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Etiological Spectrum of Neonatal Jaundice in Neonatal Intensive Care Unit in Tertiary Care Hospital

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ABSTRACT

Background: Neonatal jaundice is a frequent cause of hospital admissions in newborns and can result from a range of physiological and pathological conditions. Early identification of its underlying etiology is essential to guide appropriate management and prevent complications, especially in resource-limited settings where diagnostic tools may be limited. **Objective:** To determine the frequency of the causes of neonatal jaundice in the neonatal intensive care unit in tertiary care hospital. **Study Design:** Descriptive cross-sectional study. **Duration and Place of Study:** The study was conducted from January to May 2025 at the Neonatal Intensive Care Unit, Department of Pediatrics, CMH Abbottabad. **Methodology:** A total of 164 neonates under 28 days of age with clinical and biochemical evidence of jaundice were included using non-probability consecutive sampling. Neonates with prior phototherapy, congenital anomalies, or life-threatening illness were excluded. Data were collected on demographic characteristics and laboratory evaluations to establish the etiology of jaundice. **Results:** The most common cause of neonatal jaundice was physiological jaundice (47.6%), followed by ABO incompatibility (25.6%) and Rh incompatibility (11.6%). Statistically significant associations were observed between Rh incompatibility and male gender (p=0.043), ABO incompatibility and rural residence (p=0.007), and physiological jaundice with family history (p=0.021) and urban residence (p<0.001). ABO incompatibility also showed a significant correlation with low birth weight (p=0.049). **Conclusion:** Physiological jaundice remains the most prevalent cause of neonatal jaundice, while Rh and ABO incompatibilities also contribute significantly.

INTRODUCTION

Neonatal jaundice is one of the most frequent clinical presentations seen in neonates with an incidence of nearly 60% in term infants and up to 80% in preterm infants within the newborn period.[1] This yellow staining of the skin and sclera is caused by the accumulation of unconjugated bilirubin within tissues and results from the natural immaturity of the conjugation systems within the liver coupled with the elevated production of bilirubin secondary to increased red blood cell destruction in the neonatal age period.[2] Though most instances are benign physiological jaundice that resolves spontaneously, pathological jaundice necessitates early recognition and intervention to avoid such grave complications as kernicterus that can lead to permanent neurological sequelae such as spastic cerebral palsy, sensorineural hearing loss, and developmental regression.[3] Differentiation between physiological and pathological jaundice is based upon a number of factors such as the time of onset, rate of increase of serum bilirubin concentrations, maximum concentrations reached by the bilirubin, and accompanying clinical findings.[4]

The spectrum of etiology for neonatal jaundice in the neonatal intensive care unit is very wide with physiological jaundice representing the most frequent cause in full-term healthy neonates. Physiological jaundice typically appears after the neonate is 24 hours old, peaks at days 3 to 5, and disappears gradually within the second week in full-term neonates and within the third week in premature neonates.[5] Pathological causes constitute an increasingly larger percentage among the NICU population, particularly the hemolytic disorders ABO and Rh incompatibility that may appear within the neonate's first 24 hours with sharply increased bilirubin concentrations requiring immediate intervention.[6] ABO incompatibility occurs when maternal anti-A or anti-B antibodies cross the placenta and cause hemolysis in infants with incompatible blood types, while Rh incompatibility, though less common due to routine RhoGAM prophylaxis, can result in severe hemolytic disease of the newborn.[7] Idiopathic jaundice represents cases where no specific underlying cause can be identified

despite thorough investigation, often occurring in otherwise healthy infants with prolonged hyperbilirubinemia.[8]

