Original Research Article

Clinical profile of neonates with jaundice

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Abstract

Background: Most of the neonates frequently have jaundice which is very often physiologic jaundice due to the immaturity of the liver and is universal and resolves within first ten days of life. The other is the more ominous pathological jaundice. **Aim of the study:** To study the clinical profile and etiology of neonatal jaundice **Materials and Methods:** This was a prospective study. A total of 73 cases of neonates with jaundice were admitted in the study period. The patient demographics such as age and gender, detailed clinical history, relevant laboratory investigations, and the etiology of jaundice were noted. **Results:** There were 39 (53.4%) male and 34 (46.5%) female newborns and the male to female ratio was 1.1:1. There were 79.4% term deliveries and 20.5% preterm deliveries. There were 68.4% cases with normal birth weight and 31.5% were low birth weight newborns. Physiological jaundice was seen in 76.7% (56) cases and pathological jaundice was seen in 23.2% (17) cases. ABO and Rh incompatibility, and neonatal sepsis were common causes for pathologic jaundice. **Conclusion:** Neonatal jaundice is a common clinical problem that requires to be categorized as physiological or pathological type. It has to be addressed at the earliest so as to prevent irreversible kernicterus. ABO and Rh incompatibility, neonatal sepsis, low birth weight and prematurity are associated with neonatal jaundice. Education of expectant mothers and community in general will help to reduce the severity and burden of neonatal jaundice.

Key Words: Neonatal jaundice, neonatal sepsis, ABO incompatibility, Breast milk jaundice

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INTRODUCTION

Most of the neonates frequently have jaundice which is very often physiologic jaundice due to the immaturity of the liver and this type of jaundice usually resolves within first ten days of life. This type of physiologic neonatal jaundice is universal. Some neonates can even have pathological jaundice due to certain underlying causes where the serum bilirubin levels can rise dangerously high and cause irreversible brain damage. It is extremely important to differentiate between these two types of jaundice so as to provide appropriate investigations and care to the newborns. The treatment depends upon the cause of the jaundice and various laboratory tests have to be employed to ascertain the exact nature of such jaundice. [1] Neonatal hyperbilirubinemia is defined as a total serum bilirubin level above 5 mg per dL (86 µmol per L). Almost 60% of term newborn babies have clinical jaundice in the first week of life but very few have significant underlying disease. [2] The most common cause of neonatal hyperbilirubinemia in India is physiological jaundice. Various other conditions in decreasing order are preterm infant, blood group incompatibility, Neonatal septicemia, G-6PD deficiency, cephalhematoma, drug induced, RBC membrane disorders and many others. In the present study, we have attempted to look at the clinical profile of neonates with jaundice in our local population.

Aim of the study

To study the clinical profile and etiology of neonatal jaundice

MATERIALS AND METHODS

This was a prospective study carried out in the Department of Paediatrics, Fathima institute of Medical Sciences, over a period of one year from January 2018 to December 2018. A total of 73 cases of neonates with jaundice were admitted in the study period. The patient demographics such as age and gender, detailed clinical history, relevant laboratory investigations, and the etiology of jaundice were noted.

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Complete blood picture, peripheral smear examination, blood group of mother and new born, reticulocyte count, serum bilirubin levels, total, conjugated and unconjugated levels were done in all cases. Direct Coomb's test and Indirect Coomb's test, C-Reactive protein, test for G6PD deficiency, hemoglobin electrophoresis, sickling test, osmotic fragility test, blood culture, chest X-ray were done where required.

Inclusion criteria

All neonates admitted for jaundice and having serum bilirubin above 5 mg/dl

Exclusion criteria

Newborns who had received blood transfusion.

OBSERVATIONS AND RESULTS

Table 1: Age wise distribution of the cases

Age (in days)	No. of cases	Percent (%)
1	4	5.4%
2	17	23.2%
3	26	35.6%
4	21	28.7%
5	2	2.7%
6	2	2.7%
7	1	1.3%
Total	**73**	**100%**

Gender-wise distribution of the cases: There were 39 (53.4%) male and 34 (46.5%) female newborns.

Term versus preterm births: There were 58 (79.4%) term deliveries and 15 (20.5%) preterm deliveries.

Birth weight of the newborns: There were 50 (68.4%) cases with normal birth weight and 23 (31.5%) were low birth weight newborns.

Table 2: Etiology wise distribution of the cases

Cause of jaundice	No. of cases	Percent (%)
Physiological Jaundice	56	76.7%
Suspected ABO Incompatibility	6	8.2%
Septicemia	3	4.1%
Rh Incompatibility	4	5.4%
Cephalhematoma	1	1.3%
Breast milk jaundice	2	2.7%
G-6PD deficiency	1	1.3%
Total	**73**	**100%**

Physiological jaundice was seen in 76.7% (56) cases and pathological jaundice was seen in 23.2% (17) cases.

