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Original Research Article

Etiological Profile of Neonatal Jaundice in Bettiah district of Bihar, India.

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Abstract:

Objectives: This present study was to evaluate the aetiological profile of neonatal jaundice in Muzaffarpur, Bihar, India.

Methods: Each baby was examined clinically and relevant investigations of jaundice were performed. Each baby delivered at hospital was carefully observed from birth onwards in day light, for appearance of jaundice and in the babies with dark complexion, digital pressure over forehead was used to detect the icterus.

Results: A toral of 200 neonatal jaundice cases were enrolled in this study. Among them, preterm and term cases were 109(54.5%) and 91(45.5%) respectively. 108(54%) cases were males and 92(46%) were females. According to gestational age, 109(54.5%) cases were pre-term and 91(47.5%) cases were term baby. Exaggerated physiological jaundice 60(30%) was the most common etiology of neonatal jaundice. ABO incompatibility 56(28%) was the second most common etiology of neonatal jaundice. Others etiological factors were idiopathic 23(11.5%), Rh-incompatibility 21(10.05%), cephalhematoma 19(9.5%) and septicaemia 10(5%). Day of onset of jaundice was maximum in post-natal age of 24-72 hours. 58(29%) cases had 72 hours to 2 weeks.

Conclusions: Neonatal jaundice should be identified and assessed in babies within 24-72 hours after birth, especially in preterm and low birth weight babies to avoid life threatening and disabling complication of kernicterus. Parental counselling and monitoring of baby is the most important from the management of neonatal jaundice for prevention from the incidence of progression to severe hyperbilirubinemia and complications.

Keywords: Neonatal jaundice, gestational age, Onset of jaundice

Introduction

Neonatal jaundice is one of the most common causes of hospitalization of neonates in the first month after birth. In most cases, neonatal jaundice is transient and usually resolving at the end of the first week after birth. But when severe hyperbilirubinemia is present, there is a potential risk for acute bilirubin encephalopathy and kernicterus [1]. Neonatal hyperbilirubinemia is a common problem in neonates with an incidence of about 60% in term babies and 80% in preterm babies [2]. Jaundice refers to the yellowish discoloration of the skin and sclera of new born babies that result from accumulation of bilirubin in the skin and mucus membranes. Clinically it becomes apparent when the serum bilirubin exceeds

7 mg/dl in neonates and >2 mg/dl in adults [3].

Neonatal hyperbilirubinemia occurs due to a variety of factors. It may be physiological or pathological. Neonatal hyperbilirubinemia is a common condition requiring inpatient treatment, as well as an important reason for readmission to hospital [4]. It occurs in 70–80% of the neonates, more commonly in preterms [5]. Although, only 5–10% of the new born need to be treated due to pathological hyperbilirubinemia, the threat of neurologic damage always remains, especially with very high bilirubin level, in presence of certain risk factors and in cases where management remains inappropriate [6].

Although, up to 60 percent of term new-borns have clinical jaundice in the first week of life, few have significant underlying disease.3 Bilirubin is not a bad molecule in itself but bilirubin, like uric acid, is an important antioxidant circulating in blood of new-born. However, very high bilirubin levels might be toxic for central nervous system development and can cause behavioural and neurological impairment (neurotoxicity or Kernicterus) in new-borns. 5-10% of new-borns, who developed jaundice, required the management of hyperbilirubinemia [7]. Objectives of our study was to evaluate the aetiological profile of neonatal jaundice in Bettiah district of Bihar, India.

Material & methods

This present study was conducted in Department of Pediatrics, Government Medical College, Bettiah, Bihar, India during a period from October 2021 to May 2022.

Attendants of entire subject signed an informed consent approved by institutional ethical committee of Government Medical College, Bettiah was sought.

Inclusion criteria: 200 neonates with clinically identified Neonatal jaundice were enrolled in this study.

Exclusion criteria: Children more than 28 days of age and neonates admitted in paediatric surgical unit were excluded from this study.

