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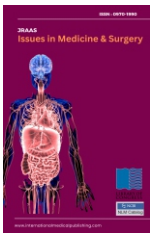

Research Article

Section: Paediatrics

Difference In Clinico - Etiological Profile of Neonatal Hyperbilirubinemia at A Tertiary Care Hospital

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HIGHLIGHTS

- Outborn neonates showed higher morbidity
- Pathological jaundice is common in outborns
- Sepsis is predominant cause among outborns
- ABO incompatibility frequent inborn etiology
- Early screening reduces preventable complications

Key Words:
Neonatal hyperbilirubinemia
Pathological jaundice
Inborn neonates
Outborn neonates
Neonatal sepsis
Prematurity
Small for gestational age
ABO incompatibility
Birth asphyxia
Tertiary care hospital

ABSTRACT

Introduction: Neonatal hyperbilirubinemia is one of the most common conditions requiring medical attention in the neonatal period. While most cases are physiological, delayed diagnosis and inappropriate management can lead to severe complications, including acute bilirubin encephalopathy and kernicterus. **Aim & Objectives:** To compare the clinico-etiological profile of neonatal hyperbilirubinemia between inborn and outborn neonates admitted to a tertiary care hospital and to identify key risk factors associated with pathological jaundice. **Material & Methods:** This hospital-based cross-sectional study was conducted in the Special Newborn Care Unit (SNCU) of a tertiary care hospital in Greater Noida over 12 months. Enrolled patients included 360 neonates ≤28 days with hyperbilirubinemia, either clinically suspected or biochemically confirmed. Data collection was done using a structured proforma, and etiological diagnosis was determined based on clinical and laboratory findings. Statistical analysis was done using SPSS v28.0; Chi-square and independent t-test were employed, with significance confirmed at $p<0.05$. **Results:** Of the 360 neonates included, 269 (74.7%) were inborn and 91 (25.3%) outborn. Pathological jaundice occurred more frequently among outborn neonates (60.4%) than inborn (28.6%) ($p=0.001$). In outborn infants, significantly higher prevalence for prematurity (36.1%), SGA status (56.1% vs 35.7%, $p=0.002$), and birth asphyxia (14.3% vs 4.8%, $p=0.02$) was observed. Neonatal sepsis was the most common etiology in outborn neonates (56.3%, $p<0.001$). On the other hand, ABO incompatibility (29.8%) and cephalohematoma (31.1%) were the principal factors among inborn infants. Total serum bilirubin levels were comparable between groups, though bilirubin testing was performed significantly earlier among inborn neonates ($p=0.001$). **Conclusion:** Significant differences exist in the etiological and clinical profile of hyperbilirubinemia between inborn and outborn neonates. Optimized early screening, infection control, and appropriate referral systems are mandatory in the reduction of preventable complications among outborn neonates.

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INTRODUCTION

Neonatal hyperbilirubinemia, which is also recognized as newborn jaundice, involves yellow discoloration of the skin and sclera resulting from excessive bilirubin accumulation in the bloodstream. Bilirubin is a normal metabolic by-product of hemoglobin catabolism occurring during erythrocyte turnover. Elevated bilirubin levels are common in newborns in the first few days of life due to immature hepatic pathways involved in bilirubin clearance [1]. Though newborn jaundice resolves without complications in majority cases; significantly higher bilirubin levels may cross the blood–brain barrier and result in bilirubin-induced neurological dysfunction or kernicterus, leading to long-term neurodevelopmental damage including complications like hearing impairment and cerebral palsy [2].

Jaundice affects an estimated 8–11% of newborns worldwide and is one of the commonest clinical presentations in the neonatal period [3,4]. Reports suggest that physiological jaundice occurs in ~60% of full-term infants and shows a higher incidence in preterm infants (~80%) [1]. According to the National Neonatal-Perinatal Database (NNPD), hyperbilirubinemia accounts for 3.3% of morbidity among inborn neonates in India, whereas the proportion rises to 22.1% among referred outborn infants [5]. The significant variation in incidence highlights differences in factors such as healthcare access, genetic susceptibility, breastfeeding patterns, infection control practices, and socioeconomic conditions [6].