Table 3: Mean Serum bilirubin value in physiological and pathological neonatal Jaundice

Jaundice	Mean Serum bilirubin value (mg/dl)	t value	p value
Physiological	12.3 +/- 2.5	11.89	<0.0001
Pathological	19.4 +/- 3.6		

DISCUSSION

Neonatal physiologic jaundice is a result of increased bilirubin production secondary to accelerated destruction of erythrocytes, and decreased excretory capacity of liver secondary to low levels of ligandin in hepatocytes, and low activity of the bilirubin-conjugating enzyme uridine diphosphoglucuronyltransferase (UDPGT). Whenever aadditional factors superimpose upon the above mechanisms, then it is more likely to become pathological jaundice. Common etiologies for increased serum bilirubin are immune or nonimmune hemolytic anemias, polycythemia, bruising, cephalhematoma, preterm delivery, blood group incompatibility, neonatal septicemia, G-6PD deficiency, drug induced hemolysis, RBC membrane disorders and many others. [1] Neonatal jaundice starts becoming apparent at serum bilirubin levelof > 2 mg/dl. The yellowish discoloration of skin progresses cephalocaudally. For the feet to be affected level generally must be over 255 µmol/l or 15 mg/dL. Neonatal physiological jaundice occurs in over 50% of term [3, 4] and 80% of preterm neonates. [5] It manifests as yellowish discoloration of the skin and sclera due to serum bilirubin levels. Jaundice is a result of the increased breakdown of red blood cells and/or decreased hepatic excretion of bilirubin. For most of the newborns hyperbilirubinemia is a natural transition that resolves within the first week of life with maturing of the liver; however, hyperbilirubinemia is also the main reason for hospital readmission during the neonatal period. [6] Hyperbilirubinemia is a primary concern associated with jaundice due to the connection between increased levels of unconjugated bilirubin and neurotoxic effects causing long-term sequelae including cerebral palsy, hearing loss, acute bilirubin encephalopathy and kernicterus (chronic bilirubin encephalopathy. [5, 7, 8] Kernicterus often has its attendant medical, economic, and social burden on the patients, families, and societies. Several reports have indicated the important contribution of severe neonatal jaundice and hyperbilirubinemia to neonatal morbidity and mortality. [9, 10, 11] In a multi-center study in six developing countries, hyperbilirubinemia was a primary diagnosis for severe illness requiring hospital admission, the cause for 12–78% of the admissions in the first 6 days of life and for 2–57% of admissions during the next 7–59 days. [11] While the majority of infants have serum levels of 5–6 mg/dl. A higher level has been found in exclusively breast-fed infants. [12-14] Worldwide, it is estimated that 10.5% of live births require phototherapy for jaundice and Nepal estimates are in the range of 3–6%. Research from a hospital-based study in Dharan, Nepal found that 9.2% of infants admitted to the neonatal intensive care unit (NICU) had pathologic jaundice. [15] Estimates from recent studies show 6.7% in Lagos, Nigeria and 10.5% and 25.3% in term

