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Research Article

An Observational Study to Determine Etiological Profile of Neonates Admitted With Indirect Hyperbilirubinemia at Sms Medical College and Hospital, Jaipur, Rajasthan

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ABSTRACT

Introduction: Neonatal hyperbilirubinemia is one of the most common causes of neonatal morbidity and hospital admissions, particularly in developing countries. While most cases are physiological and self-limiting, a significant proportion progress to pathological jaundice, which may result in acute bilirubin encephalopathy and kernicterus if untreated. This study was undertaken to evaluate the clinical profile, etiological factors, and outcomes of neonates with indirect hyperbilirubinemia.

Materials and Methodology: This prospective observational study was conducted in the Department of Paediatric Medicine and associated hospitals of SMS Medical College, Jaipur, from April 2023 to November 2024. A total of 135 neonates (<28 days old) with indirect serum bilirubin levels in the phototherapy range, as per AAP guidelines, were included. Clinical, demographic, and laboratory data were recorded. Investigations included serum bilirubin, blood grouping, Coombs test, sepsis screening, and hemolysis evaluation. Management was carried out according to AAP protocols, with phototherapy or exchange transfusion as indicated. Data were analyzed using SPSS software.

Results: Among 135 neonates, males (55.56%) predominated over females (44.44%). Most cases presented between 96–168 hours of life (49.62%). The leading cause of jaundice was ABO incompatibility (40.7%), followed by physiological jaundice (22.2%), Rh incompatibility (10.4%), sepsis (10.4%), perinatal asphyxia (7.4%), cephalohematoma (5.9%), and idiopathic cases (3%). Phototherapy was effective in 87.4% of cases, while 12.6% required exchange transfusion. Most neonates (94.1%) were discharged; mortality occurred in 3.7%, and 1.5% were discharged with sequelae.

Conclusion: ABO incompatibility was the most frequent cause of neonatal jaundice. Early recognition and timely management with phototherapy or exchange transfusion are crucial in preventing bilirubin-induced complications and improving neonatal outcomes.

Keywords: Neonatal Jaundice, Hyperbilirubinemia, ABO Incompatibility, Phototherapy, Exchange Transfusion.

INTRODUCTION:

Neonatal jaundice, defined as a yellow discoloration of the skin, sclera, and mucous membranes, is among the most frequent conditions requiring medical evaluation in newborns. It affects approximately 60% of term and nearly 80% of preterm infants within the first week of life [1]. Clinically, it becomes visible when serum bilirubin exceeds 7 mg/dl in neonates, compared to levels above 2 mg/dl in adults [2]. While most cases are physiological and self-limiting, nearly 5–10% progress to clinically significant hyperbilirubinemia requiring intervention, most commonly phototherapy [2,3].

The condition is primarily due to unconjugated hyperbilirubinemia, resulting from immature hepatic conjugation mechanisms and increased red blood cell turnover [4]. Although generally benign, in some neonate's bilirubin levels rise excessively, leading to pathological hyperbilirubinemia. If untreated, this can cause bilirubin-induced neurologic dysfunction (BIND) and kernicterus, a rare but severe complication associated with irreversible neurodevelopmental deficits such as sensorineural hearing loss, motor abnormalities, and occasionally intellectual

impairment [4,5].

Traditionally, jaundice has been assessed clinically by observing dermal bilirubin deposition, progressing cephalocaudally, as described by Kramer in 1969 [6]. However, subsequent studies, including that of Moyer et al. (2000), demonstrated considerable discrepancies between visual assessments and actual serum bilirubin levels, underscoring the unreliability of this method [7].

The etiologies of indirect hyperbilirubinemia are diverse, including hemolytic conditions such as ABO and Rh incompatibility, glucose-6-phosphate dehydrogenase (G6PD) deficiency, prematurity, sepsis, cephalohematoma, and breastfeeding-associated jaundice [8]. Their prevalence varies widely depending on genetic, geographic, and socioeconomic factors [8]. Notably, the re-emergence of kernicterus has been attributed to factors such as reduced awareness of warning signs, early neonatal discharge, misinterpretation of bilirubin toxicity risks, breastfeeding-related jaundice, and difficulties in visual assessment among infants with darker skin pigmentation [9].

In resource-limited settings like India, delayed recognition, inadequate healthcare access, and the lack of universal screening exacerbate the risks, often resulting in late presentations and adverse neurological outcomes [10]. Early identification of underlying etiologies is therefore crucial to enable timely management, reduce complications, and minimize the need for invasive interventions such as exchange transfusion [10].

