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Research Article

Study of Etiology of Neonatal Jaundice at tertiary care centre in Maharashtra

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Abstract: Jaundice is the commonest abnormal physical finding during first week of life. 25-50% of all term newborns and higher percentage of premature infants develop clinical jaundice. The objectives of present study were to know various etiology of neonatal jaundice. The present prospective descriptive study was conducted in GMC, Akola (Maharashtra-India). All neonates admitted in neonatal intensive care unit from period 1st May 2014 to 31st October 2014 were considered for study. Cases which fit in definition of physiological jaundice were not investigated further. Out of 108 jaundiced babies, 60.19% were boys and 39.81 % girls. Out of total, 32.40% were preterm and 67.61% were full term. Physiological jaundice was seen in 44.4% of cases and 55.6% were having pathological cause. Among them sepsis (12%), ABO incompatibility (11.1%), Rh incompatibility (4.6%) were three most common causes observed in present study. The higher the bilirubin level rises, the more justified are additional efforts to determine its cause. Therefore at least newborns presenting with jaundice should be screened for its deficiency of various factors. Screening for G6PD deficiency and other common hemoglobinopathies in neonatal jaundice can be adopted as a non mandatory nationwide screening similar to immunization programme in different regions in India.
Keywords: Hemoglobinopathy, Neonatal Jaundice, Hyperbilirubinemia.

INTRODUCTION

Jaundice is the commonest abnormal physical finding during first week of life. 25-50% of all term newborns and higher percentage of premature infants develop clinical jaundice [1]. Jaundice manifests at serum bilirubin level of 5mg/dl, there is cephalocaudal progression with increasing intensity. Physiological jaundice is the most common cause of hyperbilirubinemia in newborn. About 5% of newborns develop pathological jaundice. With use of anti-D immunoglobulin, incidence of jaundice resulting from Rh incompatibility has decreased significantly. Hereditary hemolytic anemia present as jaundice at birth can lead to indirect hyperbilirubinemia and encephalopathy [2].

Detection of hereditary hemolytic anemia in neonatal period helps in early diagnosis, treatment and prevention [3]. It forms the basis for genetic counseling. Keeping this fact in mind, this study of neonatal jaundice with special reference to hereditary hemolytic anemia is taken. The present study was conducted to know various etiological factors related to neonatal jaundice among jaundiced neonates.

MATERIAL & METHODS

The present prospective descriptive study was conducted in Government Medical College, Akola (Maharashtra-India). All neonates admitted in neonatal intensive care unit from period 1st May 2014 to 31st October 2014 (total six month) were considered for study. Detail history, clinical examination, serum bilirubin estimation, maternal & baby's blood group is carried out in each case. If maternal blood group is Rh negative and baby's blood group is Rh positive or in case O-AB incompatibility, direct coombs test was performed. Cases which fit in definition of physiological jaundice were not investigated further. If peripheral blood smear picture and / or reticulocyte count is suggestive of hemolysis without blood group incompatibility then G6PD assay, hemoglobin electrophoresis, sickling test & osmotic fragility test was carried out in such patients. G6PD test was done by fluorescent essay, sickling test by using 2% sodium metabisulphite, osmotic fragility carried out in serial dilutions of sodium chloride, hemoglobin electrophoresis (detection of HbS) Patients with direct hyperbilirubinemia were examined clinically in details for sepsis, neonatal metabolic disorders like galactosemia, congenital intrauterine infections. Test for particular disease was carried out on basis of high clinical suspicion of condition. Informed consent was

taken from the neonate's mother. Purpose of study was explained to them.

RESULTS

Out of 108 jaundiced babies, 65 (60.19%) were boys and 43 (39.81 %) girls. Out of total, 35 (32.40%) were preterm and 73 (67.61%) were full term. Physiological jaundice was seen in 44.4% of cases and 55.6% were having pathological cause.

