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Original Research Article

Prevalence of Neonatal Hyperbilirubinemia in Low Birth Weight and Normal Birth Weight Babies

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Abstract:

Introduction: Neonatal hyperbilirubinmia affects most newborns, with greater prevalence and severity in low birth weight infants due to physiological immaturity and impaired bilirubin metabolism. Early identification of risk and prevalence patterns across LBW and NBW neonates is vital for timely interventions, preventing complications, and strengthening neonatal care protocols globally.

Methods: This prospective study (Nov 2016–Aug 2018) at Prathima Institute included neonates with hyperbilirubinemia. Exclusions were lack of consent, incomplete evaluation, or outside births. Risk factors and clinical data were recorded. Diagnosis, monitoring, and management followed AAP guidelines, with bilirubin measured by the Diazo method and detailed laboratory evaluations performed.

Results: Among 150 neonates (50 LBW, 100 NBW), hyperbilirubinemia was more frequent in LBW (60% vs. 42%), with earlier onset and higher peak bilirubin. LBW required longer phototherapy and more exchange transfusions. Prematurity was significantly associated, while other maternal and perinatal risk factors showed higher proportions but no statistical significance.

Conclusion: The study concludes that hyperbilirubinemia is more prevalent, severe, and of earlier onset in low birth weight neonates. Prematurity was the most significant risk factor. LBW infants required longer phototherapy and more exchange transfusions, highlighting the need for rigorous screening and timely intervention to improve outcomes.

Keywords: Hyperbilirubinemia, Low Birth Weight, Normal Birth Weight, Phototherapy, Prematurity.

Introduction

Neonatal hyperbilirubinemia is one of the most common clinical conditions encountered during the early neonatal period, affecting nearly 60% of term and 80% of preterm infants worldwide [1]. It results from the imbalance between bilirubin production and elimination, and though often physiological, it may progress to severe hyperbilirubinemia leading to kernicterus if left untreated. The prevalence and severity of hyperbilirubinemia vary significantly between low birth weight (LBW) and normal birth weight (NBW) neonates due to physiological immaturity, impaired hepatic conjugation, and increased vulnerability of LBW infants [2].

LBW infants, especially those <2500 g, have immature hepatic enzyme systems and reduced albumin binding capacity, predisposing them to higher bilirubin levels. In contrast, term NBW babies generally have better bilirubin metabolism, yet still experience significant jaundice requiring intervention in a proportion of cases [3]. Identifying prevalence patterns across birth weight categories is essential for planning timely screening and treatment, particularly in resource-limited settings where delayed diagnosis contributes to morbidity and mortality.

Assessing the comparative prevalence of hyperbilirubinemia among LBW and NBW neonates will provide critical insights into risk stratification, preventive strategies, and early therapeutic interventions. Such data are vital for strengthening neonatal care protocols and reducing the burden of bilirubin-induced neurologic dysfunction (BIND) in developing countries [4].

Methods

It was a prospective study conducted in the department of Pediatrics and department of Obstetrics and Gynecology, Prathima Institute of Medical Sciences, Karimnagar. Study was conducted from November 2016 to August 2018. Informed consent was obtained from the parents. Newborn with hyperbilirubinemia, irrespective of

the gestational age (GA) were included. The study excluded neonates in whom informed consent from parents or guardians was not obtained, those for whom essential investigations or treatment could not be carried out for any reason, and babies who were born or received treatment outside the study setting.

All live-born infants delivered in the institution during the study period were screened, and detailed perinatal information was collected. Risk factors considered for the development of hyperbilirubinemia included birth weight, GA, maternal age, birth order, mode of delivery, type of anesthesia, and oxytocin induction. These parameters were recorded from maternal case sheets and delivery records. Infants who developed clinical jaundice were carefully assessed and monitored. Babies requiring admission were managed in the NICU according to standard departmental protocols. The management approach adhered to the guidelines recommended by the American Academy of Pediatrics (AAP) [5].

All neonates who underwent phototherapy had serum bilirubin levels monitored every 24 hours until 24 hours after discontinuation of phototherapy, to detect any rebound hyperbilirubinemia. In cases requiring exchange transfusion, serum bilirubin was measured after 24 and 48 hours following the procedure and continued until normalization of levels. Laboratory evaluation was performed to identify the etiology and severity of hyperbilirubinemia. A complete hemogram including hemoglobin concentration, total leukocyte count, platelet count, and reticulocyte count was obtained. Blood grouping, direct Coombs test (DCT), glucose-6-phosphate dehydrogenase (G6PD) activity, sickling test, and osmotic fragility tests were performed as clinically indicated. These investigations helped identify underlying causes such as blood group incompatibility, hemolytic disorders, G6PD deficiency, or hereditary red cell abnormalities, all of which contribute to exaggerated bilirubin production and impaired clearance [6].