The probability of severe hyperbilirubinemia increases due to a range of maternal and neonatal factors such as prematurity, low birth weight, blood group incompatibility, cephalohematoma, neonatal sepsis, inadequate feeding, and inherited enzyme deficiencies [7,8]. Late diagnosis may lead to hospital readmission that could be avoided, posing a challenge in many places [9,10]. Thus, early bilirubin screening and careful monitoring throughout the post-discharge period are indispensable components in neonatal care.

Phototherapy is the principal and optimal therapy that significantly reduces the requirement for exchange transfusion and averts neurological injury [11,12]. Clinical practice guidelines from the American Academy of Pediatrics endorse bilirubin monitoring with hour-specific nomograms to help in treatment initiation and decrease the risk of serious complications [13]. Nonetheless, in resource-limited settings, timely care may be hindered by inadequate equipment, limited training, and late presentation to healthcare facilities [14–16].

Genetic variability is crucial in the determination of susceptibility to hyperbilirubinemia. Polymorphisms in genes encoding bilirubin-conjugating enzymes, UGT1A1 and GST1, have been linked with a higher risk of clinically significant neonatal jaundice [17–19]. Insights into these factors can aid in improving screening and customized treatment strategies.

Knowing the potential for lifelong neurological damage, reducing the burden of severe neonatal hyperbilirubinemia becomes crucial by means of improved parental education, optimizing early screening protocols, and ensuring prompt treatment. The present study aims to Assess the clinico-etiological profile of neonatal hyperbilirubinemia in a tertiary care hospital setting. It focuses on evaluating the spectrum of clinical presentations, detection of underlying etiological factors, and comparison of differences in the etiological profiles of inborn and outborn neonates.

MATERIALS & METHODS

This hospital-based cross-sectional study was performed in the Special Newborn Care Unit (SNCU) of Hospital in Greater Noida, Western Uttar Pradesh over a period of 12 months. A total of 360 neonates admitted with clinically suspected or biochemically confirmed hyperbilirubinemia were enrolled, including both inborn infants delivered at the study centre and outborn infants referred from outside facilities. Neonates aged ≤28 days with jaundice whose parents provided written informed consent were included, while those with major congenital malformations or whose parents refused consent were excluded. The sample size calculation was performed with the prevalence-based formula $n = Z^2 p(1-p)/d^2$ assuming a prevalence of 60% among term and 80% among preterm infants (1), with 95% confidence level and 5% precision. Ethical clearance permissions were obtained from the Institutional Ethics Committee (GIMS IEC-ECR/1224/Inst./UP/2019), and confidentiality of patient information was confirmed.

For each enrolled neonate, detailed history including antenatal, natal, postnatal and family components was recorded using a structured proforma. This included maternal illness, drug usage during pregnancy (e.g., oxytocin), mode of delivery, perinatal complications such as birth asphyxia and delayed cord clamping, feeding practices and history of jaundice in siblings. A comprehensive clinical examination involved recording anthropometric measurements (weight, length, and head circumference) and systematic head-to-toe assessment. Baseline laboratory investigations comprised complete blood count, total, direct and indirect serum bilirubin, blood group and Rh typing of mother and infant, reticulocyte count and peripheral smear. For other clinical indications, additional investigations including thyroid function testing, TORCH screening, hemoglobinopathy evaluation, and evaluation of conjugated hyperbilirubinemia, were performed.

The clinical and laboratory evaluation was followed by etiological diagnosis assignment and categorization of neonates as physiological or pathological jaundice, accompanied by their stratification into inborn and outborn groups for comparative analysis. Management followed standard evidence-based protocols, including American Academy of Pediatrics (AAP) guidelines for phototherapy and exchange transfusion, and the treatment modality used for each participant was documented. Data were entered in Microsoft Excel and analyzed using SPSS software version 28.0 (IBM Corp., USA). Categorical data were expressed as frequencies & percentages and compared using Chi

square test, whereas continuous variables were presented as mean ± standard deviation and analyzed using independent *t*-test. A *p-value <0.05* was considered statistically significant.