Out of 108 patients, 48 (44.4%) were having physiological jaundice. Remaining 60 were having pathological causes. Among them sepsis (12%), ABO incompatibility (11.1%), Rh incompatibility (4.6%) were three most common causes observed in present study. Sepsis associated with neonatal jaundice was the presenting complaint in only 4 (30.7%) cases. The remaining were brought with complaints of lethargy, poor feeding, fever, difficulty in respiration and convulsions. Total serum bilirubin levels in these babies ranged from 10-20 mg/dl. Jaundice on an average lasted 12 days in these babies with duration of jaundice being longer (14 days) preterm than in term (10 days) babies.

Jaundice was attributable to ABO blood group incompatibility in 12 cases (11.1 %). Onset of jaundice varied from 18-48 hours after birth and the peak was seen on 5-8 'days of life. Blood group O-B incompatibility was observed 7(58.3%) and O-A incompatibility in 5(41.6%) of the mother child pair. Predominantly unconjugated hyperbilirubinemia with serum bilirubin ranging from 16 to 30 mg/dl was seen and jaundice lasted from 5-15 days (mean 10 days in these neonates). Breast milk jaundice was found in 3 (2.8%) of the babies. Jaundice was first detected 6-8 days after birth. Predominantly unconjugated hyperbilirubinemia with peak serum bilirubin levels of 15-18 mg/dl was seen. Jaundice in these babies lasted for 14-16 days of life. Rh incompatibility accounted for 5 (4.6%) of cases. History of previous still birth was present in one of the mothers. Jaundice developed within 24 hrs after birth in all the cases. Large cephalhematoma leading to neonatal jaundice was found in another 3 (2.8%) of our cases. Jaundice appeared 48-96 hours after birth in these cases & peak serum bilirubin values of 16-19 mg/dl were observed.

Jaundice because of G6PD deficiency was found in 1 (0.9%) of case which was a male. Onset of jaundice was on 3rd day of life and lasted for 7 days, with peak on 5th day (20.2mg/dl). There was history of consanguinous marriage and also death of previous 2 babies, Ist at the age of 11 months with cause not known and other was intrauterine death. No ABO or Rh incompatibility was present and coombs test was negative. Other child with hemolytic anemia turned out to be IgM CMV positive. Baby on 1st day had anemia Hb (8.8%), with jaundice (S.bil -14.Omg/dl) with peripheral smear showing evidence of hemolysis with retic count 18%. There was no ABO or Rh incompatibility. Hemoglobin electrophoresis, sickling, G6PD, DCT was negative. TORCH testing was done out of which IgM CMV turned out positive. On follow up later, baby also developed cataract and hydrocephalus.

Two patients of sickle cell trait were diagnosed. In one of babies, mother was a known case of sickle cell disease. Baby had only physiological jaundice. Sickling test was positive with hemoglobin analysis showing FAS pattern. Another baby diagnosed with sickle cell trait, mother known case of sickle cell disease also had history of birth asphyxia and prematurity. Baby developed only physiological jaundice, she had sickling test positive, hemoglobin analysis showing FAS pattern, smear showing evidence of hemolysis, Hb of 12 g/dl and reticular count of 6.5%. Despite intensive investigations, no etiology was identified in 22 (20.4%) of cases. Among them peak serum bilirubin ranged from 14-20 mg/dI. Jaundice in these neonates lasted 8-14 days of life.

Table-1: Base-line data of study subjects

Sex	Number of Cases	Percentage
Male	065	60.19
Female	043	39.81
Total	108	100
Term wise distribution of study subjects		
Preterm	035	32.40
Term	073	67.60
Total	108	100
Type of jaundice and distribution of study subjects		
Physiological	048	44.40
Pathological	060	55.60
Total	108	100

Table-2: Various etiological factors observed in neonates having neonatal jaundice

Causes of neonatal jaundice	No. of Babies	Percentage
Physiological	048	44.4
ABO incompatibility	012	11.1
Rh incompatibility	005	04.6
Sepsis	013	12.0
Breast Milk Jaundice	003	02.8
Cephalhematoma	003	02.8
G6PD deficiency	001	00.9
Cytomegalo virus infection	001	00.9
Idiopathic	022	20.4

DISCUSSION

The present study reviews the factors responsible for neonatal jaundice. In number of cases, however, even the most sophisticated investigations fail to reveal any etiological factors and these cases are then labeled as idiopathic. The frequency of such cases varies depending upon the investigative facilities available. Hereditary hemolytic anemia at birth can lead to indirect hyperbilirubinemia and encephalopathy. Detection of hereditary hemolytic anemia in neonatal period helps in early diagnosis, treatment and prevention. Keeping this fact in mind, this study of neonatal jaundice with special reference to hereditary hemolytic anemia is undertaken.

