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Original Research Article

CLINICAL, ETIOLOGICAL AND LABORATORY EVALUATION OF JAUNDICE IN INFANTS AT A TERTIARY CARE HOSPITAL

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Abstract

Background: The aim is to determine the clinical and etiological characteristics of infants presenting with jaundice at a tertiary care hospital. **Materials and Methods:** This prospective, observational study enrolled 200 infants aged 0 to 12 months with clinically evident jaundice confirmed by serum bilirubin levels. Exclusion criteria included congenital anomalies, major surgical interventions, and prior jaundice treatment. Demographic and clinical data were collected, including birth history, feeding practices, and potential risk factors like Rh incompatibility. Comprehensive physical examinations were performed, and the severity of jaundice was graded using the Kramer index. Laboratory tests included serum bilirubin, complete blood count, Coombs test, liver function tests, CRP, and G6PD assay. Urinalysis and thyroid function tests were also conducted as needed. Etiologies were categorized into physiological jaundice, breastfeeding-related jaundice, hemolytic jaundice, infective jaundice, metabolic/endocrine causes, hepatic causes, and undetermined etiologies. Statistical analysis was performed using SPSS version 25.0, with significance set at $p < 0.05$. **Result:** The mean age was 2.5 ± 1.3 months, with 56.00% males and 44.00% females. Most infants were full-term (75.00%) and delivered vaginally (60.00%). Exclusive breastfeeding was common (70.00%). Key risk factors included maternal Rh incompatibility (22.50%) and poor feeding (40.00%). Scleral icterus and yellow skin discoloration were prevalent, with varying severity per the Kramer index. Elevated bilirubin (>15 mg/dL) was observed in 40.00%, and hemolytic jaundice due to Rh incompatibility or G6PD deficiency was identified in 15.00%. Infective and hepatic causes accounted for 12.50% and 5.00%, respectively. Most infants (75.00%) improved with standard treatment, while a small subset required exchange transfusions or readmissions, with a mortality rate of 2.50%. **Conclusion:** This study reveals the multifaceted etiologies of infantile jaundice, emphasizing the need for thorough clinical and laboratory evaluation. Early identification and targeted treatment of pathological causes are essential to improve outcomes and reduce complications in this vulnerable population.

INTRODUCTION

Neonatal jaundice is a common clinical condition that presents in a significant number of infants, often causing concern among parents and healthcare providers. Characterized by a yellowish discoloration of the skin and sclera, jaundice in infants typically results from the accumulation of bilirubin, a byproduct of the breakdown of red blood cells. While jaundice is often considered a benign and self-limiting condition, it is crucial to identify the underlying etiology and assess the severity to ensure appropriate management and avoid complications. The clinical and etiological characteristics of infants presenting with jaundice are influenced by a variety of factors, including genetic, perinatal, and environmental contributors.[1] Jaundice is usually classified into two main categories: physiological and pathological. Physiological jaundice is a normal process that occurs in most newborns due to the immaturity of the liver's enzyme systems, which are responsible for processing bilirubin. This form of jaundice typically appears on the second or third day of life and resolves without intervention within the

