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CAUSES, RISK FACTORS AND COMPLICATIONS OF NEONATAL JAUNDICE: A HOSPITAL-BASED STUDY

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ABSTRACT

Background: Neonatal jaundice is one of the most prevalent clinical disorders encountered during the neonatal period, accounting for a significant proportion of neonatal hospital admissions globally. **Objectives:** To identify the causes, risk factors, and complications of newborn jaundice among neonates admitted to Al-Khansaa Teaching Hospital. **Methods:** This one-year cross-sectional study was carried out in the neonatal unit of Al-Khansaa Teaching Hospital in Mosul, Iraq, from January 2024 to December 2024. All newborns aged 28 days or younger who were admitted with clinical jaundice during the study period were eligible to participate. **Results:** The study includes 420 neonates with jaundice. The mean age at presentation was 3.9 ± 1.6 days and 96 neonates (22.9%) had a birth weight of less than 2500 gram. Moreover, 236 (56.2%) were males and 184 (43.8%) were females. With male to female ratio of 1.282:1. Term neonate accounted for 302 cases (71.9%), while 118 neonates (28.1%) were preterm. Physiological jaundice was the most frequent diagnosis, accounting for 262 cases (62.4%), while pathological jaundice was reported in 158 infants (37.6%). Pathological jaundice was significantly more prevalent in preterm infants than in term neonates (P value <0.001). In terms of severity, 262 neonates (62.4%) had mild to moderate hyperbilirubinemia (<15 mg/dL), while 158 had severe hyperbilirubinemia (>15 mg/dL). Severe hyperbilirubinemia has been associated to pathological causes of jaundice (P value < 0.001). ABO incompatibility was the commonest cause of pathological jaundice, found in 45 (28.5%) patients followed by G6PD deficiency in 37 (23.4%) patients, neonatal sepsis in 32 (20.3%) patients, prematurity-related jaundice in 24 (15.2%) patients, Rh incompatibility in 12 (7.6%) patients and cephalhematoma in 8 (5%) patients. Neonatal jaundice complications occurred in 22 (5.2%) patients. 15 (3.6%) patients required an exchange transfusion, while 7 (1.6%) patients suffered kernicterus. The two complications were significantly more common among patients with hemolytic causes and severe hyperbilirubinemia (P value < 0.001). Lastly, neonates who have mothers with gestational diabetes mellitus was significant have higher pathological jaundice (P value = 0.035). Of note, other variables (blood group O, Rh negative and previous neonatal jaundice was trend to be significant). **Conclusion:** Pathological newborn jaundice accounted for a significant proportion of cases and was significantly linked to preterm, hemolytic causes (especially ABO incompatibility and G6PD deficiency), severe hyperbilirubinemia, and maternal gestational diabetes. Severe cases were associated with sequelae such as exchange transfusions and kernicterus, emphasizing the persistent clinical burden of preventable bilirubin neurotoxicity.

KEYWORDS: Jaundice, Mosul, Newborn, Pathological, Physiological.

1-INTRODUCTION

Neonatal jaundice is one of the most prevalent clinical disorders encountered during the neonatal period, accounting for a significant proportion of neonatal hospital admissions globally.[1] It is distinguished by yellowish skin and sclera as a result of unconjugated bilirubin buildup in the blood.[2] The majority of neonates have physiological jaundice as a result of increased red blood cell turnover, immature hepatic absorption and conjugation of bilirubin, and enhanced

enterohepatic circulation. In contrast, pathological jaundice is caused by identified underlying causes such as hemolytic illnesses, infections, metabolic problems, or liver malfunction, and it can escalate quickly to fatal levels.[3-4]

Hyperbilirubinemia is of clinical importance because bilirubin is neurotoxic at high levels. Severe neonatal jaundice, if left untreated, can cause acute bilirubin encephalopathy and kernicterus, both of which are linked to irreversible neurological damage such as cerebral palsy, hearing loss, and cognitive impairment. Early detection and treatment of newborn jaundice are therefore critical to avoiding these catastrophic consequences.[5-6]