With the other significant etiological factors frequently encountered in the NICU such as cephalohematoma, sepsis, and prematurity having distinct mechanisms for causing hyperbilirubinemia different from that of extravascular hemolysis seen in cephalohematoma caused by trauma at the time of delivery with subperiosteal hemorrhage, the latter results in extravascular hemolysis with the absorbed blood resulting in increased bilirubin load that can overwhelm the newborn conjugation capacity that is yet immature.[9] Neonatal sepsis early or late can induce jaundice with multifactorial mechanisms such as hemolysis, hepatocellular damage, and defective conjugation of bilirubin with a range of typical presenting clinical Features with the necessity for keen analysis and management.[10] Premature status is a very significant risk factor owing to the fact that the premature neonate has an immature hepatic enzymatic machinery, is very susceptible to hemolysis, late commencement of feeds with resultant lower clearance of bilirubin, and higher complication rates such as sepsis and trauma at the time of delivery.[11] The management approach in the NICU is meticulous consideration of these multifactorial causative factors with a range of strategies such as phototherapy and exchange transfusions to the management approach for predisposing factors such as sepsis or hemolytic disease with very close monitoring to prevent the onset of bilirubin-induced neurologic damage.[12]

A study conducted by Zaman BU et al. reported the distribution of underlying causes among neonates admitted with jaundice in a neonatal intensive care unit. Physiological jaundice was observed in 40.5% of cases, while ABO incompatibility accounted for 20%, followed by Rh incompatibility at 16.5%. Idiopathic causes contributed to 5% of cases, with cephalohematoma and prematurity each identified in 4% of neonates. Additionally, sepsis was found in 8% of the affected infants.[13]

Neonatal jaundice is a common clinical phenomenon but its cause can be very diverse and can have severe implications if not diagnosed and managed appropriately. Identification of the spectrum of causes is imperative for early diagnosis, appropriate therapy, and the prevention of complications such as kernicterus.

METHODOLOGY

This descriptive cross-sectional analysis was undertaken in the Neonatal Intensive Care Unit of the Department of Pediatrics at CMH Abbottabad, spanning from January to May 2025. A total of 164 neonates were enrolled. The sample size was estimated using the WHO software with a 95% confidence interval, 3% margin of error, and an anticipated frequency of prematurity in jaundiced neonates set at 4%.[13]

Participants were recruited through a non-probability consecutive sampling method. Eligible neonates included both male and female infants under 28 days of age who were admitted with jaundice, characterized by visible yellow discoloration of the skin confirmed after blanching with digital pressure and supported by a serum bilirubin level exceeding 13 mg/dL. Neonates were excluded if they had prior phototherapy before hospital admission, any congenital structural abnormalities such as biliary atresia, life-threatening illness, or if they were discharged or died within 24 hours of admission.

Parental consent was obtained after ethics committee approval (CMH-Atd-ETH-183-Paeds-24). For each neonate, relevant background information—including chronological age, sex, birth weight, family history and residential status was recorded on a structured data form. To identify the contributing cause of jaundice, each case was reviewed through physical examination findings, laboratory investigations, and imaging reports. Physiological jaundice was identified in full-term neonates if yellowing appeared after the first 24 hours, serum bilirubin levels remained below 12 mg/dL in term or 15 mg/dL in preterm infants, and the condition resolved within one to two weeks in term or within two to three weeks in preterm infants. ABO incompatibility was diagnosed when the neonate's blood group was A, B, or AB and the mother had blood group O. Rh incompatibility was recorded in cases where the mother was Rh-negative and the infant Rh-positive.

Jaundice was labeled idiopathic when it emerged after 24 hours of birth, with bilirubin levels exceeding 12 mg/dL in term or 15 mg/dL in preterm neonates, without an identifiable underlying cause, and persisted beyond 14 days in term or 21 days in preterm infants. Cephalohematoma was confirmed via imaging if two or more of the following features were present: a crescent-shaped area adjacent to the outer skull table, high-attenuation areas exceeding +100 Hounsfield units indicating possible calcification, or bone remodeling signs such as cortical thickening or periosteal reaction. Neonatal sepsis was diagnosed when the infant presented with a fever above 38°C along with at least one laboratory abnormality—white blood cell differential count over 20%, micro ESR greater than 55 mm/hr, or gastric aspirate showing more than five polymorphs per high-power field. Prematurity was defined as delivery occurring before completion of 37 weeks of gestation.

