PHYSIOLOGICAL JAUNDICE IN 51 NEONATES

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ABSTRACT

Background: Neonatal jaundice is the common reason for admission & readmission of neonates to hospital throughout the ward. Among other causes of neonatal jaundice, physiological jaundice is the leading causes. It is due to short life span of neonatal red blood cells & due to immaturity of liver ability to metabolize bilirubin.

Results: Total number of neonates was 51, male 22 & female 29, 44 were breast fed while 6 were bottle fed, physiological jaundice accounted for 36 (64.8%), Rh incompatibility for 5 (9.3%), G6PD deficiency for 5 (9,3%), sepsis 3 (5.6%), hereditary spherocytosis 1 (1.9%). Hypothyroid 1 (1.9%). The mean bilirubin was 8.21 mg/dl SD 1.83. Breast feeding was statistically highly significant with physiological jaundice (P 0.0001).

Conclusion: Breast feeding is statistically significantly associated with physiological jaundice.

Key words: Jaundice, physiological.

INTRODUCTION

Neonatal jaundice may have first been described in a Chinese textbook 1000 years ago. Jaundice is the most common condition that requires medical attention and hospital readmission in newborns.[1] The yellow coloration of the skin and sclera in newborns with jaundice is the result of accumulation of unconjugated bilirubin. In most infants, unconjugated hyperbilirubinemia reflects a normal transitional phenomenon. However, in some infants, serum bilirubin levels may rise excessively, which can be cause for concern because unconjugated bilirubin is neurotoxic and can cause death in newborns and lifelong neurologic sequelae in infants who survive (kernicterus).[2] For these reasons, the presence of neonatal jaundice frequently results in diagnostic evaluation. Hyperbilirubinemia continues to be the most common cause of neonatal readmission to hospitals in North America.[1–5] This pattern continues despite attempts to identify newborns at risk of clinically important hyperbilirubinemia before they are discharged from hospital.6–9 Long-term results of hyperbilirubinemia, including bilirubin encephalopathy and kernicterus, were thought to be rare since the advent of exchange transfusion, maternal rhesus immunoglobulin prophylaxis and phototherapy.10–12 However, cases of kernicterus have been reported recently in healthy near-term and term infants with no evidence of hemolytic disease or other risk factors.[13,14] As a result, a sentinel event alert was issued by the US Centers for Disease Control and Prevention to identify cases of kernicterus in healthy term infants.[11] The resurgence of severe neonatal hyperbilirubinemia and kernicterus (a largely preventable disease) is of grave concern. Risk factors recognized to be associated with hyperbilirubinemia in newborns have included jaundice in the first 24 hours of life, jaundice noted before discharge from hospital, a sibling who had jaundice treated with phototherapy, near-term gestational age of 35–36 weeks, Asian race and the presence of infant bruising or cephalhematoma.[15–17] Causes identified by laboratory investigations include rhesus and ABO incompatibility, as well as glucose-6-phosphate dehydrogenase (G6PD) deficiency.[18,19] We conducted this study to estimate the incidence of hyperbilirubinemia in Canada and to determine the underlying causes, which would be of value in identifying and implementing strategies to prevent morbidity from this condition.

Place of study

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Study design

A random sample of convince of 95 neonates, presenting with neonatal jaundice. Out of them 49 were excluded as they had conjugated hyperbilirubinemia due to other causes like hepatitis, inborn errors of metabolism etc. 50 neonates had unconjugated hyperbilirubinemia. Neonates who less than 36 weeks old were excluded from study.

RESULTS

Total no of neonates was 51, male 22 & female 29, 44 were breast fed while 6 were bottle fed, physiological jaundice accounted for 36 (64.8%), Rh incompatibility for 5 (9.3%), G6PD deficies for 5 (9,3%), sepsis 3 (5.6%), hereditary spherocytosis 1 (1.9%). Hypothyroid 1 (1.9%). The mean bilirubin was 8.21 mg/dl SD 1.83. Breast feeding was statistically highly significant with

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Statistics: Bilirubin Level

N	Valid	51
	Missing	3
Mean		8.2157
Median		8.0000
Mode		8.00
Std. Deviation		1.83645

One-Sample Test

	Test Value = 0					
	T	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
Bilirubin	31.948	50	.000	8.21569	7.6992	8.7322
Breastfeeding	6.112	50	.000	1.33333	.8952	1.7715

physiological jaundice (P 0.0001).