RESULT

A total of 360 neonates admitted with hyperbilirubinemia were included in the study, comprising 269 inborn (74.7%) and 91 outborn (25.3%) newborns. Of the total participants, 132 (36.7%) had pathological jaundice, while 228 (63.3%) had physiological jaundice. Four neonates (1.1%) required exchangeA total of 360 neonates admitted with hyperbilirubinemia were included in the study, comprising 269 inborn (74.7%) and 91 outborn (25.3%) newborns. Of the total participants, 132 (36.7%) had pathological jaundice, while 228 (63.3%) had physiological jaundice. Four neonates (1.1%) required exchange transfusion. The participant recruitment and classification process is depicted in Figure 1.

Baseline Characteristics

As described in Table 1, among the 360 neonates enrolled, 188 (52.2%) were male and 172 (47.8%) were female. A total of 130 infants (36.1%) were preterm, with 85 inborn and 45 outborn, while 227 (63.9%) were term. Small for gestational age (SGA) status was significantly more frequent among outborn infants compared to inborn infants (56.0% vs. 35.7%, p=0.002). Birth asphyxia, defined by APGAR <5 at 5 minutes, occurred more commonly in outborn than inborn neonates (14.3% vs. 4.8%, p=0.02). Most participants (96.7%) reported no significant family history of jaundice.

Clinical and Anthropometric Profile

The mean birth weight of inborn infants was significantly higher than outborn infants (2.78 ± 0.46 kg vs 2.55 ± 0.61 kg, p=0.002). Length (48.17 ± 2.04 cm vs 47.43 ± 2.95 cm, p<0.05) & head circumference (34.00 ± 1.67 cm vs 33.56 ± 2.54 cm, p< 0.001) were also significantly higher among inborn newborn (Table 2). On physical examination, cephalohematoma was more frequent in inborn infants (8.9%) compared to outborn (6.6%), whereas hepatosplenomegaly and splenomegaly were relatively more common among outborn babies (Figure 2). No abnormal findings were detected most commonly in 85.1% of inborn vs 79.1% of outborn neonates (Table 3).

Etiology and Type of Jaundice

As described in Table 4, the etiological distribution of pathological jaundice among study participants demonstrated notable differences between inborn and outborn neonates. In this study, 16.9% of inborn infants exhibited Rh incompatibility, was observed as compared to 9.1% of outborn infants. A statistically significant association (p=0.000) was established in this group comparison. ABO incompatibility was more common among inborn babies (29.8%) than outborn (14.5%). Jaundice resulting from breastfeeding failure constituted to 7.8% of cases in the inborn group and 3.6% among outborn neonates. Neonatal sepsis appeared to be most common etiology in outborn neonates (56.3%), which was evidently higher than in inborn babies (14.3%), indicating strong statistical significance (p<0.001). Another major cause was cephalohematoma, which accounted for 31.2% of cases in inborn neonates relative to 10.9% in outborn neonates. TORCH infections were recorded only in outborn infants (3.6%), while biliary atresia was reported in 1.8% of outborn neonates, with zero cases recognized in inborn neonates.

Serum Bilirubin Profile

The mean day of bilirubin testing was significantly earlier among inborn neonates (3.09 ± 1.18 days) related to outborn neonates (3.29 ± 2.15 days, p=0.001). Other parameters such as mean total serum bilirubin level (12.88 vs 12.65 mg/dL, p=0.576), direct bilirubin (0.59 vs 0.69 mg/dL, p=0.064), or indirect bilirubin (12.29 vs 11.96 mg/dL, p=0.497) showed no significant differences between groups.

Table 1. Baseline Demographic Characteristics of Study Participants

Variable	Inborn (n=269)	Outborn (n=91)	Total (n=360)	p-value
Sex: Male	138 (51.3%)	50 (56.0%)	188 (52.2%)	-
Sex: Female	131 (48.7%)	41 (45.1%)	172 (47.8%)	-
Term	181 (67.2%)	46 (50.5%)	227 (63.9%)	-
Preterm	88 (32.7%)	45 (49.4%)	133 (36.1%)	-
SGA	96 (35.7%)	51 (56.1%)	147 (40.8%)	0.002 *
AGA	172 (64.0%)	39 (42.9%)	211 (58.6%)	-
LGA	1 (0.3%)	1 (1.1%)	2 (0.6%)	-
APGAR < 5 at 5 min	13 (4.8%)	13 (14.3%)	26 (7.2%)	0.02 *
Family history of jaundice	9 (3.4%)	3 (3.3%)	12 (3.3%)	-

