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Original Research Article

A Prospective Investigation of the Aetiology and Clinical Characteristics of Newborn Jaundice in Bihar, India

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Abstract

Aim: To evaluate the Aetiological factors and clinical profile of neonatal jaundice from Bihar, India. **Methods:** This prospective observational study was done the Department of Pediatrics, Anugrah Narayan Magadh Medical College and Hospital, Gaya, Bihar, India, from March 2020 to January 2021. Total of 100 cases were enrolled for the study. Jaundice was ascertained by clinical methods. This was confirmed with the help of biochemical tests. Serum bilirubin was estimated by Van den Bergh method. All babies with serum bilirubin value of >10mg/dl were included in the study. **Results:** Out of 100 jaundiced neonates, 91 were born at term (91%) and the remaining 9 were preterm babies (9%). Among 100 neonates studied, majority had birth weight between 2501g and 3000g (51%). Only 11 babies had birth weight <2.5kg (11%). Here maximum number of cases was due to physiological Jaundice 35 (35%). This was followed by ABO incompatibility which constituted 18%. Of these 50% were due to OA incompatibility and 50% due to OB incompatibility. Other common causes were sepsis (11%), Rh incompatibility (9%), idiopathic (9%) and prematurity (7%). Neonatal jaundice was attributed to cephalhematoma and breast feeding in 5 cases each (5%). There was one case of haemolytic anaemia diagnosed as hereditary spherocytosis. Of the 100 neonates 64 were males (64%) and 36 were females (36%). **Conclusion:** The physiological jaundice is the most common cause of neonatal jaundice in our hospital and followed by ABO incompatibility, sepsis, Rh incompatibility and idiopathic cases.

Keywords: Jaundice, Sepsis, Neonates

Introduction

Jaundice is yellow discoloration of the skin and sclera that occurs when levels of bilirubin are increased. Bilirubin is a product of heme catabolism, and 80% to 90% of hyperbilirubinemia occurs due to the breakdown of haemoglobin[1]. Neonatal hyperbilirubinemia occurs due to a variety of factors. It may be physiological or pathological. Neonatal hyperbilirubinemia is a common condition requiring inpatient treatment, as well as an important reason for readmission to hospital[2-4]. It occurs in 70–80% of the neonates, more commonly in preterms[5,6]. Although, only 5–10% of the newborns need to be treated due to pathological

hyperbilirubinemia, the threat of neurologic damage always remains, especially with very high bilirubin level, in presence of certain risk factors and in cases where management remains inappropriate[7,8]. Severe neonatal jaundice has the potential to cause bilirubin encephalopathy (kernicterus) which can evolve into chronic and permanent neurological sequelae. Thus, survivors may suffer from severe neurological handicaps like cerebral palsy, gaze palsies and deafness. This sequela is irreversible but can be prevented by early diagnosis and appropriate management of neonatal jaundice. For the management to be appropriate, identification of the etiological and risk factors is of paramount importance. Etiology of hyperbilirubinemia is not only crucial for optimal management of the patient but also it may have implications for subsequent pregnancies. However, the etiology of neonatal hyperbilirubinemia may remain obscured in more than half of the cases[9-11]. Hemolytic disease of the newborn (HDN) is one of the common pathologic cause of hyperbilirubinemia during the early neonatal period, mostly due to Rhesus (Rh) incompatibility, ABO incompatibility, G6PD deficiency, and rarely induced by other all immune anti-bodies[10,11]. The incidence, etiological and contributory factors of neonatal jaundice vary according to ethnic and geographic differences[12]. These factors in developing countries may be different from those of developed nations, probably as a result of racial, cultural and environmental differences. A recent meta-analysis of neonatal jaundice in low- and middle-income countries highlighted the need for more robust epidemiological studies to identify additional risk factors that may be particular to these settings[13].

Material and methods

This prospective observational study was done the Department of Pediatrics, Anugrah Narayan Magadh Medical College and Hospital, Gaya, Bihar, India, from March 2020 to January 2021. A total of 350 neonates were admitted in our NICU and post-natal ward during the specified period. Out of these 100 newborns were jaundiced (Serum bilirubin >10 mg/dl).14,15 So a total of 100 cases were enrolled for the study. Babies attending outpatient department only, were excluded from the study. Jaundice was ascertained by clinical methods. This was confirmed with the help of biochemical tests. Serum bilirubin was estimated by Van den Bergh method. All babies with serum bilirubin value of >10mg/dl were included in the study. Detailed history was taken. Thorough physical examination was done, and the relevant investigations were carried out. General data including age, birth weight, age at detection of jaundice, breast feeding status, family history of jaundice was documented. Further investigations were not carried out on those babies who were having physiological jaundice. Blood grouping and Rh typing of baby and mother were done. Cord blood bilirubin and haemoglobin, direct coomb's test (DCT) and bilirubin monitoring were done whenever there was a setting for Rh incompatibility. In case of ABO incompatibility, DCT was done, and bilirubin monitored. Other investigations like haemoglobin level, peripheral smear and reticulocyte count were done. If these tests showed features of haemolysis and there was no blood group incompatibility, G6PD assay, sickling test, haemoglobin electrophoresis and osmotic fragility test were done wherever appropriate. G6PD was done by fluorescent technique. 2% sodium metabisulphite was used for sickling test. Osmotic fragility test using serial dilutions of sodium chloride was done. Neonates who were suspected to have sepsis were investigated by complete blood count, septic screen and blood and urine cultures. Thyroid function tests were done as a part of screening in all neonates. In case of high index of suspicion appropriate tests were carried out to rule out neonatal metabolic disorders like galactosemia and congenital intrauterine infections.