DISCUSSION

One of the most prevalent clinical conditions in neonatology is hyperbilirubinemia[1]. Neonatal hyperbilirubinemia is a common clinical problem encountered during the neonatal period, especially in the first week of life[2,3]. Nearly 8% to 11% of neonates develop hyperbilirubinemia. When the total serum bilirubin (TSB) rises above the 95th percentile for age (high-risk zone) during the first week of life, it will be considered as hyperbilirubinemia[4,5].

Between 60%–80% of healthy infants are expected to present with idiopathic neonatal jaundice[6]. Neonatal jaundice is the discoloration of skin and sclera color to yellowish in a newborn by bilirubin[7]. Therefore it can create concern in the physician and anxiety in the parents. According to National Neonatal-Perinatal Database (NNPD) the incidence of neonatal hyperbilirubinemia in in-house live-births is 3.3%, while in extramural admissions morbidity due to hyperbilirubinemia accounted for 22.1%[8]. In neonates, the dermal icterus is first noted in the face and when the bilirubin level rises, it proceeds to the body and then to the extremities. This condition is common in 50%–60% of newborns in the first week of life[8].

Bilirubin is not merely a nuisance molecule that has dire consequences, but bilirubin such as uric acid is an important antioxidant circulating in biologic system of neonate[9–11]. However, high bilirubin levels can be toxic for central nervous system development and may cause behavioral and neurological impairment even in term newborns[12–14]. Five to ten percent of newborns developed jaundice required the management of hyperbilirubinemia[15]. Neonatal jaundice may be on account of different parameters such as birth weight, gestational age, premature rupture of membranes, maternal infectious diseases or other illness during pregnancy, having different sources of origin, hence having different types[16].

Several types of Bilirubinemia have been reported in neonates including physiological jaundice, pathological jaundice, jaundice due to breastfeeding or breast milk and hemolytic jaundice including three subtypes due to Rh factor incompatibility, ABO blood group incompatibility and Jaundice associated with Glucose-6-phosphate dehydrogenase (G6PD) deficiency[24].

Physiological jaundice is the commonest type of newborn hyperbilirubinemia, having no serious consequences[25]. Neurodevelopmental abnormalities including as athetosis, loss of hearing, and in rare cases intellectual deficits, may be related to high toxic level of bilirubin[26]. Jaundice attributable to physiological immaturity which usually appears between 24–72 h of age and between 4th and -5th days can be considered as its peak in term neonates and in preterm at 7th day, it disappears by 10–14 days of life[27]. Unconjugated bilirubin is the predominant form and usually its serum level is less than 15 mg/dl[28]. Based on the recent recommendations of the AAP, bilirubin levels up to 17–18 mg/dl may be accepted as normal in term of healthy newborns[15].

Bilirubin levels with a deviation from the normal range and requiring intervention would be described as pathological jaundice[25]. Appearance of jaundice within 24 h due to increase in serum bilirubin beyond 5 mg/dl/day, peak levels higher than the expected normal range, presence of clinical jaundice more than 2 weeks and conjugated bilirubin (dark urine staining the clothes) would be categorized under this type of jaundice.