Abbreviation: SGA: Small for Gestational Age; AGA: Appropriate for Gestational Age; LGA: Large for Gestational Age; APGAR: Appearance, Pulse, Grimace, Activity, Respiration

* *p < 0.05 considered statistically significant;* (-) indicates *no statistical comparison applicable*

Table 2: Anthropometric and Clinical Characteristics

Parameter	Inborn (mean ± SD)	Outborn (mean ± SD)	p-value
Birth weight (kg)	2.78 ± 0.46	2.55 ± 0.61	0.002 *
Length (cm)	48.17 ± 2.04	47.43 ± 2.95	<0.05*
Head circumference (cm)	34.00 ± 1.67	33.56 ± 2.54	<0.001 *

** p < 0.05 considered statistically significant*

Table 3: Head-to-toe Examination Findings

Clinical Finding	Inborn (n=269)	Outborn (n=91)
Cephalohematoma	24 (8.9%)	6 (6.6%)
Hepatomegaly	1 (0.4%)	1 (1.1%)
Hepatosplenomegaly	2 (0.7%)	2 (2.2%)
Macrosomia	1 (0.4%)	1 (1.1%)
Pallor	20 (7.4%)	6 (6.6%)
Splenomegaly	4 (1.5%)	3 (3.3%)
No abnormality detected	217 (85.1%)	72 (79.1%)

Table 4. Etiopathogenesis of Pathological Jaundice (n = 132)

Etiology	Inborn (n=77)	Outborn (n=55)	p-value
Rh incompatibility	13 (16.9%)	5 (9.1%)	0.000*
ABO incompatibility	23 (29.8%)	8 (14.5%)	—
Breastfeeding failure jaundice	6 (7.8%)	2 (3.6%)	—
Neonatal sepsis	11 (14.3%)	31 (56.3%)	<0.001*
Cephalohematoma	24 (31.2%)	6 (10.9%)	—
TORCH infection	0 (0%)	2 (3.6%)	—
Biliary atresia	0 (0%)	1 (1.8%)	—

TORCH: Toxoplasmosis, Rubella, Cytomegalovirus (CMV), Herpes Simplex Virus (HSV)
*p < 0.05 indicates statistical significance; (-) indicates *not statistically significant*

Table 5. Serum Bilirubin Profile

Parameter	Inborn (mean ± SD)	Outborn (mean ± SD)	p-value
Day of bilirubin measurement (days)	3.09 ± 1.18	3.29 ± 2.15	0.001*
Total Serum Bilirubin (mg/dL)	12.88 ± 2.61	12.65 ± 3.01	0.576
Direct bilirubin (mg/dL)	0.59 ± 0.24	0.69 ± 1.22	0.064
Indirect bilirubin (mg/dL)	12.29 ± 2.55	11.96 ± 2.93	0.497