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first week in full-term infants. On the other hand, pathological jaundice occurs when there is an underlying cause that accelerates bilirubin production, impairs bilirubin metabolism, or decreases bilirubin excretion. Pathological jaundice often requires medical attention and intervention to prevent severe complications, such as kernicterus, a form of brain damage caused by excessively high bilirubin levels. Understanding the various risk factors associated with neonatal jaundice is essential for early identification and treatment. Several perinatal and postnatal factors contribute to the development of jaundice. Maternal conditions during pregnancy, such as gestational diabetes, infections, or Rh incompatibility, can increase the risk of jaundice in newborns. Rh incompatibility, in particular, occurs when the mother's immune system produces antibodies that attack the red blood cells of an Rh-positive infant, leading to hemolysis and increased bilirubin production. Additionally, delayed cord clamping and the presence of bruising or cephalohematoma during delivery can elevate bilirubin levels in infants.[2] The method of feeding also plays a crucial role in the development of jaundice. Breastfeeding-associated jaundice is a well-documented phenomenon, with two main types: breastfeeding jaundice and breast milk jaundice. Breastfeeding jaundice typically occurs in the first week of life and is often related to inadequate intake or dehydration. On the other hand, breast milk jaundice develops later and is thought to be due to certain substances in the mother's milk that inhibit bilirubin metabolism. While breastfeeding-associated jaundice is generally benign, it is essential to monitor these infants closely to ensure that bilirubin levels do not become dangerously high. The clinical assessment of infants with jaundice involves a thorough history-taking and physical examination. Important historical factors to consider include the infant's age of onset, feeding patterns, family history of jaundice or liver disease, and any signs of illness such as lethargy or poor feeding. Physical examination findings, such as the extent of skin discoloration, the presence of scleral icterus, and any associated abnormalities like hepatomegaly or splenomegaly, can provide clues to the underlying etiology. The severity of jaundice is often graded using clinical tools such as the Kramer index, which correlates the extent of skin discoloration with serum bilirubin levels.[3] Laboratory investigations are a key component in the evaluation of jaundice in infants. Measuring serum bilirubin levels, including total and direct bilirubin, is critical in determining the severity and type of hyperbilirubinemia. A complete blood count (CBC) can provide insights into potential hemolysis or anemia, while a direct Coombs test helps identify immune-mediated hemolysis. Blood typing is essential in cases where blood group incompatibility is suspected. Additionally, liver function tests (LFTs) can indicate hepatic dysfunction, while tests for metabolic and endocrine disorders, such as thyroid function tests, may reveal underlying conditions like congenital hypothyroidism. In cases where an infectious etiology is suspected, blood and urine cultures are performed to rule out sepsis or urinary tract infections. The etiological classification of neonatal jaundice encompasses a wide range of conditions. Physiological jaundice remains the most common diagnosis, especially in full-term infants. Hemolytic jaundice, resulting from conditions such as Rh incompatibility or glucose-6-phosphate dehydrogenase (G6PD) deficiency, is another important category. G6PD deficiency is a genetic disorder that affects red blood cell stability, leading to hemolysis when the infant is exposed to certain triggers. Infective jaundice, caused by bacterial or viral infections, often presents with systemic signs of illness and requires prompt treatment. Hepatic causes, such as neonatal hepatitis or biliary atresia, are less common but can lead to prolonged and severe jaundice, necessitating specialized care and potential surgical intervention. Metabolic and endocrine disorders, though rare, should be considered in cases of unexplained or persistent jaundice.[4] The outcomes of infants with jaundice vary depending on the underlying cause and the timeliness of intervention. While most infants with physiological jaundice recover without any lasting effects, those with pathological jaundice may experience serious complications if not properly managed. Exchange transfusion, phototherapy, and hydration are some of the mainstay treatments for severe cases. The goal of management is to reduce bilirubin levels and prevent the neurotoxic effects associated with hyperbilirubinemia. Early identification and intervention are key to improving outcomes and reducing the risk of long-term sequelae.[5] Neonatal jaundice is a multifactorial condition with diverse clinical and etiological characteristics. A comprehensive approach that includes a detailed history, thorough physical examination, and appropriate laboratory investigations is essential for accurate diagnosis and effective management. Understanding the underlying causes and risk factors can help healthcare providers tailor treatment strategies and improve outcomes for affected infants. As research continues to evolve, further insights into the pathophysiology and management of neonatal jaundice will aid in optimizing care for this vulnerable population.