Procedures: Jaundice was assessed by clinical examinations and confirmed by biochemical methods. Pre-test proforma was filled to record detailed history, clinical findings and investigations in each baby with hyperbilirubinemia. Each baby delivered at hospital was carefully observed from birth onwards in day light, for appearance of jaundice and in the babies with dark complexion, digital pressure over forehead was used to detect the icterus. In addition, babies coming from peripheries were examined thoroughly clinically. And detailed investigations were performed to detect the cause of jaundice. Other investigations were performed depending upon the clinical presentation and the report of initial investigations.

Statistical Analysis

Data was analysed by using latest version of SPPS software. All data was tabulated and percentages were calculated. Mean ± standard deviation was observed.

Observations

A toral of 200 neonatal jaundice cases were enrolled in this study. Among them, preterm and term cases were 109(54.5%) and 91(45.5%) respectively. 108(54%) cases were males and 92(46%) were females.

Table 1: Distribution of the patients according to gestational age.

Gestational age	No. of patients	% of patients
Preterm	109	54.5%
Term	91	45.5%
Total	200	100%

Day of onset of jaundice was maximum in post-natal age of 24-72 hours. 58(29%) cases had 72 hours to 2 weeks.

Table 2: Onset of jaundice

Days of onset	No. of patients	Percentage
<24 hours	14	7%
24-72 Ours	122	61%
72 hours to 2 weeks	58	29%
>2 weeks	6	3%
Total	200	100%

Exaggerated physiological jaundice (60(30%) was the most common etiology of neonatal jaundice.

Table 3: Aetiology according to gestational age.

Etiology	Pre-term	Term	Total
ABO incompatibility	35(60.71%)	21(39.28%)	56(28%)
Exaggerated physiological jaundice	28(45%)	32(55%)	60(30%)
Rh incompatibility	14(65%)	7(35%)	21(10.5%)
Idiopathic	15(63.64%)	8(36.36%)	23(11.5%)
Hypothyroidism	0	0	00
G6PD	0	1(100%)	01(0.5%)
Galactosemia	1(50%)	1(50%)	02(1%)
BMJ	1(50%)	1(50%)	02(1%)
Intrauterine infection	2(33.33%)	4(66.67%)	06(3%)
Septicaemia	7(70%)	2(30%)	9(4.5%)
Cephalhematoma	6(28.57%)	14(71.43%)	20(10%)
Total	109(54.5%)	91(47.5%)	200(100%)

In this present study, out of 200 cases. According to gestational age, 109(54.5%) cases were pre-term and 91(47.5%) cases were term baby. Exaggerated physiological jaundice 60(30%) was the most common etiology of neonatal jaundice. ABO incompatibility 56(28%) was the second most common etiology of neonatal jaundice. Others etiological factors were idiopathic 23(11.5%), Rh-incompatibility 21(10.05%), cephalhematoma 19(9.5%) and septicaemia 10(5%).

Table 4: Weight wise distribution of neonatal jaundice.

Causes	**Weight (Kgs)**				**Total**
	<1.5	1.5-2.49	2.5-3.49	>3.5	
ABO incompatibility	12	14	19	11	56(28%)
Exaggerated physiological jaundice	20	17	12	10	59(29.5%)
Rh incompatibility	10	5	3	2	20(10%)
Idiopathic	07	9	03	2	21(10.5%)
Hypothyroidism	0	0	0	0	00
G6PD	0	1	0	0	01(0.5%)
Galactosemia	0	0	2	0	02(1%)
BMJ	0	0	1	1	02(1%)
Intrauterine infection	1	3	5	0	09(4.5%)
Septicaemia	2	2	4	1	9(4.5%)
Cephalhematoma	4	6	6	4	20(10%)
Total	56(28%)	57(28.5%)	56(28%)	31(15.5%)	200(100%)

There was much difference in the different age groups in which Low Birth Weight Babies (LBW) contributes to 57(28.5%), and VLBW babies contributes to 56(28%) of the total cases. 56(28%) cases had birth weight 2.5 to 3.49 kgs and 31(15.5%) cases had birth weight >3.5 kgs.