The present study aims to evaluate the etiological spectrum of indirect hyperbilirubinemia among neonates admitted to a tertiary care hospital. By analyzing clinical and laboratory profiles, it seeks to generate evidence that may inform improved diagnostic and management strategies for neonatal jaundice.

MATERIALS AND METHODOLOGY:

Study Design and Setting

This hospital-based prospective observational study was conducted in the Department of Paediatric Medicine, SMS Medical College, Jaipur, and its affiliated hospitals, namely Zanana Hospital, Gangori Hospital, Kanwatia Hospital, and Mahila Chikitsalaya. The study was carried out over a period of one year, from April 2023 to November 2024.

Study Population and Sample Size

The study population comprised neonates less than 28 days of life presenting with indirect hyperbilirubinemia requiring phototherapy as per the American Academy of Pediatrics (AAP) nomogram. A total of 135 neonates delivered and admitted to SMS Medical College and associated hospitals were enrolled. The sample size was calculated at a 95% confidence interval with a 5% absolute allowable error, based on the assumption that pathological jaundice due to ABO incompatibility occurs in 9.7% of cases, as reported in the seed article. The formula applied was:

Sample Size (n) = Z* PQ/ L

(Z= 1.96, P-Prevalence, Q= 1-P, L- Allowable Error)

where $Z = 1.96$, P = prevalence, $Q = 1–P$, and L = allowable error. This calculation provided an adequate sample for evaluating other variables as well.

Inclusion Criteria

1. Neonates <28 days of life with indirect serum bilirubin levels within phototherapy range according to AAP guidelines.
2. Parents or guardians providing informed written consent.

Exclusion Criteria

1. Neonates with direct hyperbilirubinemia.
2. Refusal of parental consent.

Screening and Enrollment

All neonates delivered and admitted at the participating hospitals were screened daily for signs of jaundice. In those with clinical suspicion, total serum bilirubin (TSB) was measured. Approximately 2–3 ml of venous blood was collected under aseptic precautions and analyzed using the diazo method. Neonates meeting AAP criteria for phototherapy were enrolled following eligibility confirmation and consent.

Data Collection

Detailed demographic and clinical information was documented in a pre-designed proforma. Variables recorded included age, birth weight, gestational age, breastfeeding status, family history of jaundice or hemolytic disorders, presence of cephalohematoma, history of perinatal asphyxia, and signs of acute bilirubin encephalopathy.

Clinical Evaluation and Investigations

All neonates underwent clinical examination and laboratory investigations:

- Blood group and Rh typing of neonate and mother.

- Direct Coombs Test in suspected ABO/Rh incompatibility.
- CBC, haemoglobin, peripheral smear, and reticulocyte count for haemolysis.
- Bilirubin monitoring for disease progression.

In cases where hemolysis was suspected without evidence of blood group incompatibility, additional tests included G6PD assay, sickling test, hemoglobin electrophoresis, and osmotic fragility test. Neonatal sepsis was diagnosed by positive blood culture or a sepsis screen, defined by ≥2 criteria (TLC <5000/mm³, reduced ANC, I/T ratio ≥20%, CRP ≥10 mg/L, or micro-ESR ≥15 mm in 1 hour).

Management Protocol

Treatment was guided by AAP recommendations. Phototherapy was the first-line intervention, while exchange transfusion was performed in cases of severe hyperbilirubinemia or acute bilirubin encephalopathy. Serum bilirubin was reassessed after 48 hours of therapy to monitor resolution.

Statistical Analysis

Data were compiled in Microsoft Excel and analyzed using SPSS software. Descriptive statistics, including means with standard deviations, medians, and ranges, were used for continuous variables, while categorical variables were expressed as frequencies and percentages.

RESULT:

Table 1 presents the distribution of neonatal jaundice cases according to demographic and clinical parameters. The majority of neonates were admitted between 96–168 hours of life (49.62%), with most being male (55.56%). In terms of birth weight, normal birth weight neonates constituted 46.67%, followed by low birth weight (44.44%), while very low and extremely low birth weight infants accounted for smaller proportions (7.41% and 1.48%, respectively). A greater proportion of neonates were term (60%) compared to preterm (40%). The majority were exclusively breastfed (77.04%). Regarding management, phototherapy was the primary treatment (87.14%), while 12.59% required combined phototherapy and exchange transfusion, predominantly due to Rh incompatibility (70.58%), with the remainder attributed to ABO incompatibility (29.41%).