*p < 0.05 indicates statistical significance

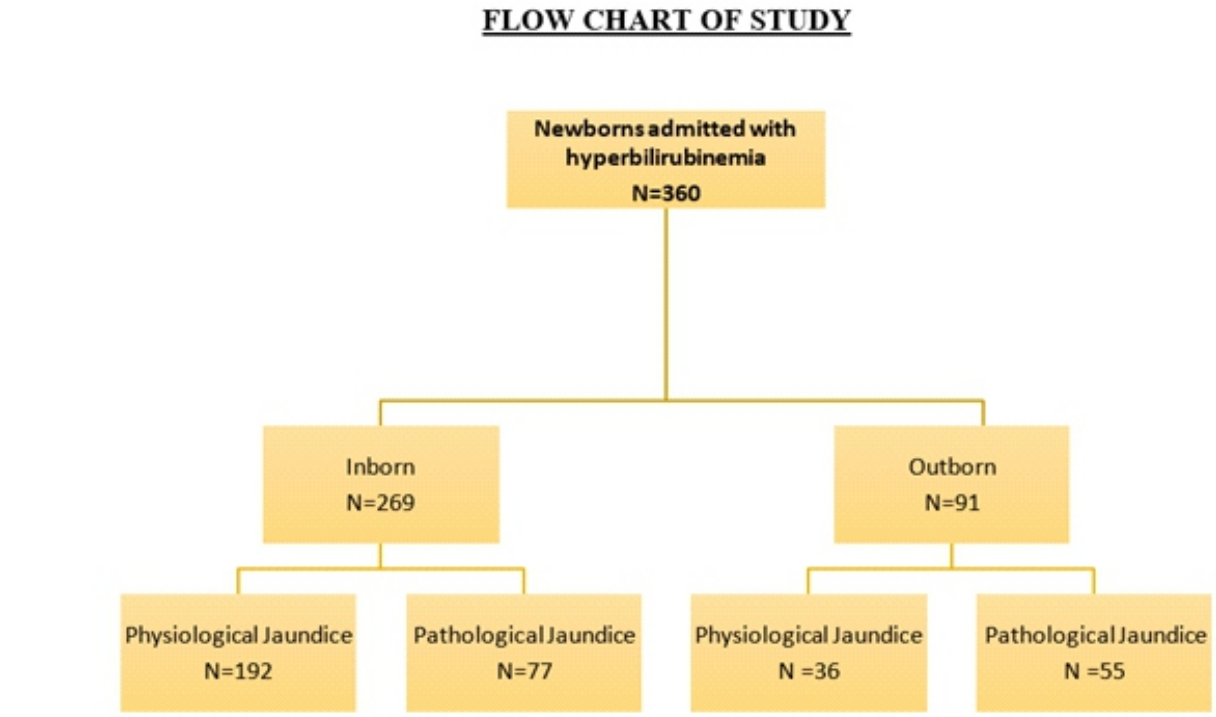

Figure 1: Study flow diagram showing distribution of inborn and outborn neonates with hyperbilirubinemia and their classification into physiological and pathological jaundice.

