Klebsiella (10.7 mg/dL), and E. coli (9.5 mg/dL).

Table 6: Bilirubin Levels by Organism (n=33)

Organism	n	Mean Bilirubin at 24h (mg/dL)
Acinetobacter	20	10.9
Klebsiella	7	10.7
E. coli	5	9.5
Pseudomonas	1	11.1

Table 7 shows common neonatal sepsis complications in jaundiced vs. non-jaundiced neonates. Neonates with jaundice had a higher incidence of respiratory distress syndrome (RDS) (37% vs. 19%; p = 0.04), hypoglycemia (47% vs. 19%; p = 0.005), and feeding intolerance (42% vs. 13%; p = 0.003). Other complications, including IVH, NEC, and MODS, did not show statistically significant differences between jaundiced and non-jaundiced groups.

Table 7: Common Neonatal Sepsis Complications in Jaundiced vs. Non-Jaundiced Neonates (N=90)

Complication	Jaundice Present (n=38)	Jaundice Absent (n=52)	p-value	OR (95% CI)
Respiratory Distress Syndrome (RDS)	14 (37%)	10 (19%)	0.04	2.6 (1.0–6.8)
Intraventricular Hemorrhage (IVH)	2 (5%)	5 (10%)	0.45	0.5 (0.1–2.6)
Hypoglycemia	18 (47%)	10 (19%)	0.005	3.8 (1.5–9.5)
Feeding Intolerance	16 (42%)	7 (1.3%)	0.003	3.7 (1.6–8.8)
Necrotizing Enterocolitis (NEC)	3 (8%)	4 (8%)	1	1.0 (0.2–4.9)
Multiple Organ Dysfunction Syndrome (MODS)	6 (16%)	3 (6%)	0.16	3.0 (0.7–12.6)

Discussion

This retrospective study examined the correlation between neonatal sepsis and hyperbilirubinemia in a cohort of 90 hospitalized neonates, revealing a statistically and clinically significant association between systemic infection and elevated bilirubin levels. The predominance of preterm births (76.67%) and low birth weight neonates (mean 1880 ± 420 g) reflects the high vulnerability of the study population to both sepsis and jaundice, which aligns with prior reports from similar low-resource neonatal intensive care units (NICUs)[2,14]. Sepsis was diagnosed in approximately three-fourths of the neonates, and among them, 43.94% developed jaundice, compared to 41.67% in non-septic neonates. However, a key finding was that septic neonates had significantly higher mean serum bilirubin levels within 24 hours of admission (10.5 ± 2.7 mg/dL) than non-septic neonates (8.2 ± 2.1 mg/dL; $p = 0.036$), suggesting that systemic infection may accelerate or intensify bilirubin production and/or impair its clearance. This observation is consistent with prior studies that reported elevated bilirubin fractions in neonates with sepsis, particularly in those with bacterial infections causing hepatocellular injury or hemolysis[1,15]. Furthermore, the phototherapy requirement was notably higher in the sepsis group (77.27%) than in non-septic neonates (41.67%), although the difference did not reach statistical significance. This trend nonetheless suggests increased disease severity or delayed bilirubin metabolism in septic neonates, a hypothesis also explored in studies from Ethiopia and India, which observed similar patterns of phototherapy usage in septic neonates[16,17]. Correlation analysis demonstrated a moderate positive relationship between serum bilirubin and CRP levels ($r = 0.38$; $p = 0.002$), and a stronger correlation with blood culture positivity ($r = 0.44$; $p = 0.001$), supporting the inflammatory basis of bilirubin elevation. CRP, as an acute-phase reactant, is a reliable indicator of infection severity and was previously shown to correlate well with hepatic dysfunction in septic neonates[18,19]. Conversely, total WBC count did not show a statistically significant correlation with bilirubin, highlighting the limited utility of leukocyte count alone in predicting hyperbilirubinemia risk in septic settings. Multivariable logistic regression confirmed that sepsis remained an independent predictor of hyperbilirubinemia (AOR = 2.81; 95% CI: 1.02–7.65; $p = 0.045$), even after adjusting for prematurity, CRP, and birth weight. While elevated CRP, prematurity, and low birth weight (<1500g) showed positive associations with hyperbilirubinemia, none reached statistical significance in the final model, corroborating earlier findings from South Asian studies that emphasized the dominant role of infection over other perinatal risk factors[16,17]. Blood culture results further underscored the biological plausibility of this association. Among neonates with positive cultures, 84.8% developed jaundice, compared to only 17.5% among culture-negative neonates ($p < 0.001$), establishing a strong link between microbiologically confirmed sepsis and bilirubin elevation. Pathogen-specific analysis added depth to this observation: Pseudomonas infections were associated with the highest mean bilirubin level (11.1 mg/dL), followed closely by Acinetobacter (10.9 mg/dL) and Klebsiella (10.7 mg/dL). These Gram-negative organisms are known to induce hepatocellular injury and hemolysis, both of which elevate bilirubin production[11,12]. The presence of multidrug-resistant organisms like Acinetobacter, which were predominant in our cohort, may further complicate clinical management by prolonging infection duration and delaying resolution, thereby extending the window for bilirubin accumulation[9]. While hyperbilirubinemia itself is not typically classified as a complication of sepsis, it appears to be closely associated with several sepsis-related morbidities. Neonates with jaundice had significantly higher rates of respiratory distress syndrome (37% vs. 19%; $p = 0.04$), hypoglycemia (47% vs. 19%; $p = 0.005$), and feeding intolerance (42% vs. 13%; $p = 0.003$) compared to their non-jaundiced counterparts. These findings suggest a shared pathophysiological basis rooted in systemic inflammation and organ immaturity. However, no significant differences were observed in the occurrence of IVH, NEC, or