Data entry and analysis were conducted using SPSS version 26. Descriptive statistics were employed to summarize variables. Quantitative variables were expressed as mean ± standard deviation or median with interquartile ranges, depending on normality checked using the Shapiro-Wilk test. Categorical variables were summarized as frequencies and percentages. Stratification was done by demographic variables to assess the distribution of jaundice etiologies. Associations were evaluated using chi-square or Fisher's exact test where appropriate, with a p-value ≤ 0.05 considered statistically significant.

RESULTS

Patient demographics reveal that the mean age of the neonates was 15.646 ± 8.229 days, with a mean weight of 2.781 ± 0.493 kg (as shown in Table 1). Among the 164 neonates, 100 (61.0%) were male and 64 (39.0%) were female. The majority of the neonates resided in urban areas, with 88 (53.7%) from urban regions and 76 (46.3%) from rural areas. A family history of jaundice was present in 45 (27.4%) neonates, while 119 (72.6%) had no such

history.

Table 1

Patient Demographics

Demographics		Mean ± SD or n (%)
Age (days)		15.646 ± 8.229
Weight (Kg)		2.781 ± 0.493
Gender	Male n (%)	100 (61.0%)
	Female n (%)	64 (39.0%)
Residential Status	Rural n (%)	76 (46.3%)
	Urban n (%)	88 (53.7%)
Family History of Jaundice	Yes n (%)	45 (27.4%)
	No n (%)	119 (72.6%)

The frequency of etiological factors in neonatal jaundice is detailed in Table 2. Physiological jaundice was the most common, occurring in 78 (47.60%) neonates. ABO incompatibility was identified in 42 (25.60%) cases, while Rh incompatibility was less frequent, found in 19 (11.60%) neonates. Idiopathic jaundice was present in 10 (6.10%) cases, cephalohematoma in 6 (3.70%), sepsis in 15 (9.10%), and prematurity in 7 (4.30%).

Table 2

Frequency of Etiological Factors in Neonatal Jaundice

Etiological Factors		Frequency	Percentage
Physiological Jaundice	Yes	78	47.60%
	No	86	52.40%
ABO Incompatibility	Yes	42	25.60%
	No	122	74.40%
Rh Incompatibility	Yes	19	11.60%
	No	145	88.40%
Idiopathic Jaundice	Yes	10	6.10%
	No	154	93.90%
Cephalohematoma	Yes	6	3.70%
	No	158	96.30%
Sepsis	Yes	15	9.10%
	No	149	90.90%
Prematurity	Yes	7	4.30%
	No	157	95.70%

The association of etiological factors with demographic factors is presented in Table 3. For age group, neonates ≤15 days had physiological jaundice in 41 (50.6%) cases, with a p-value of 0.439. ABO incompatibility was present in 21 (25.9%) neonates, with a p-value of 0.927. Rh incompatibility occurred in 12 (14.8%) neonates, with a p-value of 0.202. Idiopathic jaundice was found in 4 (4.9%) neonates, with a p-value of 0.746. Cephalohematoma was present in 5 (6.2%) neonates, with a p-value of 0.115. Sepsis was identified in 6 (7.4%) neonates, with a p-value of 0.445. Prematurity occurred in 1 (1.2%) neonate, with a p-value of 0.117. For neonates >15 days, the frequencies and p-values were as follows: physiological jaundice in 37 (44.6%) neonates, p-value 0.439; ABO incompatibility in 21 (25.3%) neonates, p-value 0.927; Rh incompatibility in 7 (8.4%) neonates, p-value 0.202; idiopathic jaundice in 6 (7.2%) neonates, p-value 0.746; cephalohematoma in 1 (1.2%) neonate, p-value 0.115; sepsis in 9 (10.8%) neonates, p-value 0.445; and prematurity in 6 (7.2%) neonates, p-value 0.117.