MATERIALS AND METHODS

This prospective, observational study was conducted to determine the clinical and etiological characteristics of infants presenting with jaundice at a tertiary care hospital. A total of 200 infants aged 0 to 12 months who presented with clinical signs of jaundice were enrolled over a period of one year. The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from the parents or legal guardians of all participating infants. The inclusion criteria included

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infants aged 0 to 12 months with clinically evident jaundice confirmed by serum bilirubin levels. Infants with congenital anomalies, a history of major surgical interventions, or those already receiving treatment for jaundice before presenting at the hospital were excluded. Additionally, infants whose parents or guardians did not consent to participate in the study were excluded. Detailed demographic and clinical data were collected from all enrolled infants. Information recorded included age, gender, birth history (such as gestational age, birth weight, and mode of delivery), feeding history (breastfeeding or formula feeding), and family history of jaundice or liver diseases. A thorough clinical history was taken to identify potential risk factors, including maternal health during pregnancy, presence of Rh incompatibility, or history of delayed cord clamping. Clinical assessments included a detailed physical examination, focusing on signs of jaundice (such as scleral icterus and yellow discoloration of the skin) and associated findings, including hepatosplenomegaly, lethargy, poor feeding, and fever. The severity of jaundice was graded based on the Kramer index, and a structured form was used to document all findings systematically.

Laboratory Investigations and Etiological Classification

All enrolled infants underwent comprehensive laboratory investigations to identify the underlying cause of jaundice and assess the severity of the condition. Serum bilirubin levels, including total and direct bilirubin, were measured to determine the extent of hyperbilirubinemia. A complete blood count (CBC) was performed to evaluate for anemia, infection, or other hematological abnormalities. Blood typing and a direct Coombs test were carried out to detect blood group incompatibility and to assess for hemolysis, which could indicate an immune-mediated cause of jaundice. Liver function tests (LFTs) were conducted to evaluate hepatic function, and additional inflammatory markers, such as C-reactive protein (CRP), were measured. Blood cultures were obtained to rule out sepsis in infants suspected of having an infectious cause.

In cases where urinary tract infection (UTI) was a potential contributing factor, urinalysis and urine culture were performed to confirm or exclude this diagnosis. Thyroid function tests were conducted to identify congenital hypothyroidism, a metabolic condition that can manifest with jaundice. Additionally, the glucose-6-phosphate dehydrogenase (G6PD) assay was used to detect G6PD deficiency, a common cause of hemolytic jaundice in certain populations. These investigations provided a comprehensive understanding of the infant's condition and facilitated the identification of specific etiological factors.

Based on the results of the clinical assessment and laboratory investigations, the causes of jaundice were categorized into distinct etiological groups. Physiological jaundice, which is a common and benign condition in neonates, was identified in infants who presented with typical features and normal bilirubin metabolism. Breastfeeding and breast milk jaundice were diagnosed in cases where prolonged jaundice was associated with exclusive breastfeeding. Hemolytic jaundice, often resulting from blood group incompatibility or G6PD deficiency, was characterized by evidence of hemolysis and confirmed through Coombs testing and enzyme assays. Infective jaundice, including cases due to sepsis or urinary tract infections, was identified through positive blood or urine cultures and elevated inflammatory markers. Metabolic and endocrine causes, such as hypothyroidism, were diagnosed based on abnormal thyroid function tests. Hepatic causes, including neonatal hepatitis and biliary atresia, were identified in infants with abnormal liver function tests and persistent direct hyperbilirubinemia. Cases that did not fit into these defined categories or where the cause remained unclear were classified under "Others/Undetermined Etiology." This structured approach to laboratory investigation and etiological classification ensured accurate diagnosis and appropriate management of infants presenting with jaundice.

Statistical Analysis: Data were analyzed using SPSS software version 25.0. Continuous variables, such as age and serum bilirubin levels, were expressed as mean ± standard deviation (SD), while categorical variables, such as gender and etiological classification, were presented as frequencies and percentages. Chi-square tests were used to analyze the association between categorical variables, and a p-value of <0.05 was considered statistically significant. Logistic regression analysis was performed to identify significant risk factors associated with severe jaundice.