Table 5: Etiology of hemolytic jaundice

ABO incompatibility MBG (O+), BBG (A+/B+)		Percentage
OA Combination	21	37.5%
OB Combination	35	62.5%
Total	56(28%)	100%
Rh incompatibility	11	55%
G6PD	1	100%
Total		

There were various causes of hemolytic jaundice, ABO incompatibility consists of 56 cases, Rh incompatibility of 11 cases and G6PD of 1 cases. Out of total 68 cases, 24 cases develop jaundice in the first 24 hours. Acute bilirubin Encephalopathy (ABE) was seen in 9 cases, of which maximum in ABO Incompatibility in which 2 had associated with septicemia. Rh isoimmunization was seen in 3 cases, out of which 1 had associated septicemia.

Table 6: Etiology of Acute Bilirubin Encephalopathy (ABE)

Etiology	Number	Percentage
ABO Incompatibility	4	
ABO + Septicemia	2	
Rh Incompatibility	2	
Rh + Septicemia	1	
Total	9	

Septicemia was a potentiating factor in causing severe neonatal hyperbilirubinemia in patients with blood group incompatibility. There was a male preponderance and majority of the patients were preterm babies (n=5) and full term (n=4).

Table 7: Peak bilirubin levels in causing Acute Bilirubin Encephalopathy (ABE).

Gestation	Serum bilirubin
Pre-term	17.4±2.9
Term	23.6±5.1

Table 8: Hyperbilirubinemia

Hyperbilirubinemia	No. of cases	Percentage
Unconjugated	174	87%
Conjugated	26	13%
Total	200	100%

Peak serum bilirubin levels causing Acute Bilirubin Encephalopathy (ABE) was 17.4 ± 2.9mg/dl with in pre-term babies. In full term babies it was 23.6±5.1 mg/dl. Conjugated hyperbilirubinemia was seen in 26(13%) of cases.

Discussions

Neonatal hyperbilirubinemia is a common condition requiring inpatient treatment and monitoring, and many times requires readmission to hospital. Estimated

incidence of jaundice in neonates is 60% to 84% of late term and term infants [8].

In this present study, we were included a total of 200 neonates with less than 28 days of life. Among them preterm and term cases were 109 (54.5%) and 91 (45.5%) respectively. 108 (54%) cases were males and 92 (46%) were females.

This is Comparable to study done by Effiong et al, Nigeria, 1972, Narang et al, 1996 India and Korejo et al, 2007 Karachi [9,10]. A probable explanation may be due to social bias, males being more cared for, and promptly brought to medical attention.

Bhutani et al in their study found out that prematurity was a significant risk factor for hyperbilirubinemia and is known to be a basis for increased biologic vulnerability to risk of bilirubin induced neurotoxicity [11]. Bajpai et al, Indian Journal of pediatrics, had shown an incidence of 14% as physiologic jaundice with prematurity [12]. Onyearugha et al, prematurity was the second leading cause of NNJ both in inborn and outborn babies [13]. Singhal et al, had given an incidence of 16.7% (Prematurity) as a cause of neonatal jaundice probably more because of physiological handicaps in premature, LBW babies [14]. Hussain et al, Karachi, too had shown that prematurity and LBW was an important risk factor for development of severe hyperbiliru-binemia [15]. Preterm newborns are prone to developing jaundice due to immaturity of their bilirubin conjugating system, higher rate of hemolysis, increased enterohepatic circulation and decreased caloric intake.

In the present study, jaundice was detected maximum on Live hour (24-72) which consist of 122 cases (61%) followed by 72 hours to 14 days which contributes to 58 cases (29%). The onset of jaundice during live hour 24 was 14 cases (7%) which is always an indicative of pathological jaundice. After 2 weeks of post-natal age, the number of cases decreased significantly to 1 case (0.5%). All three case was cholestatic jaundice. The results in our study are similar to work done by Anand et al, where the highest incidence of jaundice was on 3rd (45%) post-natal day followed by 4th day (35.5%). This may be because of increased bilirubin production due to increased RBC volume per kilogram and decreased RBC survival, increase ineffective erythropoiesis and increased turnover of nonhemoglobin heme proteins [16].

