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Original Research Article

Aetiological factors and clinical profile of neonatal jaundice from a rural area of North Kerala, India

Shemeena Valiyat[1]*, Harsha T. Valoor[2], Sayujya Radhamadhavan[3], Salina Sasi Vayalil[3]

[1]Department of Pediatrics, Kunhitharuvai Memorial Charitable Trust Medical College, Mukkam, Kerala, India
[2]Department of Pediatrics, Government Medical College, Manjeri, Kerala, India
[3]Department of Pathology, Kunhitharuvai Memorial Charitable Trust Medical College, Mukkam Kerala, India

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***Correspondence:**
Dr. Shemeena Valiyat,
E-mail: drshemeenav@gmail.com

ABSTRACT

Background: Neonatal jaundice is the most common problem in the first week of life leading to delayed hospital discharge and readmissions. Early recognition of neonatal hyperbilirubinemia is important to prevent serious complications. This study was done in a teaching hospital (KMCT Medical College, Mukkam, Kozhikode), in a rural area of North Kerala. It is an attempt to identify the common aetiological factors of neonatal jaundice in this setting.
Methods: This observational study was conducted over a period of 6 months from January 2014 to June 2014. A total of 110 jaundiced neonates were enrolled. Data collection was done by history taking, clinical examination and relevant laboratory investigations.
Results: In this study, out of 110 jaundiced neonates, 102 (92.5%) were term babies and 8 (7.3%) were preterm, 69 (62.75%) were males and 41 (37.27%) females. Physiological jaundice was seen in 44 (40%) of neonates. Various other aetiologies were ABO incompatibility 24 (21.8%), sepsis 11 (10%), Rh incompatibility 9 (8%), idiopathic 9 (8%), prematurity 8 (7.3%), cephalhematoma 7 (6.4%), breast feeding jaundice 7 (6.4%) and haemolytic anaemia 1 (0.9%).
Conclusions: Physiological jaundice accounted for the bulk of cases of neonatal jaundice in our area. This was followed by ABO incompatibility. This highlights the importance of appropriate monitoring of neonates with this underlying risk factor.

Keywords: ABO incompatibility, Neonatal hyperbilirubinemia, Physiological jaundice, Rh incompatibility

INTRODUCTION

Jaundice is the most common problem in the first week of life worldwide. It is observed in 60% of full term infants and 80% of preterm babies in the first week.[1] Jaundice is also the commonest reason for delayed hospital discharge and readmissions in the first week of life.[2,3]

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.

The incidence, etiological and contributory factors of neonatal jaundice vary according to ethnic and geographic differences.[4] These factors in developing

countries may be different from those of developed nations, probably as a result of racial, cultural and environmental differences. Though we have many studies addressing these factors from other parts of the world and a few studies from other parts of India, there are very few studies from this part of the country. 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.[5]

The objective of this study was to study the clinical profile and the underlying aetiological factors leading to neonatal jaundice in this rural setting of North Kerala.

METHODS

This prospective observational study was conducted in the neonatal intensive care unit (NICU) and Post Natal ward of KMCT Medical College, Kozhikode. The study was conducted over a period of 6 months (January 2014 to June 2014). A total of 380 neonates were admitted in our NICU and post-natal ward during the specified period. Out of these 110 newborns were jaundiced (Serum bilirubin >10 mg/dl).[6,7] So a total of 110 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.

Informed consent was obtained from the parents. Data was analysed. Percentages and ratios were calculated. Obtained results were depicted in Tables and Figures.

RESULTS

Results were expressed as percentages and ratios. There was no comparative group in this study.

Out of 110 jaundiced neonates, 102 were born at term (92.7%) and the remaining 8 were preterm babies (7.3%) (Table 1).

Table 1: Distribution of jaundiced neonates according to gestational age (n=110).

Gestational age (weeks)	Number	Percentage
≥ 37	102	92.73
34-36	07	6.36
30-34	01	0.9
Total	110	100

Among 110 neonates studied, majority had birth weight between 2501g and 3000g (52.73%). Only 12 babies had birth weight <2.5kg (10.9%) (Table 2).

Table 2: Distribution of jaundiced neonates based on birth weight (n=110).