RESULTS

The study population consisted of 200 infants with jaundice, and the demographic characteristics are summarized in Table 1. The mean age of the infants was 2.5 ± 1.3 months. Gender distribution was fairly balanced, with 56.00% males (112 infants) and 44.00% females (88 infants), with a p-value of 0.68, indicating no significant difference. The majority of infants were born full-term (75.00%, or 150 infants), while 25.00% (50 infants) were preterm ($p = 0.54$). Regarding the mode of delivery, 60.00% (120 infants) were delivered vaginally, and 40.00% (80 infants) were delivered via cesarean section, with a p-value of 0.72. Feeding history showed that 70.00% (140 infants) were exclusively breastfed, and 30.00% (60 infants) were formula-fed, with no significant difference observed ($p = 0.61$). These demographic findings suggest a diverse sample with a balanced representation of gender, gestational age, and delivery methods.

[Table 2] outlines the clinical history and risk factors associated with jaundice in the study population. Maternal Rh incompatibility was present in 22.50%

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(45 infants), with a p-value of 0.45. A history of delayed cord clamping was reported in 25.00% (50 infants), with a p-value of 0.53. A family history of jaundice was noted in 17.50% (35 infants, p = 0.40). Poor feeding was a common risk factor, observed in 40.00% (80 infants), while lethargy was present in 32.50% (65 infants). The p-values for these clinical risk factors ranged from 0.31 to 0.53, indicating no statistically significant associations. These data highlight common clinical concerns associated with jaundice in infants, emphasizing the need to consider multiple risk factors in diagnosis and management.

[Table 3] details the clinical presentation and severity of jaundice among the infants. Scleral icterus was present in 90.00% (180 infants), with a p-value of 0.25, while yellow skin discoloration was observed in all infants (100.00%). Hepatomegaly was noted in 30.00% (60 infants, p = 0.48), and splenomegaly was present in 10.00% (20 infants, p = 0.52). Jaundice severity, assessed using the Kramer index, showed that 15.00% (30 infants) had Grade 1 jaundice, 25.00% (50 infants) had Grade 2, 35.00% (70 infants) had Grade 3, and 25.00% (50 infants) had Grade 4, with a p-value of 0.62. The high prevalence of scleral icterus and yellow skin discoloration indicates the widespread nature of jaundice, while the varying Kramer grades reflect the spectrum of severity.

[Table 4] presents the laboratory findings. Elevated total bilirubin levels (>15 mg/dL) were found in 40.00% (80 infants), with a p-value of 0.39. Direct Coombs test results were positive in 12.50% (25 infants, p = 0.42), indicating cases of hemolytic jaundice. Elevated liver enzymes were detected in 15.00% (30 infants, p = 0.51), and positive blood cultures were obtained in 10.00% (20 infants, p = 0.35), suggesting an infective etiology. Low hemoglobin levels (<10 g/dL) were observed in 20.00% (40 infants, p = 0.49), and G6PD deficiency was identified in 9.00% (18 infants, p = 0.60). These findings highlight the importance of a comprehensive workup to identify the underlying causes of jaundice, as multiple etiologies may coexist.

[Table 5] categorizes the etiological factors associated with jaundice. Physiological jaundice was the most common, affecting 35.00% (70 infants, p = 0.50). Breastfeeding-related jaundice, including breast milk jaundice, was observed in 25.00% (50 infants, p = 0.38). Hemolytic jaundice, caused by Rh incompatibility or G6PD deficiency, accounted for 15.00% (30 infants), with each condition contributing equally (7.50%, or 15 infants each). Infective jaundice was diagnosed in 12.50% (25 infants, p = 0.41), while metabolic or endocrine causes were identified in 5.00% (10 infants, p = 0.55). Hepatic causes, such as neonatal hepatitis or biliary atresia, were also found in 5.00% (10 infants, p = 0.62). Cases classified as “Others/Undetermined” made up 2.50% (5 infants, p = 0.73). This classification underscores the diverse etiological landscape of infantile jaundice, necessitating individualized treatment approaches.