The causes and patterns of newborn jaundice differ depending on geographic region, genetic background, G6PD deficiency prevalence, prenatal and postnatal treatment quality, and availability of diagnostic facilities.[7] In countries with limited resources, delayed presentation, home delivery, and restricted access to laboratory investigations raise the risk of severe hyperbilirubinemia and accompanying consequences.[8]

The aim of study is to identify the causes, risk factors, and complications of newborn jaundice among neonates admitted to Al-Khansaa Teaching Hospital.

2-PATIENTS AND METHODS

This one-year cross-sectional study was carried out in the neonatal unit of Al-Khansaa Teaching Hospital in Mosul, Iraq, from January 2024 to December 2024. All newborns aged 28 days or younger who were admitted with clinical jaundice during the study period were eligible to participate.

Demographic information gathered included gender, gestational age, birth weight, mode of delivery, and the age onset of jaundice. Maternal information includes age, blood group, Rh status, parity, past newborn jaundice, diabetes mellitus, and a history of infection while pregnant. The clinical evaluation of neonates included the degree of jaundice, feeding pattern, pallor, hepatosplenomegaly, and neurological symptoms suggestive of bilirubin encephalopathy.

Total and direct serum bilirubin, hemoglobin concentration, mother and newborn blood group and Rh typing, direct Coombs test, C-reactive protein, and blood culture were the laboratory tests performed when neonatal sepsis was suspected. The G6PD level was tested in neonates who had unexplained jaundice or a family history of hemolytic diseases. Additional investigations were conducted as clinically necessary.

Neonatal jaundice was defined as physiological when it started after 24 hours of life, progressed gradually, and disappeared without indication of underlying disease. Pathological jaundice was identified when jaundice started during the first 24 hours, bilirubin levels climbed fast, lasted more than 14 days, or was linked to an established pathological etiology. Pathological causes were further classified as hemolytic (ABO and Rh incompatibility, G6PD deficiency), infectious (neonatal sepsis), prematurity-related jaundice, birth trauma, and breastfeeding-associated jaundice. Neonatal jaundice complications included the requirement for an exchange transfusion or the development of kernicterus-like clinical features.

The statistical analysis was carried out using SPSS software (Statistical Packages for Social Sciences, version 30). The data were presented as frequencies and percentages. The chi-square test was performed to determine difference between groups of variables. A p-value of <0.05 was considered statistically significant.

3- RESULTS

The study includes 420 neonates with jaundice. The mean age at presentation was 3.9 ± 1.6 days and 96 neonates (22.9%) had a birth weight of less than 2500 gram. Moreover, 236 (56.2%) were males and 184 (43.8%) were females. With male to female ratio of 1.282:1. As shown in figure 1.

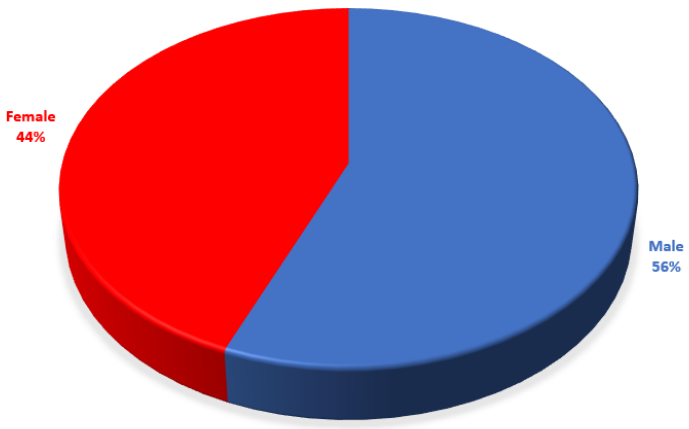


Figure 1: Distribution of the study patients according to their gender.