In the present study incidence of pathological jaundice (55.6%) was more as compared to other studies like Narang A et al [4], Singhal P.K. [5], Bahl L et al [6]. However our study cannot be compared directly with other reports as our study is based on selective group of hyperbilirubinemic neonates admitted in NICU. In previous studies done in Lucknow Bajpai PC et al [7], Pune (Anand VR et al [8], Shimla Bahl L et al [6], incidence of physiological jaundice was observed to be 57%, 47.6% and 63.8% respectively which is similar to ours of 44.4%.

Among the conditions leading to hemolysis, ABO incompatibility was the most important etiological factor. . Blood group O-B incompatibility was observed in 7 and O-A incompatibility in 5 of the mother child pair. Bahl et al [6], however, reported a higher incidence (60%) of OA incompatibility as against study by Bajpai P C et al [7] who reported higher incidence of O-B incompatibility. The difference could be due to different distribution of blood group among population in various regions.

Our study reported Rh incompatibility in 4.6 %. Various Indian studies have reported incidence from 1.6% Bajpai P C et al [7] to 9.8% Verma M et al [9]. Breast milk jaundice was found in 2.8% which is similar to study by Bahl L, Sharma R et al [6]. Cephalhematoma accounted for 2.8% in study conducted by Singhal PK [5]. Jaundice due to G6PD deficiency was found in 0.9% of cases. This is lower as compared to other studies like Narang A et al [4], Verma M et al [9]. Various studies in India have shown a variable frequency ranging from 0.2 to 16%. Higher frequency in male is in conformity with earlier observations. First expanded neonatal screening programme initiated in Hyderabad to screen all the newborns born in four major Government Maternity Hospitals by heel prick capillary blood collected on S & S-903 filter paper [10]. Out of 8000 newborns screened for the red cell enzyme G6PD, 4 were tested positive with an incidence of 1:2000. The importance of the study lies in the fact that incidence of complications due to this disease can be brought down by preventive measures. 50% of cases were male and 50% females. Our study had used same newborn screening method for testing babies for G6PD deficiency. Routine screening of all newborns for G6PD deficiency may not be feasible in our country due to financial constraints and lack of government support.

Two patients of sickle cell trait did not develop pathological jaundice. One was small for gestational age and other had birth asphyxia and respiratory distress. Study conducted by Brown A K et al [11] on newborns with sickle cell disease have shown complication as jaundice, fetal distress, anemia and respiratory distress.

No etiological factor for the jaundice could be established in 22 (20.4%) babies. The main yield of routine hyperbilirubinemia evaluation was the diagnosis of ABO and Rh isoimmunization. Various Indian studies have shown incidence of idiopathic hyperbilirubinemia to be ranging from 8.8% [5] to 57 % [6], Verma M et al [9]. Test recommended for diagnosis of other causes are expensive. A rational determination of which laboratory test to do on infants with jaundice requires information that is currently unavailable.

CONCLUSION

Until more definitive data are available, we recommend a flexible response in which clinician orders tests they find easy to interpret when non physiologic jaundice seems likely. The higher the bilirubin level rises, the more justified are additional efforts to determine its cause. Therefore at least

newborns presenting with jaundice should be screened for its deficiency of various factors and hemoglobinopathies. Close surveillance is required for G6PD deficient infants for the first week of life, the period of increased risk. Future catastrophes will also be prevented due to timely detection of deficient status and genetic counseling can be done. Recent resurgence of malaria and the increased use of anti-malarials and antipyretics makes this all the more important. Screening for G6PD deficiency and other common hemoglobinopathies in neonatal jaundice can be adopted as a non mandatory nationwide screening similar to immunization programme in different regions in India. The impact of such screening will be definitely great in terms of the benefit it provides to the society by preventing mental retardation and cases of cerebral palsy due to neonatal jaundice.

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