The measurement of serum bilirubin was carried out using the Diazo method of Pearlman and Lee, which remains a reliable and widely utilized biochemical technique for total serum bilirubin estimation [7]. All biochemical analyses were performed in the institutional central laboratory under standardized quality control procedures. The severity of hyperbilirubinemia was classified according to AAP treatment guidelines, and interventions were tailored accordingly. Clinical monitoring was supplemented with standardized documentation of jaundice progression and response to therapy. The combination of clinical examination, laboratory evaluation, and structured monitoring ensured accurate diagnosis, timely initiation of treatment, and prevention of complications such as bilirubin encephalopathy. Data from all neonates were systematically recorded, analyzed, and stratified by LBW and NBW status, enabling the identification of prevalence patterns and risk factor associations.

Results

Baseline characteristics of 150 neonates, including 50 LBW and 100 NBW. LBW infants had significantly lower mean birth weight and GA compared to NBW (P<0.001). Maternal age, gender distribution, and mode of delivery did not differ significantly between the groups, indicating comparable demographic backgrounds. Table 1 highlights the prevalence and severity of hyperbilirubinemia. It was significantly higher in LBW babies (60%) than NBW (42%, P=0.04). LBW neonates developed jaundice earlier, with higher mean peak bilirubin levels (16.2 mg/dL vs. 14.1 mg/dL, P=0.02). Severe cases were more frequent in LBW, though not statistically significant. Table 2 demonstrates interventions for hyperbilirubinemia. Phototherapy was more frequent in LBW infants (48%) compared to NBW (34%), though not significant. Exchange transfusion was required more in LBW (12% vs. 4%). Mean phototherapy duration was significantly longer in LBW (62 vs. 48 hours, P=0.01). Rebound hyperbilirubinemia was infrequent. Prematurity was significantly associated, affecting 73.3% of LBW versus 23.8% of NBW infants (P<0.001). Other factors, including oxytocin induction, birth asphyxia, meconium staining, maternal diabetes/PIH, and previous sibling jaundice, showed higher proportions in LBW but were not statistically significant between the groups.

Table 1: Prevalence and severity of Hyperbilirubinemia among the study babies

Parameter	LBW	NBW	Total	P value
Hyperbilirubinemia (%)	30 (60.0)	42 (42.0)	72 (48.0)	0.04
Mean Age at Onset (days)	2.4 ± 0.9	3.1 ± 1.0	2.8 ± 1.0	0.01
Mean Peak Serum Bilirubin (mg/dL)	16.2 ± 3.1	14.1 ± 2.8	15.0 ± 3.0	0.02
Severe Cases (>20 mg/dL) (%)	8 (16.0)	10 (10.0)	18 (12.0)	0.32

Table 2: Interventions for Hyperbilirubinemia to the study infants				
Intervention	**LBW**	**NBW**	**Total**	**P value**
Phototherapy (%)	24 (48.0)	34 (34.0)	58 (38.7)	0.12
ET (%)	6 (12.0)	4 (4.0)	10 (6.7)	0.08
MDP in hrs	62 ± 18	48 ± 14	54 ± 16	0.01
RH (%)	4 (8.0)	3 (3.0)	7 (4.7)	0.21
Exchange Transfusion (ET); Mean Duration of Phototherapy (MDP); Rebound Hyperbilirubinemia (RH)				

Discussion

In the present study, baseline characteristics of 150 neonates comprising 50 LBW and 100 NBW infants were compared. As expected, LBW neonates demonstrated significantly lower mean birth weight and gestational age compared to NBW infants (P<0.001), consistent with existing literature showing the strong association between prematurity and reduced birth weight [8]. LBW and preterm infants are physiologically more vulnerable, with immature hepatic function and reduced ability to metabolize bilirubin, placing them at greater risk of hyperbilirubinemia. This finding highlights the importance of stratifying neonatal outcomes by birth weight and gestational maturity in epidemiological and clinical studies [9].

On the other hand, maternal age, gender distribution, and mode of delivery did not differ significantly between the two groups, suggesting that demographic factors were relatively uniform across study populations. This comparability strengthens the validity of observed differences in neonatal outcomes being attributable to biological vulnerability associated with LBW and prematurity rather than maternal or perinatal confounders. Prior research has similarly shown that although cesarean delivery and maternal age may influence neonatal morbidity in some populations, birth weight and gestational age remain the strongest predictors of early neonatal complications including hyperbilirubinemia [10]. Thus, the current baseline analysis reinforces the need for close monitoring of LBW neonates, while also confirming that maternal age and mode of delivery may not independently predispose infants to hyperbilirubinemia in comparable cohorts.

Discussion

The present study demonstrates that the prevalence of hyperbilirubinemia was significantly higher among LBW neonates (60%) compared to NBW neonates (42%) (p=0.04). These findings highlight the increased vulnerability of LBW infants to bilirubin accumulation, largely attributable to physiological immaturity, reduced hepatic glucuronyl transferase activity, and limited bilirubin conjugation capacity [6]. Furthermore, the earlier onset of jaundice in LBW neonates (mean age 2.4 ± 0.9 days) compared to NBW (3.1 ± 1.0 days, p=0.01) is consistent with prior reports suggesting that prematurity and low weight predispose neonates to more rapid bilirubin accumulation [1]. The higher mean peak serum bilirubin levels observed in LBW infants (16.2 ± 3.1 mg/dL vs. 14.1 ± 2.8 mg/dL, P=0.02) further reinforces their greater risk of severe hyperbilirubinemia, emphasizing the need for stringent monitoring in this subgroup.