MODS, implying that bilirubin levels may not directly correlate with all major neonatal complications but may serve as a surrogate marker for systemic burden. Overall, the results of this study reinforce the emerging understanding that neonatal sepsis and hyperbilirubinemia are not isolated phenomena but are interconnected via shared mechanisms including inflammatory cytokine activity, hepatic dysfunction, and hemolysis. These findings validate and extend earlier reports by Das et al., Vaz et al., and Olusanya et al., and highlight the urgent need for integrated protocols that account for bilirubin dynamics during sepsis management in NICUs, particularly in low- and middle-income countries like Bangladesh[1,3,10].

Limitations of The Study

This study was limited by its retrospective design and single-center setting, which may restrict the generalizability of the findings to other NICU populations. The sample size also limited the statistical power for subgroup analyses. Additionally, important confounding variables such as ABO or Rh incompatibility, G6PD deficiency, and the timing of phototherapy initiation were not consistently documented and thus could not be adjusted for in the multivariate analysis.

Conclusion

This study demonstrated a significant association between neonatal sepsis and elevated serum bilirubin levels in hospitalized newborns. Septic neonates exhibited higher mean bilirubin concentrations and were more likely to require phototherapy, although not all comparisons reached statistical significance. Sepsis was independently predictive of hyperbilirubinemia, and the correlation strengthened in neonates with positive blood cultures. Among pathogens, Pseudomonas and Acinetobacter infections were associated with the highest bilirubin levels. Furthermore, jaundiced neonates had a higher incidence of complications such as respiratory distress, hypoglycemia, and feeding intolerance, underscoring the clinical relevance of this association. These findings highlight the need for integrated management protocols that address both sepsis and bilirubin monitoring in neonates, especially in resource-limited NICU settings.

Recommendation

Routine bilirubin monitoring should be implemented in all neonates diagnosed or suspected of sepsis, especially those with positive blood cultures or elevated CRP levels. Blood culture results and inflammatory markers like CRP should be prioritized when assessing jaundice risk in septic neonates. Pathogen-specific risk stratification may help predict the severity of hyperbilirubinemia and optimize phototherapy decisions. Larger prospective multicenter studies are recommended to validate these findings and to incorporate broader variables such as hemolytic markers, genetic predispositions, and treatment timelines.

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