Term neonate accounted for 302 cases (71.9%), while 118 neonates (28.1%) were preterm. As shown in figure 2.

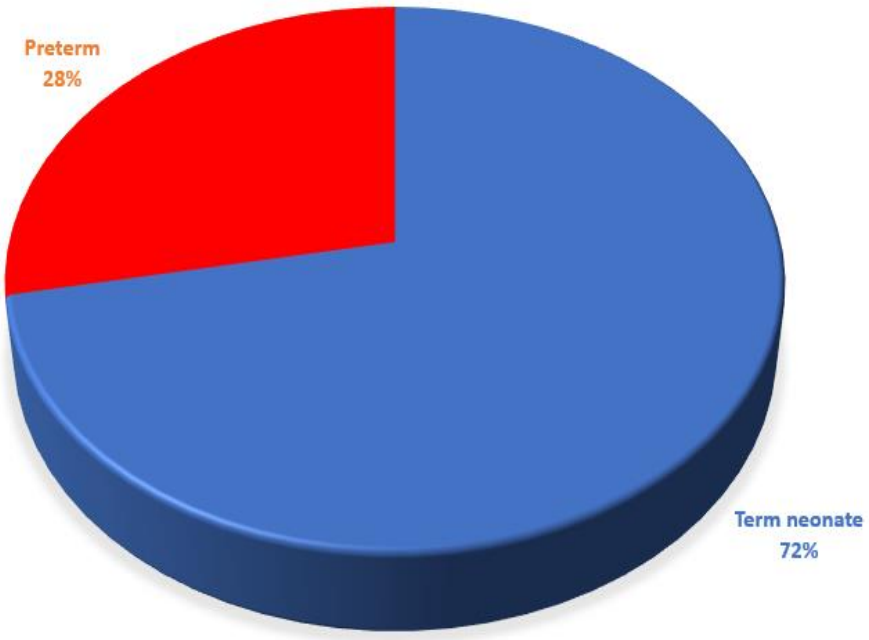


Figure 2: Distribution of the study patients according to their gestational age.

Physiological jaundice was the most frequent diagnosis, accounting for 262 cases (62.4%), while pathological jaundice was reported in 158 infants (37.6%). Pathological jaundice was significantly more prevalent in preterm infants than in term neonates (P value <0.001). As shown in table 1.

Table 1: Comparison between patients with physiological and pathological jaundice regarding their basic characteristics (number =420).

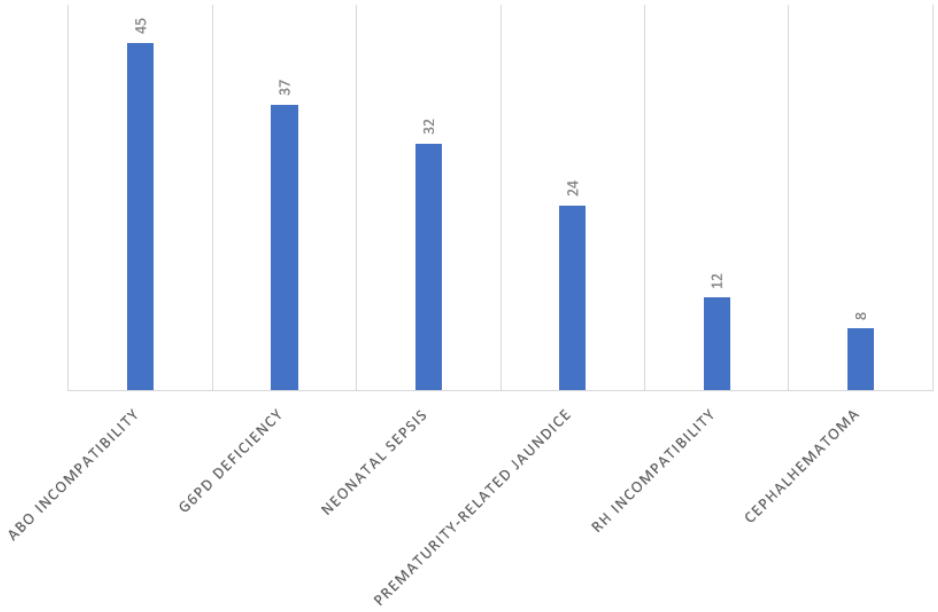
Variables	Physiological jaundice, number = 262	Pathological jaundice, number = 158	P value
Gender, number (%):			
-Male	150 (57.3%)	86 (54.4%)	0.482
-Female	112 (42.7%)	72 (45.6%)	
Gestational age, number (%):			
-Term	215 (82.1%)	87 (55.1%)	**<0.001**
-Preterm	47 (17.9%)	71 (44.9%)	