Although the proportion of severe hyperbilirubinemia cases (>20 mg/dL) was higher in LBW infants (16%) compared to NBW (10%), this difference did not reach statistical significance (P=0.32). Similar trends have been documented in other studies, where LBW neonates consistently demonstrated higher bilirubin levels but differences in severe cases varied depending on population characteristics and intervention protocols [3]. The clinical implications of these findings are profound, as LBW neonates, by virtue of earlier onset and higher bilirubin peaks, are more prone to complications such as bilirubin-induced neurologic dysfunction (BIND) and kernicterus if untreated [11]. Therefore, targeted screening, early phototherapy initiation, and standardized follow-up protocols are warranted in LBW populations to prevent adverse outcomes.

The analysis of interventions for hyperbilirubinemia in this study reveals important differences between LBW and NBW neonates. Phototherapy was more frequently required among LBW infants (48%) compared to NBW (34%), though this difference did not reach statistical significance. Nevertheless, this trend indicates a higher clinical burden of jaundice in LBW neonates, aligning with earlier studies that report increased phototherapy requirements due to immature hepatic conjugation pathways and heightened bilirubin production [6]. The mean duration of phototherapy was significantly longer in LBW babies (62 ± 18 hours) than in NBW babies (48 ± 14 hours, P=0.01), emphasizing their slower bilirubin clearance and higher risk of rebound hyperbilirubinemia. Similar findings have been documented by Kumar et al., who demonstrated that LBW neonates required prolonged therapy compared to their term counterparts [9].

ET, though less common overall, was more frequently required among LBW neonates (12% vs. 4%). This reflects the tendency of LBW infants to develop higher bilirubin levels at an earlier age, predisposing them to severe hyperbilirubinemia and complications such as acute bilirubin encephalopathy if not aggressively managed [3].

Rebound hyperbilirubinemia following cessation of phototherapy was infrequent in both groups, indicating the effectiveness of vigilant monitoring protocols in preventing relapse. The findings underscore the need for stricter surveillance of LBW infants, as they are more prone to both requiring phototherapy and, in some cases, escalating to ET. Adhering to standardized protocols, such as those recommended by the American AAP, is crucial in optimizing outcomes, minimizing morbidity, and reducing the risk of long-term neurologic impairment [5].

The analysis of risk factors demonstrated that prematurity was the most significant determinant of neonatal hyperbilirubinemia, affecting 73.3% of LBW infants compared with 23.8% of NBW neonates ($P<0.001$). This observation is consistent with previous research indicating that immature hepatic glucuronyl transferase activity, reduced albumin-binding capacity, and higher red blood cell turnover in preterm and LBW neonates predispose them to exaggerated bilirubin production and impaired clearance [6]. These pathophysiological vulnerabilities explain the earlier onset and higher severity of jaundice observed in LBW neonates. Furthermore, prematurity is widely recognized as a critical risk factor in international guidelines for hyperbilirubinemia screening, underscoring the importance of early monitoring and timely intervention in this subgroup [5].

Other perinatal and maternal risk factors including oxytocin induction, birth asphyxia, meconium staining, maternal diabetes or pregnancy-induced hypertension (PIH), and previous sibling jaundice were observed more frequently in LBW infants but did not reach statistical significance in this study. Similar findings have been reported in multi-center analyses, where maternal conditions such as diabetes and PIH have been associated with altered neonatal red blood cell dynamics and increased risk of jaundice, though their effect sizes were variable depending on the study population [1]. Additionally, oxytocin induction and perinatal stressors like birth asphyxia may exacerbate hemolysis or impair bilirubin metabolism, but consistent associations have been difficult to establish [3]. These results suggest that while prematurity remains the dominant predictor of neonatal hyperbilirubinemia, maternal and perinatal factors may contribute synergistically in susceptible infants. Hence, comprehensive neonatal risk assessment incorporating both biological maturity and perinatal exposures is essential for early identification and prevention of severe hyperbilirubinemia.

Conclusion

This study highlights the higher prevalence, earlier onset, and greater severity of hyperbilirubinemia among LBW neonates compared to NBW infants. LBW babies required more frequent and prolonged phototherapy and had a greater need for exchange transfusion, underscoring their increased vulnerability. Prematurity emerged as the most significant risk factor, while maternal and perinatal factors such as oxytocin induction, birth asphyxia, and diabetes/PIH showed contributory roles without statistical significance. These findings emphasize the importance of vigilant monitoring, timely intervention, and risk-based management strategies to prevent complications and improve neonatal outcomes.

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