Birth weight (Grams)	Number	Percentage
1000-1500	01	0.9
1501-2000	04	3.64
2001-2500	07	6.36
2501-3000	58	52.73
>3000g	40	36.36
Total	110	100

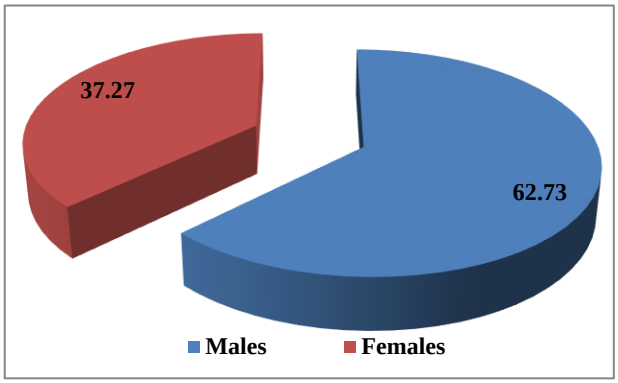

Figure 1: Sex distribution of neonates with hyperbilirubinemia (n=110).

Of the 110 neonates 69 were males (62.75%) and 41 were females (37.27%) (Figure 1).

Here maximum number of cases was due to physiological jaundice 44 (40%) (Table 3).

Table 3: Aetiology of neonatal jaundice.

Aetiology	Number	Percentage
Physiological	44	40
ABO incompatibility	24	21.8
Sepsis	1	10
Rh incompatibility	9	8
Idiopathic	9	8
Prematurity	8	7.3
Cephalhematoma	7	6.4
Breast feeding	7	6.4
Haemolytic anaemia	1	0.9
G6PD deficiency	0	0
Hypothyroidism	0	0

This was followed by ABO incompatibility which constituted 21.8%. Of these 50% were due to OA incompatibility and 50% due to OB incompatibility. Other common causes were sepsis (10%), Rh incompatibility (8%), idiopathic (8%) and prematurity (7.3%). Neonatal jaundice was attributed to cephalhematoma and breast feeding in 7 cases each (6.4%). There was one case of haemolytic anaemia diagnosed as hereditary spherocytosis.

DISCUSSION

In this study of ours, majority of the babies with neonatal jaundice were of term gestation. Only 7% 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.[8,9] 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 110 neonates studied, 63% were males and only 37% were females. This matches earlier studies by Effiong et al, Narang et al and Korejo et al where majority of the babies were males.[10-12]

Most of the babies studied had birth weight 2501-3000g (52%), 36% had birth weight >3kg. Only 11% babies had birth weight $\leq$2.5Kg. As our study had 93% term babies, majority had normal birth weight.

In this study, physiological jaundice was the diagnosis in the majority of the cases i.e. 44 out of 110 cases (40%). This is in concordance with previous studies.

Bahl et al had reported that physiological jaundice contributed to the majority (63.8%) of cases studied.[13] Singhal et al (16.7%) and Merchant et al (25.3%) had also shown high incidence of physiological jaundice in their studies.[9,14]

This was followed by ABO incompatibility as the next leading cause of neonatal jaundice (21.8%). This is very similar to the findings by Verma et al and Merchant et al that ABO incompatibility contributed to 22.6% of cases.[15,14] 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.[13,16]

Sepsis constituted 10% 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 [14,15,11]

Rh incompatibility was responsible for 8% 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.[16,15] Our finding is comparable with the study by Singhal et al where Rh incompatibility was present in 8.1% of neonates.[9] Thus ABO incompatibility was more prevalent than Rh incompatibility. This is in agreement with older studies done abroad.[17,18] Similar findings were reported from India too.[9,14,15]

No known cause could be established in 9 cases (8%). Previous Indian studies have reported incidence of idiopathic jaundice to be ranging from 8.8% to 57%.[9,13]

Cephalhematoma contributing to jaundice was found in 6.4% of our cases. This is comparable to the study by Narang et al which found an incidence of 6.3%.[11]

There were 7 cases of breast feeding jaundice (6.4%) which regressed after improving the frequency and method of breast feeding.

There was one case of haemolytic anaemia (0.9%) 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.[14,11]

There were some limitations to this study. It is well known that there may be marked geographic variations in the pattern of aetiological factors in neonatal jaundice. Therefore, our findings may not be reflective of the pattern in other regions. Another drawback was that some aetiological factors leading to neonatal jaundice like

Gilbert syndrome and Criggler-Najar Syndrome were not investigated. Hence there is a possibility that some of the cases classified as idiopathic may have these underlying diagnosis.

CONCLUSION

This study concludes that physiological jaundice is the most common cause of neonatal jaundice in our hospital. This is 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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