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CLINICO-DEMOGRAPHIC PROFILE OF NEI ARES ADMITTED WITH HYPERBILIRUBINEMIA IN GB PANT HOSPITAL KASHMIR

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ABSTRACT

Aims and Objective: The purpose of this study was to determine the occurrence, etiological and other associated factors of neonatal hyperbilirubinemia in GB Pant hospital, Kashmir. Jaundice is a common problem in neonatology. Early recognition of the cause of jaundice is very important as delay in management may lead to serious complications or even death.

Materials and methods: In present study, newborns with jaundice were evaluated during a six months period between Nov 2014 – April 2015. 124 newborns with jaundice were enrolled in the study. Data regarding demographic profile of new born, physical examination and laboratory investigations were gathered and analyzed to interpret the common etiologies giving rise to neonatal hyperbilirubinemia.

Results: Out of 124 cases of neonatal hyperbilirubinemia, 24 cases were of physiological jaundice, breast feeding jaundice, breast milk jaundice, jaundice due to prematurity and pathological jaundice comprised the rest 100 as 84, 5, 5 and 6 cases respectively. Patholgical causes for jaundice included neonatal sepsis (2cases), neonatal hypothyroidism (2cases), congenital biliary atresia (1case) and ABO incompatibility (1case).

Conclusion: Present study concludes that breast feeding or inadequate feeding jaundice forms the bulk of cases of neonatal hyperbilirubinemia in this region, followed by breast milk jaundice, prematurity jaundice and pathological causes.

INTRODUCTION

Hyperbilirubinemia is a common problem during the neonatal period occurring in upto 60% of term and 80% of preterm babies in the first week of life (Slusher *et al*., 2004 and Haque and Rahman, 2000). Some of the most common causes of neonatal jaundice include physiological jaundice, breast feeding jaundice, breast milk jaundice, prematurity leading to jaundice and various pathological causes like haemolytic disease, liver dysfunction, neonatal sepsis, deficiency of G6PD enzyme, hypothyroidism and rare conditions such as gilbert's syndrome etc. (Madan *et al*., 2004 and Laforgia *et al*., 2002). Extreme hyperbilirubinemia is rare, however, if left untreated especially in premature infant, indirect hyperbilirubinemia may lead to kernicterus, a serious neurological problem and social and economic burden on the patient's family and society (Wang *et al*., 2005 and Ho, 2002). Elevation of direct bilirubin constitute the pathological causes of jaundice and should be promptly treated either by medical or surgical means (Barbara and Kliegman, 2004 and Maisels, 2001). Neonatal jaundice is related to breast feeding in two primary clinical situations: firstly, newborns who receive inadequate breast feeding and have high concentration of indirect bilirubin during the first post natal week (breast feeding jaundice) (Maria and Cecilia, 2007). Rise in indirect bilirubin in this type is due to enhancement of enterohepatic circulation. Moreover, presence of large amounts of bilirubin in meconium and delay in emptying of meconium have been shown to contribute to an increase in serum bilirubin levels in early days of life further increasing intestinal biliary absorption (Weissman *et al*., 1988 and Broderson and Herman, 1963). The jaundice in this case can usually be ameliorated by frequent breast feeding sessions of sufficient duration to stimulate adequate milk production (Cynthia, 2002). Passage of baby through vagina during birth helps stimulate milk production in the mother's body, so infants born by caesarean section are at higher risk for this condition. Secondly, breast fed infant who experience prolonged unconjugated hyperbilirubinemia known as breast milk jaundice (Marian and Cecilia, 2007). Several hypotheses have been proposed for breast milk jaundice, including the presence of UDP glucuronosyltransferase inhibitor, β

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glucuronidase or yet unidentified factor in human milk that could inhibit excretion of bilirubin and results in hyperbilirubinemia. There is increase in enterohepatic circulation of bilirubin in this type also and may be attributed to increased levels of epidermal growth factor (EGF) in breast milk (Kumal *et al*., 2009). Consideration of all these etiologies is essential in evaluating neonates with jaundice. Complete history of newborn, family history, sibling history, complete physical examination and laboratory investigations are key points in managing these patients (Mishra *et al*., 2008). The aim of our study is to determine the underlying aetiologies of neonatal hyperbilirubinemia and to explore most common cause of neonatal jaundice in areas adjoining this tertiary care teaching hospital.