and near-term (35–37 weeks) newborns, respectively, in Turkey. [4] Several maternal and neonatal risk factors such as preeclampsia, G6PD deficiency, ABO incompatibility, prematurity, birth weight, intrauterine growth retardation, metabolic abnormalities, neonate's gender, birth weight, and nutrition have been identified as risk factors for neonatal jaundice. [16] In our study, a slight male preponderance (53.4%) was seen for neonatal jaundice and the male to female ratio was 1.1:1. Similar male preponderance for neonatal jaundice was observed by Rasul CH *et al*[17], Mantani *et al*[18] and Sharma et al. [19] They reported the male to female ratio as 1.3:1, 1.6:1 and 1.3:1 respectively. Paridhi *et al*[20] (n=108) in a similar study observed male preponderance with 65 (60.19%) boys and 43 (39.81 %) girls. In our study, all the 15 (20.5%) preterm babies were observed to have pathologic jaundice. The premature babies are increased risk for pathologic jaundice as they may not be able to eliminate the bilirubin as quickly as full term babies do. Also, preterms have feeding difficulties and have fewer bowel movements. These conditions result in less bilirubin being eliminated from the preterm's body. Dehydration and low intake of calories from poor breast feeding may contribute to the onset of jaundice. [21] In the study by Paridhi *et al*[20] there were 32.40% preterms and 67.61% were full term newborns. Preterm newborns are more likely to develop jaundice due to immaturity of their bilirubin conjugating system, increased rate of haemolysis, increased enterohepatic circulation, and decreased caloric intake. [22] In our study, 76.7% of the study newborns displayed physiologic jaundice and 23.2% had pathologic jaundice. Paridhi *et al*[20] in their study observed physiological jaundice in 44.4% of cases and pathological jaundice in 55.6% cases. Various studies have given different percentages for neonatal pathological jaundice. In present day clinical practice due to better availability and use of anti-D immunoglobulin, incidence of jaundice resulting from Rh incompatibility has decreased significantly and is reported around 5%. [23] In our study there were 5.4% cases of Rh incompatibility leading to pathological jaundice. Another important cause is hereditary hemolytic anemia which can lead to indirect hyperbilirubinemia and encephalopathy. In our study physiologic jaundice was seen in 76.7% cases which is slightly higher than is reported in literature by various authors. Anand VR *et al*[24] and Bahl L [25] *et al*have reported incidence of physiologic jaundice to be 47.6% and 63.8% respectively. Paridhi *et al*[20] reported pathological jaundice in 60 of their newborns. For them, sepsis (12%), ABO incompatibility (11.1%), Rh incompatibility (4.6%) were three most common causes observed. Sepsis associated with neonatal jaundice was the presenting complaint in only 4 (30.7%) cases.They also observed large cephalhematoma leading to neonatal jaundice in 3 (2.8%) of their cases. In our study too ABO incompatibility (8.2%) Rh incompatibility (5.4%) and sepsis (4.1%) were seen as important causes for pathological jaundice. Cephalhematoma contributing to jaundice was observed in 1 (1.3%)of our cases. They also observed G6PD deficiency in 1 (0.9%) of case which was a male.We also encountered one case (1.3%) of G6PD deficiency. Adoba *et al*[26] in their study on neonatal jaundice in Ghana observed G6PD abnormality in 12% of the neonates with jaundice and ABO incompatibility in 18% of their cases. Narang *et al*[27] observed in their study (n=551) that ABO incompatibility and Rh-isoimmunization accounted for 6.1% and 2.9% of cases respectively. Low neonatal birth weight and prolonged duration of labour are also known to be risk factors for neonatal jaundice. [26] In our study, 31.5% of the newborns had low birth weight. The duration of labor was not recorded in our study. Neonatal sepsis is a blood infection that occurs in an infant younger than 90 days old. Early-onset sepsis is seen in the first week of life. Late onset sepsis occurs after 1 week through 3 months of age. Neonatal septicemia is an important cause for neonatal jaundice. In our study there were 4.1% cases of neonatal sepsis. In a study by Bharat Kumar *et al*[28] jaundice due to septicemia ranked second in their series of neonatal hyperbilirubinemia. Jaundice is an important manifestation of bacterial septicemia and UTI and should be seriously considered when it first appears after 3 days of birth and persists beyond two weeks of life. In their study, in 18 cases of infection, pneumonia was found to be underlying problem in 6 cases (33.3%), followed by others. The commonly isolated organisms in such cases are E.coli, Proteus mirabilis, Bacteroides species and Kleibsiella pneumoneae. The red flags for pathologic jaundice are: Jaundice in first 24 hours, Rapidly rising total bilirubin, concentration (>86umol/L/day), Younger gestational age, Previous sibling with jaundice, Significant bruising, Jaundice persisting for more than 2-3 weeks and East Asian ethnicity. The first measurement of serum bilirubin should occur between 24-72h of life, or earlier if visible jaundice is observed and can be repeated serially. Assessing jaundice by skin appearance is inaccurate, especially in darker skinned infants, and serum bilirubin should be measured mandatorily to assess for hyperbilirubinemia. Whereas breast feeding jaundice is a mechanical problem, breast milk jaundice is more of a biochemical problem. It is a diagnosis of exclusion and is applicable to newborns who are exclusively fed on breast milk and in whom there are no other attributable causes for the jaundice. It usually appears at the end of first week of life from day 6 to day fourteen and hence overlaps with the physiological jaundice and can last for up to two months. Several factors are thought to be responsible for this condition. [21, 29] It may develop in up to one third of healthy

breastfed infants. Pathophysiology of breast milk jaundice is not well understood, but it is thought that substances in breast milk, such as beta-glucuronidases and nonesterified fatty acids, may inhibit normal bilirubin metabolism. Sometimes the neonatal hyperbilirubinemia is idiopthic and the cause remains unknown even after extensive investigatons. Various reports from our country have revealed that idiopathic hyperbilirubinemia ranges between 8.8 to 57.6%. [27] The treatment of neonatal hyperbilirubinemia includes phototherapy and exchange transfusion and addressing the cause of hyperbilirubinemia.

CONCLUSION

Neonatal jaundice is a common clinical problem that requires to be categorized as physiological or pathological type. The latter requires thorough work up to determine the exact cause and to guide the treatment. It has to be addressed at the earliest so as to prevent irreversible kernicterus. ABO and Rh incompatibility, neonatal sepsis, low birth weight and prematurity are associated with neonatal jaundice. Education of expectant mothers and community in general will help to reduce the severity and burden of neonatal jaundice.

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