Table 1. Distribution of Cases According To Age and Gender

Parameters		Number (%)
Age	within 24 hrs	2 (1.48%)
	24 to 96 hrs	61 (45.18%)
	96 to 168 hrs	67 (49.62%)
	>168 hrs	5 (3.70%)
Gender	Male	75 (55.56%)
	Female	60 (44.44%)
Birth weight	LBW (Low Birth Weight)	60 (44.44%)
	Normal Birth Weight	63 (46.67%)
	VLBW (Very Low Birth Weight)	10 (7.41%)
	ELBW (Extremely Low Birth Weight)	2 (1.48%)
Term	Pre-term	54 (40%)
	Term	81 (60%)
Brest Feeding Status	Yes	104 (77.04%)
	No	31 (22.96%)
Treatment	Phototherapy	118 (87.14%)
	Phototherapy + blood transfusion	17 (12.59%)
Etiology of Blood exchange	Rh incompatibility	70.58%
	ABO Incompatibility	29.41%

Table 2. Comparison of Age According To Cause of Jaundice

Cause of Jaundice	Number of Cases (N)	Mean Age (Hrs)
ABO Incompatibility	55 (40.74%)	86.49 ± 30.27
Perinatal asphyxia	10 (7.41%)	94.90 ± 13.77
Cephalohematoma	8 (5.93%)	94.00 ± 11.25

Idiopathic	4 (2.96%)	111.25 ± 8.85
Physiological	30 (22.22%)	108.83 ± 20.09
Rh Incompatibility	14 (10.37%)	46.29 ± 21.20
Sepsis	14 (10.37%)	123.79 ± 41.26
P value	<0.001	

Table 2 compares the mean age at admission according to the underlying cause of neonatal jaundice. The most common etiology was ABO incompatibility (40.74%), with a mean age of 86.49 ± 30.27 hours. Cases due to Rh incompatibility presented much earlier, with the lowest mean age (46.29 ± 21.20 hours), reflecting the rapid onset and severity of hemolytic disease. In contrast, sepsis-associated jaundice had the highest mean age (123.79 ± 41.26 hours), indicating a delayed presentation. Physiological jaundice (22.22%) also showed later onset (108.83 ± 20.09 hours) compared to hemolytic causes. Other etiologies, such as perinatal asphyxia (94.90 ± 13.77 hours), cephalohematoma (94.00 ± 11.25 hours), and idiopathic cases (111.25 ± 8.85 hours), demonstrated intermediate presentations. The difference in mean age across etiologies was statistically significant ($p < 0.001$), underscoring the variability in onset patterns depending on the underlying cause.

Table 3. Distribution of Jaundice Causes by Gestation

Causes of Jaundice	**Pre-term (N=54)**	**Term (N=81)**	**p value**
Physiological	33.33%	14.81%	0.02
Perinatal asphyxia	5.55%	8.64%	0.05
ABO Incompatibility	50%	33.33%	0.04
Cephalohematoma	3.70%	7.40%	0.2
Idiopathic	1.85%	3.70%	0.5
Rh incompatibility	16.66%	6.17%	0.05
Sepsis	11.11%	9.87%	0.4

Table 3 illustrates the distribution of jaundice etiologies according to gestational age. Physiological jaundice was more frequent among preterm neonates (33.33%) compared to term neonates (14.81%), showing a statistically significant difference ($p = 0.02$). Similarly, ABO incompatibility was more common in preterm infants (50%) than term infants (33.33%, $p = 0.04$). Rh incompatibility also demonstrated a higher prevalence in preterms (16.66%) compared to terms (6.17%, $p = 0.05$). In contrast, conditions such as perinatal asphyxia, cephalohematoma, idiopathic jaundice, and sepsis showed no statistically significant variation between preterm and term groups ($p > 0.05$). These findings highlight that preterm neonates are more vulnerable to hemolytic and physiological causes of jaundice, while non-hemolytic etiologies appear distributed more evenly across gestational groups.

Table 4. Comparison of Total Serum Bilirubin after Treatment

	Mean	**P value**
Pre-treatment TSB (N=135)	17.73 ± 4.68	<0.001
Post-Treatment TSB(N=135)	3.12 ± 1.02	

Table 4 compares the mean total serum bilirubin (TSB) levels before and after treatment. The mean pre-treatment TSB was 17.73 ± 4.68 mg/dL, which decreased significantly to 3.12 ± 1.02 mg/dL following therapy ($p < 0.001$). This marked reduction demonstrates the high efficacy of the treatment modalities employed—predominantly phototherapy with or without exchange transfusion—in lowering bilirubin levels and preventing complications associated with severe hyperbilirubinemia.