Regarding gender, male neonates had physiological jaundice in 44 (44.0%) cases, with a p-value of 0.254. ABO incompatibility was present in 25 (25.0%) male neonates, with a p-value of 0.823. Rh incompatibility occurred in 16 (16.0%) male neonates, with a p-value of 0.043. Idiopathic jaundice was found in 4 (4.0%) male neonates, with a p-value of 0.19. Cephalohematoma was present in 6 (6.0%) male neonates, with a p-value of 0.082. Sepsis was identified in 7 (7.0%) male neonates, with a p-value of 0.233. Prematurity occurred in 7 (7.0%) male neonates, with a p-value of 0.043. In female neonates, the frequencies and p-values were as follows: physiological jaundice in 34 (53.1%) neonates, p-value 0.254; ABO incompatibility in 17 (26.6%) neonates, p-value 0.823; Rh incompatibility in 3 (4.7%) neonates, p-value 0.043; idiopathic jaundice in 6 (9.4%) neonates, p-value 0.19; cephalohematoma in 0 (0.0%) neonates, p-value 0.082; sepsis in 8 (12.5%) neonates, p-value 0.233; and prematurity in 0 (0.0%) neonates, p-value 0.043.

For weight group, neonates ≤2 kg had physiological jaundice in 4 (44.4%) cases, with a p-value of 1. ABO incompatibility was present in 5 (55.6%) neonates, with a p-value of 0.049. Rh incompatibility occurred in 0 (0.0%) neonates, with a p-value of 0.391. Idiopathic jaundice was found in 2 (22.2%) neonates, with a p-value of 0.096. Cephalohematoma was present in 0 (0.0%) neonates, with a p-value of 1.000. Sepsis was identified in 1 (11.1%) neonate, with a p-value of 1.000. Prematurity occurred in 0 (0.0%) neonates, with a p-value of 1.000. In neonates >2 kg, the frequencies and p-values were as follows: physiological jaundice in 74 (47.7%) neonates, p-value 1; ABO incompatibility in 37 (23.9%) neonates, p-value 0.049; Rh incompatibility in 19 (12.3%) neonates, p-value 0.391; idiopathic jaundice in 8 (5.2%) neonates, p-value 0.096; cephalohematoma in 6 (3.9%) neonates, p-value 1.000; sepsis in 14 (9.0%) neonates, p-value 1.000; and prematurity in 7 (4.5%) neonates, p-value 1.000.

In terms of residential status, rural neonates had physiological jaundice in 25 (32.9%) cases, with a p-value of <0.001. ABO incompatibility was present in 27 (35.5%) rural neonates, with a p-value of 0.007. Rh incompatibility occurred in 7 (9.2%) rural neonates, with a p-value of 0.377. Idiopathic jaundice was found in 4 (5.3%) rural neonates, with a p-value of 0.753. Cephalohematoma was present in 3 (3.9%) rural neonates, with a p-value of 1.000. Sepsis was identified in 4 (5.3%) rural neonates, with a p-value of 0.173. Prematurity occurred in 2 (2.6%) rural neonates, with a p-value of 0.425. Urban neonates had the following frequencies and p-values: physiological jaundice in 53 (60.2%) neonates, p-value <0.001; ABO incompatibility in 15 (17.0%) neonates, p-value 0.007; Rh incompatibility in 12 (13.6%) neonates, p-value 0.377; idiopathic jaundice in 6 (6.8%) neonates, p-value 0.753; cephalohematoma in 3 (3.4%) neonates, p-value 1.000; sepsis in 11 (12.5%) neonates, p-value 0.173; and prematurity in 5 (5.7%) neonates, p-value 0.425.

Family history of jaundice also showed significant associations. Neonates with a family history had physiological jaundice in 28 (62.2%) cases, with a p-value of 0.021. ABO incompatibility was present in 12 (26.7%) neonates, with a p-value of 0.849. Rh incompatibility occurred in 5 (11.1%) neonates, with a p-value of 1. Idiopathic jaundice was found in 2 (4.4%) neonates, with a p-value of 0.729. Cephalohematoma was present in 0 (0.0%) neonates, with a p-value of 0.19. Sepsis was identified in 2 (4.4%) neonates, with a p-value of 0.242.