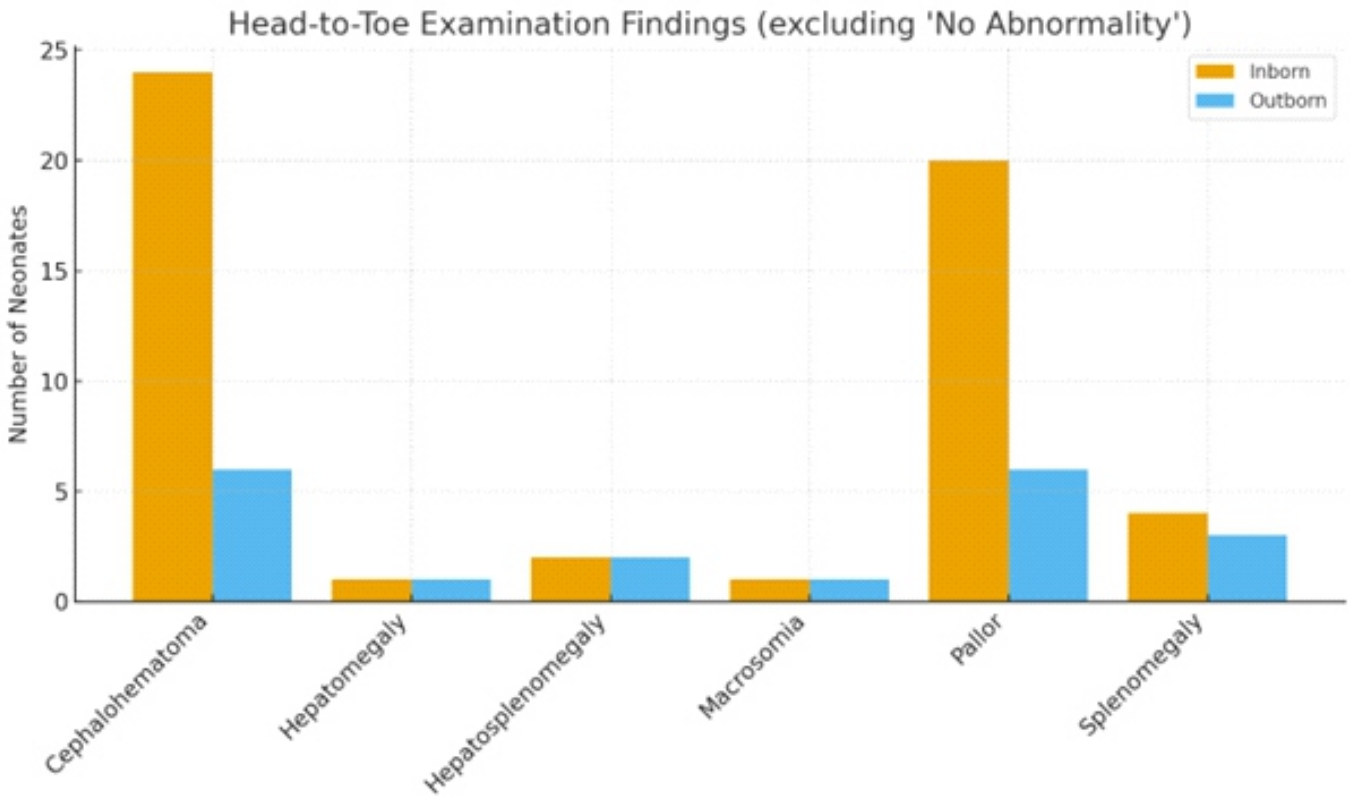

Figure 2: Comparison of head-to-toe examination findings between inborn and outborn neonates with hyperbilirubinemia (excluding normal examinations).

Cephalohematoma and pallor were more common in inborn infants, while splenomegaly and hepatosplenomegaly were more frequent in outborn infants.

DISCUSSION

In this study of 360 neonates admitted with hyperbilirubinemia, pathological jaundice accounted for slightly more than one-third of cases, occurring significantly more often in outborn infants (60.4%) compared with those born within the institution (28.6%). Similar patterns have been reported by Scrafford et al. [4] and Olusanya et al. [6], who noted that neonates delivered outside formal healthcare facilities frequently present later and with more advanced disease due to delayed recognition and limited access to early screening. The higher prevalence of prematurity, small-for-gestational-age (SGA) status, and birth asphyxia among outborn neonates in the present cohort is also supported by prior evidence, with Amin et al. [7] and Huang et al. [8] demonstrating that such perinatal vulnerabilities substantially heighten susceptibility to severe hyperbilirubinemia through impaired bilirubin clearance and increased hemolysis. A clear etiological distinction was observed between the two groups: neonatal sepsis was the leading cause of pathological jaundice among outborn neonates, a finding consistent with the observations of Olusanya et al. [6], whereas hemolytic causes such as ABO incompatibility and cephalohematoma predominated in the inborn group, corroborating the work of Muchowski et al. [3] and Amin et al. [7]. Although mean serum bilirubin concentrations were similar in both groups, bilirubin assessment occurred significantly earlier among inborn neonates. These findings aligned well with the conclusions of **Maisels and Kring** et al. [10], who emphasized the importance of timely bilirubin monitoring in reduction of the progression to severe disease and the need for escalated interventions. Overall, the study highlights the impact of delivery conditions and healthcare availability on the clinical profile and severity of neonatal jaundice progression. This highlighted the requirement for enhanced community-level surveillance, effective infection control practices, early bilirubin monitoring initiatives, and structured referral mechanisms, especiallyx for outborn infants.

In the current analysis, pathological jaundice accounted for 36.7% of cases, with a notably higher prevalence in outborn neonates (60.4%) compared to inborn newborns (28.6%, p=0.001). This pattern is similar to what **Scrafford et al.** [4] and **Olusanya et al**. [6] who reported a higher prevelence of neonatal jaundice and more severe clinical presentation among community-born infants. could be linked to late diagnosis of jaundice, limited availability of structured postnatal follow-up, and late recommendation to tertiary care facilities. These variables may have led to the significantly elevated rates of pathological jaundice among outborn infants in the current study.

Prematurity and small-for-gestational-age (SGA) status were significantly more prevalent among outborn neonates, findings that align with the evidence presented by **Amin et al.** [7] and **Huang et al.** [8], who identified these perinatal vulnerabilities as key determinants of severe hyperbilirubinemia due to immaturity of hepatic conjugation pathways and an increased bilirubin load secondary to hemolysis. Outborn neonates had a higher incidence of birth asphyxia (p=0.02), corroborating the findings of Scrafford et al. [4], which indicated that hypoxia damage disrupts hepatic function, consequently hindering bilirubin removal. These results underscore the necessity of enhancing antenatal care, secure delivery protocols, and prompt neonatal stabilization.