Table 5. Association of TSB According To Jaundice Cause

Jaundice Cause	**N**	**Pretreatment TSB (Mean ± SD)**	**Post-treatment TSB (Mean ± SD)**

ABO Incompatibility	55	16.27±4.30	3.21±0.91
Birth Asphyxia	10	17.5±2.46	3.1±0.87
Cephalohematoma	8	19.25±0.88	3±0.92
Idiopathic	4	17.25±1.7	3.25±0.95
Physiological	30	17.16±3.45	2.73±0.73
RH Incompatibility	14	24.5±5.86	4±1.61
Sepsis	14	17.35±4.70	2.78±0.97
Total	135	17.73±4.68	3.12±1.02
p-value		<0.0001	0.007

Table 5 presents the association of total serum bilirubin (TSB) levels with different causes of jaundice. The highest mean pre-treatment TSB was observed in neonates with Rh incompatibility (24.5 ± 5.86 mg/dL), followed by cephalohematoma (19.25 ± 0.88 mg/dL), while the lowest levels were seen in cases of ABO incompatibility (16.27 ± 4.30 mg/dL). Post-treatment TSB levels declined significantly across all etiologies, with the greatest reduction noted in physiological jaundice (17.16 ± 3.45 to 2.73 ± 0.73 mg/dL) and sepsis-related jaundice (17.35 ± 4.70 to 2.78 ± 0.97 mg/dL). Despite variability in baseline bilirubin levels, treatment was uniformly effective in achieving clinically safe bilirubin concentrations, though Rh incompatibility cases continued to demonstrate relatively higher post-treatment values (4 ± 1.61 mg/dL). The differences across groups were statistically significant both before ($p < 0.0001$) and after treatment ($p = 0.007$), emphasizing that etiology plays a critical role in determining both severity at presentation and response to therapy.

Table 6. Comparison of Reduction in TSB According To Causes of Jaundice.

Jaundice cause	N	Mean	Std. Deviation	P value LS
ABO Incompatibility	55	-13.05	4.01	<0.001S
Birth Asphyxia	10	-14.4	1.95	
Cephalohematoma	8	-16.25	1.66	
Idiopathic	4	-14	1.82	
Physiological	30	-14.43	3.23	
RH Incompatibility	14	-20.5	5.76	
Sepsis	14	-14.57	5.37	
Total	135	-14.6	4.47	

Table 6 compares the reduction in total serum bilirubin (TSB) levels across different causes of neonatal jaundice. The overall mean reduction in TSB among the cohort was −14.6 ± 4.47 mg/dL, reflecting a significant therapeutic response. The greatest decline was observed in cases of Rh incompatibility (−20.5 ± 5.76 mg/dL), indicating the high baseline severity and the effectiveness of intensive interventions such as exchange transfusion. Substantial reductions were also noted in cephalohematoma (−16.25 ± 1.66 mg/dL) and physiological jaundice (−14.43 ± 3.23 mg/dL). In contrast, ABO incompatibility cases demonstrated a relatively smaller but still significant reduction (−13.05 ± 4.01 mg/dL). The differences across etiological groups were statistically significant ($p < 0.001$), suggesting that the extent of bilirubin reduction is closely related to the underlying cause of jaundice, with hemolytic etiologies such as Rh incompatibility requiring more aggressive management but also showing the most pronounced treatment response.

Table 7. Clinical Outcome of Patients

Outcome	Number	Percentage%
Death	5	3.70
Discharge with signs of encephalopathy	2	1.48
Discharged	127	94.07
Total	135	100.00

Table 7 presents the clinical outcomes of neonates with indirect hyperbilirubinemia. The majority of cases (127; 94.07%) were successfully managed and discharged without

complications, reflecting the overall effectiveness of timely interventions such as phototherapy and exchange transfusion. However, a small proportion of neonates were discharged with residual signs of encephalopathy (2; 1.48%), underscoring the risk of bilirubin-induced neurologic dysfunction despite treatment. Mortality was documented in 5 cases (3.70%), highlighting that severe hyperbilirubinemia continues to pose a risk of fatal outcomes, particularly in resource-limited settings where late presentation and comorbidities such as sepsis and perinatal asphyxia are common. These findings emphasize the importance of early detection, prompt initiation of therapy, and close monitoring to improve survival and reduce neurological sequelae.

DISCUSSION:

Neonatal hyperbilirubinemia remains one of the leading causes of hospital admissions during the neonatal period and constitutes an important public health concern, particularly in resource-limited healthcare systems. Timely recognition and appropriate management are essential to prevent serious complications, including bilirubin-induced neurologic dysfunction and kernicterus. The present study was undertaken to evaluate the clinical profile, risk factors, etiological distribution, and treatment outcomes among neonates with indirect hyperbilirubinemia admitted to a tertiary care hospital.