MATERIALS AND METHODS

We limited our study in 124 neonates. Patients' characteristics and general data were documented including age, birth weight, age at onset, type of delivery, type of feeding, history of jaundice in sibling. All cases were thoroughly examined on admission. Various investigations were carried out including serum bilirubin total and conjugated by Jendrasik and Graf method, liver function tests (aspartate aminotransferase, alkaline phosphatases) by IFCC method by fully automatic chemistry analyzer, full blood count by automated cell analyzer, thyroid function tests by automated immunoassay by TOSOH machine, Coomb's test and urine test for culture. A conjugated bilirubin of greater than 20% of total bilirubin was considered to be abnormal. Statistical analysis was made using graph pad prism. Data are shown as mean (S.D.).

RESULTS

According to history and other signs and symptoms of the new born, they were grouped into five classes. 24 cases belonged to physiological jaundice. Breast feeding jaundice -84 cases, breast milk jaundice -5 cases, jaundice due to prematurity -5 cases and pathological jaundice -6 cases (2 of neonatal sepsis, 2 of neonatal hypothyroidism, 1 of congenital biliary atresia and 1 of ABO incompatibility). The mean age of onset of jaundice in these patients was different in different groups (table 1). There is history of hyperbilirubinemia in siblings of 30% of cases. Klebsiella and pseudomonas was detected in blood culture of two cases conferring the diagnosis of neonatal sepsis. TSH and T4 levels in neonatal hypothyroidism were highly abnormal. Reticulocyte count is highest in ABO incompatibility cases as hemolysis is maximum in these newborns. Conjugated bilirubin level and liver function tests were highly abnormal in case of congenital biliary atresia. Demographic profile and general data are summarized in table 1. Laboratory results are summarized in table 2.

DISCUSSION

In current study, breast feeding jaundice emerged as the most common aetiology of neonatal jaundice. Bilirubin level

Table 1. Demographic Profile & General Data of Newborns

Type of Jaundice	Cases	Sex	Age (Days) (SD)	Age of onset (Days) (SD)	Birth Weight (Gram) (SD)	Feeding Method
Physiological	24	M-13	4.33	2.58	2870	BF-91.66%
		F-11	(1.09)	(0.72)	(188.26)	Bot-8.33%
Breast Feeding	84	M-53	6.6	3.19	2848	BF-245
		F-31	(1.49)	(0.78)	(150)	Bot-33.3%
						MF-39%
Breast Milk	5	M-4	22.4	16.6	2765	BF-80%
		F-1	(2.4)	(0.89)	(155)	MF-20%
Prematurity	5	M-3	4.2	2.00	2092	BF-20%
		F-2	(0.84)	(0.71)	(85.03)	Bot-20%
						MF-60%
Biliary Atresia	1	M-1	8	1	2468	BF
Neonatal Sepsis	2	M-2	7.5	1.5	2395	BF-50%
			(0.71)	(0.71)	(148.49)	MF-50%
Neonatal Hypothyroidism	2	M-2	37.5	5.0	2900	BF-100%
			(10.61)	(1.41)	(141)	
ABO Incompatibility	1	F-1	4	1	2568	BF