Table 1: Demographic Characteristics of the Study Population.

Characteristic	Frequency (n=200)	Percentage (%)	p-value
Age (in months, mean ± SD)	2.5 ± 1.3	-	-
Gender			0.68
Male	112	56.00	
Female	88	44.00	
Gestational Age			0.54
Full-term	150	75.00	
Preterm	50	25.00	
Mode of Delivery			0.72
Vaginal	120	60.00	
Cesarean Section	80	40.00	
Feeding History			0.61
Exclusive Breastfeeding	140	70.00	
Formula Feeding	60	30.00	

Table 2: Clinical History and Risk Factors

Risk Factor	Frequency (n=200)	Percentage (%)	p-value
Maternal Rh Incompatibility	45	22.50	0.45
History of Delayed Cord Clamping	50	25.00	0.53
Family History of Jaundice	35	17.50	0.40
Poor Feeding	80	40.00	0.36
Lethargy	65	32.50	0.31

Table 3: Clinical Presentation and Severity of Jaundice

Clinical Sign	Frequency (n=200)	Percentage (%)	p-value
Scleral Icterus	180	90.00	0.25
Yellow Skin Discoloration	200	100.00	-
Hepatomegaly	60	30.00	0.48
Splenomegaly	20	10.00	0.52
Kramer Index Grade			0.62
Grade 1	30	15.00	
Grade 2	50	25.00	
Grade 3	70	35.00	

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Grade 4	50	25.00	

Table 4: Laboratory Findings

Investigation	Abnormal Cases (n=200)	Percentage (%)	p-value
Total Bilirubin > 15 mg/dL	80	40.00	0.39
Direct Coombs Positive	25	12.50	0.42
Elevated Liver Enzymes	30	15.00	0.51
Positive Blood Culture	20	10.00	0.35
Low Hemoglobin (<10 g/dL)	40	20.00	0.49
Positive G6PD Deficiency	18	9.00	0.60

Table 5: Etiological Classification of Jaundice

Etiology	Frequency (n=200)	Percentage (%)	p-value
Physiological Jaundice	70	35.00	0.50
Breastfeeding/Breast Milk Jaundice	50	25.00	0.38
Hemolytic Jaundice	30	15.00	0.45
- Rh Incompatibility	15	7.50	
- G6PD Deficiency	15	7.50	
Infective Jaundice	25	12.50	0.41
Metabolic/Endocrine Causes	10	5.00	0.55
Hepatic Causes	10	5.00	0.62
Others/Undetermined	5	2.50	0.73

Table 6: Outcomes and Follow-Up

Outcome	Frequency (n=200)	Percentage (%)	p-value
Improved with Treatment	150	75.00	0.30
Required Exchange Transfusion	10	5.00	0.47
Persistent Jaundice >1 Month	15	7.50	0.53
Readmission Required	20	10.00	0.40
Mortality	5	2.50	0.69

[Table 6] summarizes outcomes and follow-up data. A significant majority of infants (75.00%, or 150 infants) improved with standard treatment, with a p-value of 0.30. Exchange transfusions were required in 5.00% (10 infants, p = 0.47), and 7.50% (15 infants) experienced persistent jaundice lasting more than one month (p = 0.53). Readmission was necessary for 10.00% (20 infants, p = 0.40), and the mortality rate was 2.50% (5 infants, p = 0.69). These results indicate that while most infants responded well to treatment, a small subset required more intensive management, and some cases had severe outcomes, emphasizing the need for vigilant monitoring and timely intervention.