In terms of severity, 262 neonates (62.4%) had mild to moderate hyperbilirubinemia (<15 mg/dL), while 158 had severe hyperbilirubinemia (>15 mg/dL). Severe hyperbilirubinemia has been associated to pathological causes of jaundice (P value < 0.001).

Table 2: Comparison between patients with physiological and pathological jaundice regarding their bilirubin severity (number =420).

Variables	Physiological jaundice, number = 262	Pathological jaundice, number = 158	P value
Hyperbilirubinemia, number (%):			
-Mild to moderate (<15 mg/dL)	201 (76.7%)	61 (38.6%)	**<0.001**
-Severe (>15 mg/dL)	61 (23.3%)	97 (61.4%)	

Figure 3 shows the different causes of pathological jaundice. ABO incompatibility was the commonest cause, found in 45 (28.5%) patients followed by G6PD deficiency in 37 (23.4%) patients, neonatal sepsis in 32 (20.3%) patients, prematurity-related jaundice in 24 (15.2%) patients, Rh incompatibility in 12 (7.6%) patients and cephalhematoma in 8 (5%) patients.



Neonatal jaundice complications occurred in 22 (5.2%) patients. 15 (3.6%) patients required an exchange transfusion, while 7 (1.6%) patients suffered kernicterus. The two complications were significantly more common among patients with hemolytic causes and severe hyperbilirubinemia (P value < 0.001).

Table 3 shows neonate who have mothers with gestational diabetes mellitus was significant have higher pathological jaundice (P value = 0.035). Of note, other variables (blood group O, Rh negative and previous neonatal jaundice was trend to be significant).

Table 3: Comparison between patients with physiological and pathological jaundice regarding their maternal risk factors (number =420).

Variables	Physiological jaundice, number = 262	Pathological jaundice, number = 158	P value
Blood group O, number (%):	98 (37.4%)	78 (49.4%)	0.086
Rh negative, number (%):	18 (6.9%)	22 (13.9%)	0.106
Gestational diabetes mellitus, number (%):	20 (7.6%)	28 (17.7%)	0.035
Previous neonatal jaundice, number (%):	22 (8.4%)	28 (17.7%)	0.062

4. DISCUSSION

Neonatal jaundice is still one of the most frequent clinical disorders seen during the early newborn period, affecting up to 60% of term and 80% of preterm neonates globally, with a lesser number developing clinically severe hyperbilirubinemia that necessitates intervention.[9-10] The study of 420 neonates found that the mean age of presentation (3.9 ± 1.6 days) is consistent with the natural progression of early neonatal hyperbilirubinemia, which typically peaks between the third and fifth days of life in term infants.[9]

A male predominance (56.2%) was found, with a male-to-female ratio of 1.28:1. Although gender was not significantly associated with pathological jaundice in this group, earlier studies suggest that male sex may be a minor risk factor for severe hyperbilirubinemia, probably due to genetic and hormonal influences on bilirubin metabolism.[10-11] Similar male preponderance has been observed in regional and international study, however the relationship is not always statistically significant.[11]

