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Original Research Article

Study of Neonatal Hyperbilirubinemia at a Tertiary Care Hospital

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ABSTRACT

Objectives: The purpose of this study is to estimate the incidence of neonatal hyperbilirubinemia.

Methods: All treated cases of neonatal hyperbilirubinemia were analyzed and data on gender, gestation age, mode of delivery, blood group incompatibility, sepsis, parity and birth weight were obtained.

Results: Out of 700 newborns, 400 (57.14%) newborns developed clinical jaundice. Out of 400 newborns with clinical jaundice, 175 (50%) newborns developed physiological jaundice and 107 (39%) newborns developed non physiological jaundice requiring therapeutic intervention in the form of phototherapy or exchange transfusion.

Conclusion: Blood Group incompatibilities, sepsis, and cephalohematoma were the common causes of hyperbilirubinemia, however in nearly a third of all cases etiology could not be determined. Preterm gestation and low birth weight were associated risk factors. Present study concludes that the leading cause of pathological jaundice is ABO incompatibility followed by SGA Baby and sepsis.

Keywords: Hyperbilirubinemia, Non-Physiological Jaundice, ABO Incompatibility.

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INTRODUCTION

Neonatal jaundice affects up to 84% of term newborns[1] and is the most common cause of hospital readmission in the neonatal period. Severe hyperbilirubinemia (total serum bilirubin [TSB] level of more than 20 mg per dL [342.1 µmol per L]) occurs in less than 2% of term infants and can lead to kernicterus (i.e., chronic bilirubin encephalopathy) and permanent neurodevelopmental delay.[2]

Neonatal hyperbilirubinemia is a common clinical problem encountered during the neonatal period; especially in the first week of life.[3] Nearly 8% to 11% of neo-nates develop hyperbilirubinemia.[4,5] When the total serum bilirubin (TSB) rises above the 95th per-centile for age (high-risk zone) during the first week of life, it will be considered as hyperbilirubinemia. Between 60%-80% of healthy infants are expected to present with idiopathic neonatal jaun-dice.[6] Neonatal jaundice is the discoloration of skin and sclera color to yellowish in a newborn by bilirubin.[7]

Some of the most common causes of neonatal jaundice include physiological jaundice, breast feeding or non-feeding jaundice, breast milk jaundice, prematurity leading to jaundice & various pathological causes like haemolytic disease, liver dysfunction, neonatal sepsis, deficiency of G6PD enzyme, hypothyroidism and rare conditions such as Gilbert's syndrome etc.[8]

Extreme hyperbilirubinemia is rare, however, if left untreated especially in premature infant, indirect hyperbilirubinemia may lead to kernicterus, a serious neurological problem and social & economic burden on the patient's family & society. Elevation of direct bilirubin constitute the pathological causes of jaundice & should be promptly treated either by medical or surgical means. Worldwide about 24 million neonates are born each year who are at risk of complications due to hyperbilirubinemia. Although kernicterus is preventable with prompt treatment, it is estimated that at least 114,000 infants die every year from hyperbilirubinemia, and more than 63,000 live with permanent neurological impairment.[3] The purpose of this study is to estimate the incidence of neonatal hyperbilirubinemia.

MATERIALS AND METHODOLOGY

The present study was conducted in Department of Paediatrics, Geetanjali Medical College & Hospital, Udaipur, Rajasthan, India. A predesigned proforma has aided the enrollment of newborns into the study. Significant hyperbilirubinemia was defined as the value of bilirubin according to AAP guidelines in term neonates and Cockington's charts in preterm, above which phototherapy or exchange transfusion or both are required.[9,10] Clinical jaundice is visible yellowish discoloration of skin of newborns in day light. The

following situations suggest non physiologic jaundice and require evaluation {onset of jaundice occurs before 24 hours of age, elevation of serum bilirubin requires phototherapy, a rise in serum bilirubin levels of 0.2mg/dl/hour, signs of underlying illness in any infant (vomiting, lethargy, poor feeding, excessive weight loss, apnea, tachypnea or temperature instability), jaundice persisting after 8 days in a term baby or after 14 days in a premature infant.[11]

Inclusion Criteria: All babies born at postnatal ward, who had clinical jaundice irrespective of the gestational age and birth weight was included in the present study.