Results

Out of 100 jaundiced neonates, 91 were born at term (91%) and the remaining 9 were preterm babies (9%) table 1. Among 100 neonates studied, majority had birth weight between 2501g and 3000g (51%). Only 11 babies had birth weight <2.5kg (11%) Table 2. Here maximum number of cases was due to physiological jaundice 35 (35%) Table 3. This was followed by ABO incompatibility which constituted 18%. Of these 50% were due to OA incompatibility and 50% due to OB incompatibility. Other common causes were sepsis (11%), Rh incompatibility (9%), idiopathic (9%) and prematurity (7%). Neonatal jaundice was attributed to cephalhematoma and breast feeding in 5 cases each (5%). There was one case of haemolytic anaemia diagnosed as hereditary spherocytosis. Of the 100 neonates 64 were males (64%) and 36 were females (36%).

Discussion

In this study of ours, majority of the babies with neonatal jaundice were of term gestation. Only 9% of babies studied were preterm. Studies by Bhutani et al and Singhal et al had found a higher percentage of premature babies in their studies[16,17]. Our institution mainly managed low to moderate risk pregnancies and hence majority of our babies were of term or near-term gestation. This could be the reason for the higher percentage of term babies in our study. Out of 100 neonates studied, 64% were males and only 36% were females. This matches earlier studies by Effiong et al, Narang et al and Korejo et al where majority of the babies were males[18-20]. Most of the babies studied had birth weight 2501-3000g (51%), 38% had birth weight >3kg. Only 11% babies had birth weight <2.5Kg. As our study had 91% term babies, majority had normal birth weight.

In this study, physiological jaundice was the diagnosis in the majority of the cases i.e. 35 out of 100 cases (35%). This is in concordance with previous studies. Bahl et al had reported that physiological jaundice contributed to the majority (63.8%) of cases studied[21]. Singhal et al (16.7%) and Merchant et al (25.3%) had also shown high incidence of physiological jaundice in their studies.[17,22] This was followed by ABO incompatibility as the next leading cause of neonatal jaundice (18%). This is very similar to the findings by Verma et al and Merchant et al that ABO incompatibility contributed to 22.6% of cases[22,23]. The number of OA and OB incompatibility cases was equal in this study. Bahl et al had reported a higher incidence of OA incompatibility (60%) whereas Bajpai PC et al had observed higher incidence of OB incompatibility[21,24].

Sepsis constituted 11% of the cases studied. This is in concordance with earlier studies which showed a similar trend. Sepsis was found to be the cause of jaundice in 8% neonates by Merchant et al, in 11.6% by Verma et al and in 9.6% by Narang et al.[19,22,23].

Rh incompatibility was responsible for 9% of cases in this study. Bajpai PC et al reported an incidence of 1.6% for Rh incompatibility while Verma et al found that to contribute to 9.8% of the cases[23,24]. Our finding is comparable with the study by Singhal et al where Rh incompatibility was present in 8.1% of neonates[17]. Thus, ABO incompatibility was more prevalent than Rh incompatibility. This is in agreement with older studies done abroad[25,26]. Similar findings were reported from India too[17,22,23]. No known cause could be established in 9 cases (9%). Previous Indian studies have reported incidence of idiopathic jaundice to be ranging from 8.8% to 57%[17,21]. Cephalhematoma contributing to jaundice was found in 6% of our cases. This is comparable to the study by Narang et al which found an incidence of 6.3%[19]. There were 4 cases of breast-feeding jaundice (4%) which regressed after improving the frequency and method of breast feeding. There was 1 case of haemolytic anaemia (1%) which was later diagnosed as hereditary spherocytosis. This

baby's mother was suffering from hereditary spherocytosis and had undergone splenectomy. We did not get any case of G6PD deficiency in our study. This could be a reflection of the regional variation in the aetiology of neonatal jaundice and G6PD deficiency seems to be an uncommon problem in our area. G6PD deficiency was reported in 2.6% neonates by Merchant et al and 3.4% by Narang et al.[19,22]

Conclusion

The present study concluded that physiological jaundice is the most common cause of neonatal jaundice in our hospital and followed by ABO incompatibility, sepsis, Rh incompatibility and idiopathic cases. Less common causes are cephalhematoma, breast feeding jaundice and haemolytic anaemia. Understanding the aetiological and risk factors for neonatal jaundice in our setting helps in prioritizing the group of neonates who require more intensive monitoring for early identification and timely management of this condition.

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