BF- Breast feeding, Bot F- Bottle feeding, MF- Mixed feeding

Table 2. Laboratory Investigations of Newborns

Type Of Jaundice	Hb%	Reticulocyte Count	Highest Total Bilirubin (mg%)	Conjugated Bilirubin (mg%)	TSH uIU/ml	T_4 ug/dl	ALT u/l	AST u/l	ALP u/l
Physiological	13.54	1.9	6.917	0.158	2.5	14.0	15.88	24	166.13
	(1.25)	(0.42)	(0.67)	(0.072)	(0.41)	(1.38)	(4.07)	(6.7)	(53.28)
Breast Feeding	13.78	2.010	14.908	1.38	2.8	12.35	19.70	21.43	152.08
	(1.25)	(0.30)	(1.96)	(0.22)	(0.34)	(0.88)	(4.41)	(7.16)	(50.41)
Breast Milk	11.40	1.48	15.20	1.88	2.72	11.6	21.6	25.6	92.6
	(2.07)	(0.47)	(1.92)	(0.497)	(0.26)	(1.14)	(4.28)	(2.3)	(17.34)
Prematurity	10.00	1.14	7.6	0.66	1.32	12.2	14.8	19.6	58.2
	(0.71)	(0.27)	(1.14)	(0.13)	(0.37)	(0.84)	(2.77)	(2.30)	(3.19)
Neonatal Sepsis	8.5	5.00	17.00	0.35	0.9	8.5	41	49	67
	(0.71)	(1.41)	(1.41)	(0.07)	(0.14)	(0.71)	(1.41)	(1.41)	(4.24)
Biliary Atresia	13	3	18	16	3.0	13	50	63	4000
ABO Incompatibility	14	9	23	2.4	3.0	13	15	29	160
Neonatal Hypothyroidism	12	0.95	14	2.05	47	1.57	16.5	28	120
		(0.07)	(1.41)	(0.21)	(9.90)	(0.18)	(0.71)	(2.8)	(7.1)

Values are mean (S.D.)

regressed after increasing the frequency and improving the method of breast feeding. Najati *et al*. (2010) also concluded that majority of patients had an unconjugated hyperbilirubinemia probably due to breast feeding. Other causes in their study were G6PD deficiency, hypothyroidism, UTI, septicaemia, Down syndrome and ABO incompatibility. Our study also has few cases of these clinical conditions as a cause of neonatal hyperbilirubinemia. Gartner (2001) stated in his study that insufficient caloric intake resulted from maternal and/or infant breast feeding difficulties may also increase unconjugated serum bilirubin concentration. Another study by Bertini (2001), stated that fasting plays an important role in pathogenesis of neonatal hyperbilirubinemia and forms bulk of the cases. Breast milk jaundice was the diagnosis of exclusion as also stated by Prashant (2010). All 5 newborns were exclusively breast fed.

Icterus started developing after 1st week of life and peak occurred at 3rd week (12-15mg/dl) and thereafter it regressed by its own requiring no treatment. This type is not a clinical disorder but recognized to be a normally occurring extension of physiological jaundice of new born and breast feeding should not be interrupted. Schneider (1986) also concluded in his study that breast milk jaundice is one of the common causes of neonatal jaundice. Prematurity was another reason for neonatal hyperbilirubinemia. 5 cases were there. These newborns are prematurely delivered at the mean gestational age of 32 ± 2 weeks. 1 patient out of 5 showed neurological sequelae depicting the diagnosis of kernicterus (Camille *et al*., 2004). Onyearugha *et al*. (2011), concluded prematurity as the second leading cause of neonatal jaundice. According to another study, these neonates fed on human milk had higher peak concentration of plasma bilirubin and more prolonged hyperbilirubinemia than those fed on an artificial infant formula (Lucas and Baker, 1986).

Two cases of neonatal sepsis were reported. Blood culture showed presence of klebsiella and pseudomonas respectively. Extensive chemotherapy was instituted resulted in regression of jaundice. Onyearugha *et al*. (2011) also found sepsis as the second leading cause of jaundice in neonates in Nigeria. Two cases of neonatal hypothyroidism was noted. Scott *et al*. (2004) and Najati *et al*. (2010) concluded in their study that hypothyroidism led to neonatal hyperbilirubinemia. ABO incompatibility and congenital biliary atresia also contributed as etiologies of hyperbilirubinemia in neonates. Careful education about breast feeding and monitoring of mothers as well as assessment of newborns for the risk of developing hyperbilirubinemia can aid in preventing neonatal jaundice. Treatment is based on total serum bilirubin concentration 6 hourly during phototherapy and exchange transfusion.

Conclusion

The present study concludes that breast feeding is the most common cause of neonatal hyperbilirubinemia in GB Pant Hospital Kashmir, a tertiary care teaching hospital, followed by other causes like breast milk jaundice, jaundice due to prematurity, neonatal sepsis, neonatal hypothyroidism, Congenital biliary atresia and ABO incompatibility.

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