Prematurity occurred in 2 (4.4%) neonates, with a p-value of 1.000. In neonates without a family history, the frequencies and p-values were as follows: physiological jaundice in 50 (42.0%) neonates, p-value 0.021; ABO incompatibility in 30 (25.2%) neonates, p-value 0.849; Rh incompatibility in 14 (11.8%) neonates, p-value 1; idiopathic jaundice in 8 (6.7%) neonates, p-value 0.729; cephalohematoma in 6 (5.0%) neonates, p-value 0.19; sepsis in 13 (10.9%) neonates, p-value 0.242; and prematurity in 5 (4.2%) neonates, p-value 1.000.

Table 3

Association of Etiological Factors with Demographic Factors

Demographic Factors	Etiological Factors													
	PJ	p-value	AI	p-value	RI	p-value	IJ	p-value	CH	p-value	S	p-value	P	p-value
Age Group (days)														
≤15	41 (50.6%)	0.439	21 (25.9%)	0.927	12 (14.8%)	0.202	4 (4.9%)	0.746	5 (6.2%)	0.115	6 (7.4%)	0.445	1 (1.2%)	0.117
>15	37 (44.6%)		21 (25.3%)		7 (8.4%)		6 (7.2%)		1 (1.2%)		9 (10.8%)		6 (7.2%)	
Gender														
Male	44 (44.0%)	0.254	25 (25.0%)	0.823	16 (16.0%)	0.043	4 (4.0%)	0.19	6 (6.0%)	0.082	7 (7.0%)	0.233	7 (7.0%)	0.043
Female	34 (53.1%)		17 (26.6%)		3 (4.7%)		6 (9.4%)		0 (0.0%)		8 (12.5%)		0 (0.0%)	
Weight Group (Kg)														
≤2	4 (44.4%)	1	5 (55.6%)	0.049	0 (0.0%)	0.391	2 (22.2%)	0.096	0 (0.0%)	1.000	1 (11.1%)	1.000	0 (0.0%)	1.000
>2	74 (47.7%)		37 (23.9%)		19 (12.3%)		8 (5.2%)		6 (3.9%)		14 (9.0%)		7 (4.5%)	
Residential Status														
Rural	25 (32.9%)	<0.001	27 (35.5%)	0.007	7 (9.2%)	0.377	4 (5.3%)	0.753	3 (3.9%)	1.000	4 (5.3%)	0.173	2 (2.6%)	0.425
Urban	53 (60.2%)		15 (17.0%)		12 (13.6%)		6 (6.8%)		3 (3.4%)		11 (12.5%)		5 (5.7%)	
Family History of Jaundice														
Yes	28 (62.2%)	0.021	12 (26.7%)	0.849	5 (11.1%)	1	2 (4.4%)	0.729	0 (0.0%)	0.19	2 (4.4%)	0.242	2 (4.4%)	1.000
No	50 (42.0%)		30 (25.2%)		14 (11.8%)		8 (6.7%)		6 (5.0%)		13 (10.9%)		5 (4.2%)	

PJ: Physiological Jaundice, AI: ABO Incompatibility, RI: Rh Incompatibility, IJ: Idiopathic Jaundice, CH: Cephalohematoma, S: Sepsis, P: Prematurity.

DISCUSSION

The findings from this study provide valuable insights into the etiological spectrum of neonatal jaundice and its association with various demographic factors. The high prevalence of physiological jaundice, which was the most common cause identified, is consistent with its natural occurrence in the neonatal period due to the immaturity of the liver's ability to metabolize bilirubin. The significant association of Rh incompatibility with male gender (p-value 0.043) may be attributed to the higher incidence of hemolytic diseases in males, potentially due to genetic factors and the influence of sex hormones on immune responses. The higher frequency of ABO incompatibility in urban neonates (p-value 0.007) could be related to differences in genetic predisposition and environmental factors between urban and rural populations. The lack of significant association between family history of jaundice and most etiological factors suggests that while genetic factors may play a role, environmental and physiological factors are more dominant in the development of neonatal jaundice. These results highlight the multifactorial nature of neonatal jaundice and underscore the importance of considering both demographic and physiological factors in its management and prevention.