DISCUSSION

The demographic characteristics of the study population showed a mean age of 2.5 ± 1.3 months, with a nearly equal gender distribution (56.00% males and 44.00% females, p = 0.68). These results align with the findings of Bahl et al. (2019), who reported a similar age range and balanced gender distribution among infants with jaundice.[6] The predominance of full-term births (75.00%, p = 0.54) and a higher prevalence of vaginal deliveries (60.00%, p = 0.72) are consistent with research by Kumar et al. (2021), who noted that jaundice is more commonly observed in full-term infants, though the mode of delivery was not significantly associated with the incidence of jaundice.[7] Additionally, the feeding history revealed that 70.00% of the infants were exclusively breastfed, mirroring findings by Mehta et al. (2020), which highlighted the association between exclusive breastfeeding and prolonged physiological jaundice.[8] Maternal Rh incompatibility was present in 22.50% of cases, a finding comparable to the study by Desai et al. (2018), which identified Rh incompatibility as a significant contributor to hemolytic jaundice.[9] However, the absence of statistically significant associations with other risk factors, such as delayed cord clamping and family history of jaundice, suggests that multiple interrelated factors may contribute to the development of jaundice. Similar conclusions were drawn by Shah et al. (2022), who emphasized the complex interplay of genetic and perinatal factors in neonatal jaundice.[10] The clinical presentation and severity of jaundice, revealed that scleral icterus and yellow skin discoloration were the most common signs, affecting 90.00% and 100.00% of infants, respectively. These findings are consistent with Patel et al. (2019), who also reported scleral icterus as a predominant feature of neonatal jaundice.[11] The varying severity, assessed using the Kramer index, reflects the broad spectrum of the condition and is in line with Singh et al. (2018), who highlighted the need for careful clinical grading to guide management.[12] Laboratory findings indicated that 40.00% of infants had elevated total bilirubin levels (>15 mg/dL), a result similar to the study by Verma et al. (2020), which reported high bilirubin levels in 38.00% of infants with severe jaundice.[13] The presence of positive Coombs tests in 12.50% of cases aligns with Agarwal et al. (2017), who found immune-mediated hemolysis in a comparable proportion of infants.[14] The detection of G6PD deficiency in 9.00% of infants corroborates findings

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by Chatterjee et al. (2018), emphasizing the importance of screening for this genetic disorder in populations with a high prevalence.[15] The etiological factors, with physiological jaundice being the most common (35.00%), followed by breastfeeding-related jaundice (25.00%). These rates are in agreement with research by Roy et al. (2019), which documented a similar prevalence of physiological and breastfeeding-associated jaundice.[16] The occurrence of hemolytic jaundice (15.00%) due to Rh incompatibility and G6PD deficiency echoes findings from Sharma et al. (2021), who highlighted these conditions as major causes of neonatal hyperbilirubinemia.[17] Infective jaundice, identified in 12.50% of cases, and hepatic causes (5.00%) were comparable to observations made by Malik et al. (2018), who emphasized the role of infections and hepatic dysfunction in prolonged jaundice cases.[18] The outcomes and follow-up data. The high improvement rate of 75.00% with standard treatment is consistent with Gupta et al. (2019), who reported successful outcomes in a majority of jaundiced infants with appropriate management.[19] The requirement for exchange transfusion in 5.00% of cases is in line with findings by Bhattacharya et al. (2017), who noted a similar proportion of infants needing intensive intervention.[20] The low readmission rate (10.00%) and mortality rate (2.50%) reflect effective management protocols and are consistent with data from Das et al. (2021), who emphasized the importance of timely diagnosis and treatment to reduce complications and improve outcomes.[21]

CONCLUSION

In conclusion, this study highlights the diverse clinical and etiological characteristics of infants presenting with jaundice at a tertiary care hospital. Physiological jaundice was the most prevalent, but a significant proportion of cases had underlying pathological causes, such as hemolytic conditions, infections, or metabolic disorders. Early and accurate identification of these etiologies is crucial to prevent complications and ensure timely management. The findings underscore the importance of comprehensive clinical assessment and targeted investigations to guide appropriate treatment strategies and improve outcomes for affected infants.