Exclusion Criteria

1. Babies with major congenital malformations.
2. Newborns who expired before complete evaluation.

Statistical Analysis

Descriptive data are presented as number and percentages. Chi-square test was used to assess the association between neonatal jaundice with various factors. Microsoft word and SPSS software were used for the analysis of the results. A p value of 0.05 or less was considered for statistical significance.

Table 1: Causes of Neonatal Jaundice requiring photo therapy and exchange blood transfusion

Causes	Phototherapy No	Exchange blood transfusion No
ABO incompatibility	35	8
Rh incompatibility	10	6
Sepsis	25	3
SGA	30	3
HIE	20	0
Total	120	20

RESULTS

In the present study, there were 700 newborns delivered during the study period and 400 (57.14%) newborns developed clinical jaundice. Out of 400 newborns with clinical jaundice, 200 (50%) newborns developed physiological jaundice. The overall incidence of non-physiological jaundice in the study group is 25%. (175 out of 700 newborns)

Sex factor had an influence on the incidence of non-physiological jaundice among the neonates showing that males 71.4% (125 out of 175) had higher incidence compared to females 28.6% (50 out of 175) with p value <0.05. All 4 (100%) newborns with history of birth asphyxia developed pathological jaundice.

DISCUSSION

In our study 400 neonates required either phototherapy or exchange transfusion as treatment for unconjugated hyperbilirubinemia. The incidence of hyperbilirubinemia in our study was 17%. The most common etiology or risk factor implicated was ABO incompatibility. These observations are similar to the results of other studies. A study done in Kahramanmaraş, Turkey concluded that ABO incompatibility was the most common cause of severe neonatal hyperbilrubinemia.[12] An Iranian study in Fars province revealed that the most common causes of severe indirect hyperbilirubinemia were sepsis, blood group incompatibility, G6PD deficiency, and unknown. Risk factors of severe hyperbilirubinemia were Male sex, previous siblings with severe hyperbilirubinemia, male sex, normal vaginal delivery, and breast feeding.[13]

In the present study the overall incidence of non-physiological jaundice is 25% (175 out of 400). In a study conducted by Anil Narang et al. in 1996 at Nehru hospital, Chandigarh, of 3791 live births, 551 (14.5%) developed neonatal jaundice needing therapeutic intervention, i.e., either phototherapy or exchange transfusion.[14]

A Croatian study showed that neonatal jaundice was associated with birth weight, maternal infections, gestational age and premature rupture of membranes.[15] More than one risk factor may be present in a neonate, both preterm gestation age and blood group incompatibility may co- exist and contribute in causing hyperbilirubinemia. A review article published in North America suggested that the etiology of neonatal hyperbilirubinemia is multifactorial. Late preterm gestational age, exclusive breastfeeding, glucose-6-phosphate dehydrogenase deficiency, ABO haemolytic disease, East Asian ethnicity and a history of a sibling being treated for hyperbilirubinemia were the most common risk factors associated.[16]

Breastfeeding jaundice tops the list in non-physiological jaundice. In a study done by Osborn LM et al, it was found that Breastfed newborns may be at increased risk for early-onset exaggerated physiologic jaundice because of relative caloric deprivation in the first few days of life.[17] In another study done by Schneider AP, it was stated that decreased volume and decreased frequency of feedings may result in mild dehydration and the delayed passage of meconium. Compared with formula-fed newborns, breastfed infants are three to six times more likely to experience moderate jaundice (total serum bilirubin level above 12 mg per dL).[18]

ABO incompatibility has become the second most common cause of non-physiological jaundice in newborn in our study. In a study of a population of newborns in Turkey, there was a 25% incidence of ABO incompatibility, with 21.3% of these babies exhibiting significant hyperbilirubinemia and 4.4% exhibiting severe ABO hemolytic disease.[19] The reason behind having more premature babies may be lack of education, poor socioeconomic condition and early marriage.

CONCLUSION

Present study concludes that the leading cause of non-physiological jaundice is ABO incompatibility followed by SGA Baby and sepsis. Physiological jaundice contributes maximum number of cases among total cases. Neonatal jaundice was prevalent among 31.4% of neonates. Premature babies were higher in number (59%). Most common cause of Neonatal Jaundice was physiological jaundice followed by ABO incompatibility and sepsis. Mostly the cases of ABO

incompatibility, Sepsis and SGA required phototherapy. Exchange blood transfusion required in few cases of ABO incompatibility, Rh incompatibility, sepsis and SGA. Preterm gestation, primi delivery and low birth weight also showed a strong association with neonatal hyperbilirubinemia.

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