Our study findings demonstrate a consistent pattern with the existing literature regarding the predominant causes of neonatal jaundice. Physiological jaundice emerged as the most common etiology in our cohort, affecting 78 (47.60%) neonates, which aligns closely with several previous studies. This finding is remarkably similar to the study by Ihsan-ul-Haq et al. [13] and closely matches Davis Manuel and Shajahan R A [14] where physiological jaundice accounted for 45% of cases, and Garg Paridhi et al. [15] who reported 44.4% of cases. Additionally, our results are consistent with Akash Gupta et al. [16] who found physiological jaundice in 35.00% of cases, and Syed Adnan Ali et al. [17] who reported 66.67% of cases being physiological. The consistency across these studies reinforces the understanding that physiological jaundice remains the leading cause of neonatal jaundice in term infants, reflecting the normal adaptation process of neonatal liver function and bilirubin metabolism.

ABO incompatibility was identified as the second most common cause in our study, occurring in 42 (25.60%) neonates. This finding is consistent with multiple studies from the Indian subcontinent and Middle East. Our results closely match those reported by Davis Manuel and Shajahan R A [14] who found ABO incompatibility in 25% of cases, and are nearly identical to the findings by Jasraj Bohra et al. [18] who reported 24.4% of cases. Similarly, Dr. Ahmad Ali and Dr. Anurag Tomar [19] and Dr. Mohamed Fawzy Meslhy et al. [20] both reported ABO incompatibility in 32.7% of cases, slightly higher than our findings. Ihsan-ul-Haq et al. [13] found ABO incompatibility in 31.84% of cases, and Saeed Ahmad Malik et al. [21] reported 13% of cases. The geographic clustering of these high ABO incompatibility rates in South Asian studies suggests regional genetic factors and blood group distribution patterns that predispose to this etiology.

Interestingly, our study revealed a significant association between ABO incompatibility and low birth weight neonates (≤2 kg), with 55.6% of cases in this weight group having ABO incompatibility (p-value 0.049). This finding suggests that low birth weight neonates may be more susceptible to hemolytic disease, possibly due to immature hepatic function and reduced capacity for bilirubin conjugation. Additionally, we found a higher prevalence of ABO incompatibility in rural neonates (35.5%) compared to urban neonates (17.0%) with a significant p-value of 0.007, which may reflect differences in antenatal care, early detection, or genetic population characteristics between urban and rural areas.

Rh incompatibility accounted for 19 (11.60%) cases in our study, which is consistent with several previous studies. Our findings align with Garg Paridhi et al. [15] who reported similar rates, and are comparable to Ihsan-ul-Haq et al. [13] who found Rh incompatibility in 13.45% of cases. Jasraj Bohra et al. [18] found 13.8% of cases, while Davis Manuel and Shajahan R A [14] reported 8% of cases. However, our rates are lower than those reported by some studies, which may reflect differences in population genetics, effectiveness of Rh immunoglobulin prophylaxis programs, or improved antenatal care practices. Notably, our study demonstrated a significant gender association, with Rh incompatibility more common in male neonates (16.0%) compared to female neonates (4.7%) with a p-value of 0.043, which warrants further investigation into potential gender-specific susceptibility factors.

Sepsis was identified in 15 (9.10%) neonates in our study, which shows varying concordance with previous studies. Our findings are consistent with Garg Paridhi et al. [15] who reported sepsis in 12% of cases, and Syed Adnan Ali et al. [17] who found septicemia in 12.86% of cases. However, our results differ from Muhammad Omer et al. [22] who reported neonatal sepsis in 22% of cases of persistent jaundice, and contrast sharply with Dr. Arjun Tandon et al. [23] who found sepsis to be the most common cause of conjugated jaundice at 51.1%. This discrepancy likely reflects the different study populations, as Tandon et al. [23] specifically studied conjugated jaundice, while our study included all types of neonatal jaundice. The lower sepsis rate in our study may also indicate better infection control practices or early antibiotic intervention in our healthcare setting.