In the current study, out of 135 neonates, a predominance of males (55.56%) was observed, consistent with reports by Olusanya et al. and Trivedi et al., who demonstrated a higher incidence of neonatal jaundice in male infants [11,12]. This has been attributed to higher red blood cell mass and possible hormonal influences on hepatic enzyme maturation in male neonates.

With respect to gestational age, term infants accounted for the majority (60%) of cases, similar to observations by Valiyat S. et al. [13]. In contrast, Bhutani et al. [14] and Singhal et al. [15] reported a higher proportion of preterm neonates in their cohorts. Birth weight distribution revealed that nearly half of the neonates (46.67%) weighed more than 2500 g, while 44.44% were low birth weight, and smaller proportions belonged to very low and extremely low birth weight categories. These findings are in line with those of Nepal et al., [16] who also reported a predominance of neonates with adequate birth weight.

The majority of neonates presented between 96 and 168 hours of life, which corresponds to the physiological peak of serum bilirubin levels in term infants. This emphasizes the need for vigilant postnatal surveillance, particularly during the first week of life, to ensure early identification and timely treatment.

The etiological spectrum of neonatal hyperbilirubinemia in our study was multifactorial, with pathological jaundice accounting for 77.78% of cases. Among these, ABO incompatibility was the leading cause (52.38%), followed by sepsis (13.33%), Rh incompatibility (13.33%), perinatal asphyxia (9.52%), cephalhematoma (7.62%), and idiopathic causes (3.81%). Physiological jaundice, including cases associated with breastfeeding, accounted for 22.22%. These findings are consistent with Indian studies by Narang et al. [17] and Meharban et al.,[18] as well as international studies from Nigeria, Iran, and Turkey, all of which reported hemolytic jaundice, prematurity, and sepsis as major contributors [1,19]. Our findings further support the observation that ABO incompatibility has surpassed Rh incompatibility as the more common etiology, likely due to widespread use of anti-D immunoglobulin prophylaxis.

Similar to our results, Mishra et al. reported physiological jaundice in nearly one-third of neonates. Factors such as immaturity of liver enzymes, ethnicity, prematurity, male gender, cephalhematoma, breastfeeding, and delayed stooling have been recognized as contributors to its occurrence [20]. Cephalhematoma, though less common, was identified in 7.62% of our cases, which is comparable to the incidence reported by Valiyat S. et al. (5.3%).

Treatment strategies were primarily based on AAP guidelines. Phototherapy was the mainstay of management, used in 87.41% of cases, while 12.59% required exchange transfusion in addition to phototherapy, particularly in severe cases due to Rh and ABO incompatibility. This is comparable with the findings of Rasul et al., who reported 62% treated with phototherapy and 5.2% with exchange transfusion [21], and Khaton et al., who found 61% managed with phototherapy alone [22]. The relatively higher rate of exchange transfusion in our study reflects the higher severity of hyperbilirubinemia in some cases.

The effectiveness of treatment was demonstrated by a significant reduction in mean total serum bilirubin (14.61 ± 3.66 mg/dL; $p < 0.001$). Among etiologies, Rh

incompatibility showed the greatest decline, correlating with its higher baseline bilirubin levels. These findings are comparable to those of Adhikari et al., who also reported significant bilirubin reduction following phototherapy [23]. The greater decline observed in our cohort following exchange transfusion supports the continued role of this intervention in select severe cases, as also reported by Piyush Kanodia et al.

The outcomes in our study were encouraging, with the majority of neonates (94.07%) being discharged after successful management. However, mortality occurred in 3.70% of cases, while 1.48% were discharged with signs of bilirubin encephalopathy. These results underscore the importance of timely recognition and management to prevent adverse neurological outcomes.

Overall, our findings reinforce the importance of systematic evaluation of neonatal jaundice, with particular attention to maternal and neonatal blood group compatibility, risk of sepsis, birth asphyxia, and breastfeeding practices. Early initiation of phototherapy and timely use of exchange transfusion, where indicated, were effective in reducing serum bilirubin levels and preventing long-term sequelae.

CONCLUSION:

Neonatal hyperbilirubinemia remains a common cause of neonatal morbidity. In this study, ABO incompatibility was the leading etiology, followed by physiological jaundice, Rh isoimmunization, sepsis, and perinatal asphyxia. Most cases occurred within 4–7 days of life, with male and term neonates more frequently affected. Phototherapy was effective in the majority, while exchange transfusion was required in severe cases, particularly with hemolytic causes. Although most neonates (94%) recovered and were discharged, a small proportion developed encephalopathy or died, emphasizing the risks of delayed recognition. Early diagnosis, timely treatment, and improved neonatal care are essential to reduce complications and mortality associated with neonatal jaundice.

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