Exclusively infants with breastfeeding have a different physiological pattern for jaundice compared with artificially feed babies[24]. Jaundice in breast fed babies usually appears between 24–72 h of age, peaks by 5–15 days of life and disappears by the third week of life. Higher bilirubin levels have been reported in these infants[29]. In case of breastfed newborns, mild jaundice may take 10–14 days after birth or may reoccur during the breast feeding period[30]. Very large amounts of bilirubin rarely accumulate in the blood and cause cerebral lesions, a situation known as nuclear jaun-

dice[31]. These cuts may be followed by hearing loss, mental retardation, and behavioral disorders. A mild clinical jaundice has been observed in one third of all breastfed babies in the third week of life, which may persist for 2 to 3 months after birth in a few babies[32]. Decreased frequency of breastfeeding is associated with exaggeration of physiological jaundice. One of the significant procedures to manage the jaundice in a term healthy baby is the mothers' encouragement to breastfeed their babies at least 10–12 times per day[33]. In our study breast feeding was statistically significantly associated with hyperbirubinemia (P 0.0001)

Hyperbilirubinemia is also associated with breast milk of mother in neonates[34]. About 2%–4% of exclusively breastfed babies have jaundice in excess of 10 mg/deal in the third week of life. These babies in the third week of life with bilirubin serum levels higher than 10mg/dl should be considered for prolonged jaundice[35]. A diagnosis of breast milk jaundice should be investigated if the serum bilirubin is predominantly unconjugated, other causes of prolonged jaundice have been eliminated and the infant is in good health, vigorous and feeding well and gaining weight adequately[36]. Mothers should be advised to continue breastfeeding at more frequent intervals and bilirubin levels usually diminish gradually. Discontinuity of breastfeeding is not recommended unless levels exceed 20 mg/dl[37].

The most common causes of hemolytic jaundice include (a) Rh hemolytic disease, (b) ABO incompatibility and (c) Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency and minor blood group incompatibility.

Rhesus hemolytic disease of the newborns (RHDN) results from maternal red-cell alloimmunization[38]. Maternal antibodies are produced against the fetal red blood cells, when fetal red blood cells are positive for a certain antigen, usually at what time a baby having Rh positive born to an Rh-negative mother[21] (and Rh-positive father), then maternal immunoglobulin (IgG) antibodies might cross the placenta into the fetal circulation and cause a wide variety of symptoms in the fetus, ranging from mild to severe hemolytic anaemia and fetal hydrops[39,40]. To facilitate early treatment in neonates who are dubitable to have Rh factor, a blood group and Rh typing, DCT, PCV (packed cell volume) and serum bilirubin on cord blood should be performed. A reticulocyte count should be sent before the first exchange transfusion (ET). Vigorous phototherapy is required immediately after the birth and it should be continued until a level, which is 5 mg/dl less than the level estimated for exchange blood transfusion[41]. In preterm babies, lower values of intervention for treatment of Rh hemolytic disease have been demonstrated. Phototherapy and exchange blood transfusion are recommended when a level is greater than 0.5% and 1% birth weight (kg) respectively[29]. Eight intravenous immunoglobulin (IVIG) can be used in a dose of 500 mg/kg 12 hourly × 2 doses after the first ET. After the first ET starting of Phenobarbitone 5 mg/kg/day × 5 may be recommended[24].

The incidence of the incompatibility of the ABO blood groups of the mother and fetus, when the mother has the blood group O and the newborn has the A or B blood group, is 15–20% of all pregnancies[42]. Babies with O-blood group mothers should be closely checked for and discharged after 72 h. Routine cord blood screening is not recommended for newborns with O-group mothers[43]. Jaundice owing to ABO incompatibility usually appears 24 h after the birth. In the presence of significant jaundice or jaundice appearing within 24 h, the work up for pathological jaundice should be done[44]. Intensive phototherapy is advised at SB 12–17 mg/dl depending upon postnatal age of the baby. Exchange blood transfusion is indicated at TSB. The weight at birth as a criterion for phototherapy and ET may be used for preterm newborns[45].

Deficiency, hereditary spherocytosis, and minor group incompatibilities should be managed similar to ABO incompatibility. G6PD, most common enzymopathy, is the deficiency of an enzyme in RBCs. It is the most vital disease of the pathway of hexose monophosphate. Investigations for G6PD deficiency should be considered in infants with severe jaundice in a family with a history of significant jaundice or in a geographic origin associated with G-6-PD deficiency. Decreased bilirubin conjugation resulted from variation in the UGT1A1 and OATP2 genes play an important role in the progression of hyperbilirubinemia in G6PD deficient newborns[17].

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