Idiopathic jaundice was present in 10 (6.10%) cases in our study, which is consistent with Davis Manuel and Shajahan R A [14] who reported 8% idiopathic cases. Our findings are also comparable to Muhammad Kabir et al. [24] who found idiopathic causes in 15% of cases with persistent jaundice, and Muhammad Omer et al. [22] who reported idiopathic neonatal hepatitis in 28% of persistent jaundice cases. The variation in idiopathic rates likely reflects differences in diagnostic capabilities and study populations, with higher rates in persistent jaundice studies suggesting that prolonged cases are more likely to remain undiagnosed.

Cephalohematoma was identified in 6 (3.70%) cases, closely matching the 4% reported by Davis Manuel and Shajahan R A.[14] Interestingly, our study found cephalohematoma exclusively in male neonates (6.0% vs. 0.0% in females, p-value 0.082), which may reflect differences in birth trauma susceptibility or delivery complications between genders.

Prematurity accounted for 7 (4.30%) cases in our study, which is consistent with the 5% reported by Davis Manuel and Shajahan R A.[14] Similar to cephalohematoma, prematurity showed a significant gender association, occurring only in male neonates (7.0% vs. 0.0% in females, p-value 0.043), suggesting potential gender-specific risk factors or differences in gestational age distribution.

Our study revealed several significant demographic associations that provide valuable insights into neonatal jaundice epidemiology. The urban-rural distribution showed marked differences, with physiological jaundice being more common in urban neonates (60.2%) compared to rural neonates (32.9%) with a p-value <0.001. Conversely, ABO incompatibility was more prevalent in rural areas (35.5%) compared to urban areas (17.0%) with a p-value of 0.007. These findings suggest that healthcare access, antenatal care quality, and early detection programs may differ between urban and rural settings, influencing the apparent distribution of jaundice etiologies.

The family history association was particularly noteworthy, with neonates having a family history of jaundice showing higher rates of physiological jaundice (62.2% vs. 42.0%, p-value 0.021). This finding suggests genetic predisposition factors that may influence bilirubin metabolism and clearance, warranting further investigation into hereditary components of neonatal jaundice.

The consistency of our findings with the existing literature from similar geographic regions reinforces the importance of developing region-specific guidelines for neonatal jaundice management. The predominance of physiological jaundice followed by ABO incompatibility suggests that screening protocols should prioritize these conditions while maintaining vigilance for less common but more serious causes like sepsis and metabolic disorders.

Our study notably did not identify G6PD deficiency as a separate category, which contrasts with Ihsan-ul-Haq et al. [13] who found G6PD deficiency in 28.35% of cases. This difference may reflect regional variations in genetic prevalence or different screening practices, highlighting the importance of region-specific enzyme deficiency screening programs as suggested by Garg Paridhi et al. [15]

The significant demographic associations identified in our study highlight the need for tailored approaches to neonatal jaundice management based on geographic, socioeconomic, and genetic factors. Future research should focus on understanding the mechanisms underlying these associations and developing targeted interventions to improve outcomes across different population groups.

While our study provides valuable insights into the etiological spectrum of neonatal jaundice, it should be interpreted within the context of its limitations. The single-center design may limit generalizability, and the relatively small sample size of 164 neonates may not capture rare etiologies. However, the detailed demographic analysis and statistical associations provide important insights that complement the existing literature and contribute to our understanding of neonatal jaundice epidemiology in this population.

CONCLUSION

This study reveals that neonatal jaundice is predominantly physiological in nature, reflecting the normal developmental immaturity of hepatic bilirubin metabolism. The significant male predominance in Rh incompatibility and urban clustering of ABO incompatibility suggest important demographic risk factors that warrant targeted screening approaches. While genetic predisposition appears less influential than previously assumed, the multifactorial etiology of neonatal jaundice emphasizes the need for comprehensive, demographically-informed clinical assessment and management strategies in neonatal care.

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