Preterm neonates made up 28.1% of the study sample and had considerably higher rates of pathological jaundice than term newborns. This finding is consistent with previous studies.[9,12] Indicating that prematurity is a major risk factor for severe hyperbilirubinemia due to hepatic immaturity, reduced bilirubin conjugation, increased enterohepatic circulation, and increased vulnerability to hemolysis. The American Academy of Pediatrics' 2022 clinical practice guideline stresses lower treatment thresholds and greater monitoring in preterm and near-term newborns due to their heightened vulnerability to bilirubin neurotoxicity.[9]

Physiological jaundice was found in 62.4% of neonates, whereas 37.6% had pathological jaundice. Severe hyperbilirubinemia (>15 mg/dL) was substantially

related with pathogenic reasons. This aligns with the pathophysiological concept that hemolysis, infection, and metabolic abnormalities are major contributors to fast rising or markedly raised bilirubin levels.[9,13] Studies from low- and middle-income countries have found a significant prevalence of pathological jaundice in hospitalized neonates, which is frequently associated with avoidable causes and delayed presentation.[13]

The most common pathogenic cause was ABO incompatibility (28.5%), followed by G6PD deficiency (23.4%) and newborn sepsis (20.3%). ABO incompatibility is still one of the leading causes of immune-mediated hemolytic disease in neonates, particularly in populations with a high prevalence of maternal blood group O.[14] G6PD deficiency is especially important in Middle Eastern populations, where it is relatively common and represents a well-known risk factor for severe neonatal hyperbilirubinemia and kernicterus.[15] Sepsis-related jaundice is caused by both hemolysis and decreased hepatic function, and it has repeatedly been linked to more severe illness and worse outcomes.[13]

Complications were reported in 5.2% of cases, including exchange transfusion (3.6%) and kernicterus (1.6%). These problems were substantially more likely in newborns with hemolytic causes and severe hyperbilirubinemia. Despite advances in screening and phototherapy, kernicterus is still being reported, especially in resource-limited areas where delayed detection and inadequate follow-up remain difficulties.[16] The need for exchange transfusions in a significant number of patients illustrates the persistent burden of preventable severe hyperbilirubinemia and emphasizes the significance of following evidence-based treatment guidelines.

In terms of maternal risk factors, gestational diabetes mellitus (GDM) was strongly related with pathological jaundice. Infants of diabetic mothers are more likely to have polycythemia, preterm, and metabolic abnormalities, all of which can lead to hyperbilirubinemia.[17] Although blood group O, Rh negative, and past newborn jaundice exhibited only slight trends toward significance, these characteristics are biologically plausible and have been identified in earlier studies as contributing to immune-mediated hemolysis and recurrent neonatal jaundice.[14,18]

This study has many limitations. Since this is a single-center hospital-based study, the results may not be applicable to the larger newborn community. The cross-sectional method limits the capacity to determine causal correlations between identified risk variables and pathological jaundice. Furthermore, numerous potential confounding variables, such as precise feeding habits, timing of bilirubin testing, and long-term neurodevelopmental follow-up, were not thoroughly examined, which may have influenced the observed associations.

5- CONCLUSION

In conclusion, pathological newborn jaundice accounted for a significant proportion of cases and was significantly linked to preterm, hemolytic causes (especially ABO incompatibility and G6PD deficiency), severe hyperbilirubinemia, and maternal gestational diabetes. Severe cases were associated with sequelae such as exchange transfusions and kernicterus, emphasizing the persistent clinical burden of preventable bilirubin neurotoxicity. Early risk stratification, routine screening for hemolytic disorders (particularly in high-prevalence areas), close monitoring of preterm and high-risk neonates, and adherence to updated evidence-based management guidelines are strongly advised to reduce morbidity and prevent long-term neurological sequelae.

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Conflict of intertest

About this study, the authors disclose no conflicts of interest.

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