REFERENCES

1. Sharma P, Gupta S, Bhattacharjee S. Analyzing the effectiveness of phototherapy in reducing serum bilirubin levels in neonatal jaundice: A randomized study. Indian J Neonatal Med. 2020;12(3):144-150.
2. Mishra A, Jain S, Singh K. Risk factors for severe hyperbilirubinemia in neonates: A cohort study. J Perinatal Neonatal Care. 2019;8(2):89-95.
3. Kumar R, Sinha T, Bose A. Role of early screening in the prevention of neonatal jaundice complications: A population-based analysis. Neonatal Health J. 2021;15(4):298-304.
4. Roy T, Sengupta P, Basu R. Evaluating the association between sepsis and neonatal jaundice in a clinical setting. J Clin Neonatal Res. 2018;13(5):329-335.
5. Joshi M, Agarwal R, Rao P. Impact of delayed cord clamping on neonatal bilirubin levels: A clinical trial. Perinatal Med J. 2017;19(2):45-52.
6. Bahl R, Prasad K, Sharma A. Age distribution and gender characteristics of infants with neonatal jaundice: A hospital-based study. J Neonatal Med. 2019;6(4):210-215.
7. Kumar V, Joshi P, Verma S. Prevalence of jaundice in full-term vs. preterm infants and associated risk factors. Indian Pediatr. 2021;58(1):34-40.
8. Mehta S, Arora D, Patel N. The impact of exclusive breastfeeding on the duration of physiological jaundice in neonates. Breastfeed Rev. 2020;28(3):14-19.
9. Desai R, Gupta M, Shah V. Rh incompatibility and its contribution to neonatal hemolytic jaundice: A retrospective analysis. J Perinatol. 2018;38(7):901-907.
10. Shah P, Roy S, Sen A. Genetic and perinatal factors influencing neonatal jaundice: A comprehensive review. J Clin Neonatol. 2022;11(1):45-51.
11. Patel K, Singh R, Narang P. Clinical features and management of neonatal jaundice: A focus on scleral icterus. Pediatr Health. 2019;34(5):198-203.
12. Singh T, Malik R, Choudhury A. Kramer index as a reliable tool for grading neonatal jaundice: Correlation with serum bilirubin levels. Neonatal Care J. 2018;7(2):125-130.
13. Verma A, Saini P, Bhardwaj H. Hyperbilirubinemia in neonates: Analysis of risk factors and outcomes. J Pediatr Hematol Oncol. 2020;42(9)
14. Agarwal N, Yadav V, Mishra R. Immune-mediated hemolytic jaundice in neonates: The role of Coombs testing. Hematol Rep. 2017;9(3):7078.
15. Chatterjee S, Banerjee D, Roy K. G6PD deficiency and neonatal jaundice: Prevalence and screening recommendations. J Med Genet. 2018;55(11):780-785.
16. Roy B, Dutta P, Choudhary S. Etiological factors and management strategies for neonatal jaundice: A multi-center study. Neonatol Res. 2019;4(2):89-96.
17. Sharma R, Kapoor A, Jain M. Major causes of neonatal hyperbilirubinemia: A focus on hemolytic conditions. Neonatal Med J. 2021;56(1):45-50.
18. Malik S, Verma N, Joshi H. Infective and hepatic causes of prolonged neonatal jaundice: A tertiary care perspective. J Hepatol Neonatol. 2018;10(4):302-307.
19. Gupta A, Sharma K, Patel R. Outcomes of jaundiced infants treated at a tertiary care center: Success rates and management. Pediatr Ther. 2019;10(5):515-520.
20. Bhattacharya S, Ghosh T, Roy N. Exchange transfusion in severe neonatal jaundice: Indications and outcomes. J Neonatal Care. 2017;5(1):23-29.
21. Das R, Bhatia N, Mehta P. Timely intervention in neonatal jaundice: Reducing complications and improving outcomes. J Neonatol. 2021;38(8):870-878.

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