A clear etiological difference was noted for the two groups in terms of neonatal sepsis, which was the principal cause of pathological jaundice among outborn neonates (56.3%, $p<0.001$), supported by the findings of **Olusanya et al.** [6], who underlined the role of infection as a key driver of severe jaundice in low-resource settings. In contrast, hemolytic etiologies including ABO incompatibility (29.8%) and cephalohematoma (31.1%) were more frequently observed among inborn neonates. These results support the work of **Muchowski et al.** [3] and **Amin et al.** [7], who reported that hemolysis and birth trauma constitute major contributors to pathological jaundice in institutional settings.

Mean serum bilirubin levels showed no significant difference between inborn and outborn neonates. Noticeably, bilirubin estimation was performed significantly earlier in inborn neonates (3.09 vs 3.29 days, $p=0.001$). The findings paralleled with the studied from **Maisels and Kring et al.** [10]. They established the association of early bilirubin surveillance with a decreased risk of bilirubin increase, related complications, and readmission. These findings collectively highlight the impact of delivery setting, perinatal care quality, and referral efficiency on the prevalence and severity of neonatal hyperbilirubinemia, underscoring the critical necessity for enhanced community-level screening, improved infection control measures, prompt referral systems, and comprehensive parental education to avert preventable morbidity in outborn neonates.

A significant aspect of this study is its systematic comparison of etiological and clinical parameters related to hyperbilirubinemia between inborn and outborn newborns in a tertiary care setting, supported by a considerable sample size of 360 participants. The combination of comprehensive maternal, perinatal, and neonatal variables for an extensive investigation of various risk factors contributed to strengthening the internal validity. In addition, consistent clinical methods of evaluation and standardized laboratory investigations supported the findings. The study is also significantly relavent for healthcare systems in resource-constrained environments, where the delayed referral of outborn neonates still remains a major hurdle. Nevertheless, some limitations warrant consideration. Being a single-center investigation, the findings may not be representative of wider populations or different levels of care. Dependence on referral records for outborn infants may introduce documentation variability. In addition, the unavailability of genetic testing for bilirubin metabolism disorder & long-

term neurodevelopmental follow-up limits conclusions regarding underlying molecular mechanisms and subsequent outcomes.

CONCLUSION

The results of this study highlight the significant effect of birth setting, perinatal treatment quality, and prompt newborn evaluation on the onset and severity of hyperbilirubinemia. The occurrence of sepsis and hypoxia in outborn newborns underscores the deficiencies in early newborn surveillance and referral efficiency. In addition, the elevated incidence of hemolytic etiologies, including ABO incompatibility and cephalohematoma in neonates, highlights the importance of regular antenatal blood group screening and vigilant intrapartum management. Early institutional bilirubin monitoring is still critical in preventing the levels from rising to the point where an exchange transfusion is needed. Improving coordinated maternal-infant care and developing standardized bilirubin screening techniques could help lower unnecessary illness, especially in places where resources are limited and referrals are needed.

LIMITATIONS & FUTURE PERSPECTIVES

The study's limitations include a single-centre setting, a relatively small sample size, and a short study duration, which may limit the broader applicability of the results. Future studies should incorporate multicentre designs with larger populations to enhance validity, assess long-term outcomes, and investigate advanced diagnostic and management approaches. Such efforts will improve overall patient care and help minimize complications.

CLINICAL SIGNIFICANCE

The clinical significance of this study lies in its potential to bridge the gap between research findings and practical healthcare applications. It emphasizes the importance of trans-lating scientific observations into meaningful improve-ments in patient care, diagnosis, and treatment outcomes. By highlighting real-world relevance, the study contributes to evidence-based medical practice and supports informed clinical decision-making. Ultimately, the findings aim to enhance patient quality of life, optimize therapeutic strategies, and promote better disease management in clinical settings.

ABBREVIATIONS

SNCU: Special Newborn Care Unit
SGA: Small for Gestational Age
TSB: Total Serum Bilirubin
ABO: Blood group system (A, B, O)

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AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by the International Committee of Medical Journal Editors (ICMJE) guidelines.

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CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

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All necessary consent & approval was obtained by authors.

CONSENT FOR PUBLICATION

All necessary consent for publication was obtained by authors.

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All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a re-asonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)

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