In this study, all the different etiologies were much higher in the preterm delivered babies except in physiological jaundice. Preterm babies contribute to 109(54.5%) of the cases and in term babies 91(45.5%) cases. ABO, Rh incompatibility and septicemia were more common in the preterm babies. There was much difference in the different age groups in which Low Birth Weight Babies (LBW) contributes to 57(28.5%), and VLBW babies contributes to 56(28%) of the total cases. Rh incompatibility and ABO incompatibility contributes to highest percentage in the LBW and VLBW babies. Similar study done Narang et al, the incidence of significant NNJ was 82.9% at gestational age > 28 weeks reduced where to 56.9% at gestational age of 35-36 weeks [17].

In this study, it was observed that exaggerated physiological jaundice was highest which accounts for 60 cases (30%). In the study by Bahl et al, had shown that physiological jaundice contributed to highest 63.8% incidence [18]. It was comparatively higher as compared to our study. Singhal et al, (16.7%) and Merchant et al, (25.3%) too had reported highest incidence of physiological jaundice in their studies [14,19]. In the study by Bedowra et al, Bangladesh (n=60), physiological jaundice contributes to 53.3% as the most common cause in their study [20]. It was comparatively higher too as compared to our study. These higher incidences of physiologic jaundice may be due to increased enterohepatic circulation, decreased intestinal bacteria and decreased gut motility with poor evacuation of

bilirubin-laden meconium, defective up take of bilirubin from plasma caused by decreased ligandin and binding of ligandin by other anions and defective conjugation due to decreased UGT activity and decreased hepatic excretion of bilirubin.

In this present study, we were observed that ABO incompatibility was 56 (28%). In the study conducted by Shao-wen et al, ABO incompatibility was 18.3% and Rh incompatibility was 2.4% G6PD 20% which is higher [21]. Farhad et al and Joshi et al in their studies found that ABO was 38.1% and 28.8% respectively, in which the incidences were similar to our study. In the study conducted by Sgro M et al concluded that ABO incompatibility 51.6% was the most common cause, which was much higher than in our studies [22,23].

In our study, there was various causes of hemolytic jaundice, ABO incompatibility consists of 56 cases, Rh incompatibility of 11 cases and G6PD of 1 cases. ABO incompatibility (56 cases) was found to be the most common cause, followed by Rh incompatibility (11 cases) and G6PD (1 case). The results are consistent with Sgro M et al, in which ABO (51.6%) incompatibility was the most common cause followed by G6PD (21.5%) and other antibody incompatibility (12%). Farhad et al, too had consistent results with ABO (38.1%), Rh (16.1%) and G6PD (3.4%) [22,23].

In this present study, exaggerated physiological jaundice was highest which accounts for 60 cases (30%).

In this present study, hyperbilirubinemia was occurred due to septicemia 10(5%) cases and G6PD deficiency accounted for 1(0.5%) case. The incidence is very low as compared to that of ABO and Rh incompatibility due to limited laboratory diagnostic facilities and earlier discharge of the newborns and lack of proper follow up. Galactosemia was contributed to 2(1%) cases. Cephalhematoma (enclosed hemorrhage) is a benign condition of the newborn. Which was found in 20(10%) cases following vacuum and forceps deliveries. Herman W. Hyatt et al, too had mentioned about risk of hyperbilirubinemia in the newborn probably related to cephalhematoma [24].

In our study, onset of jaundice was maximum after the 3rd post-natal age. It was more in full term delivered, AGA babies (32%), and 16% in preterm babies in the study. Anil Narang et al, too had shown 4% incidence in their study [25].

In this present study, Acute Bilirubin Encephalopathy (ABE), was seen in 9(4.5%) cases, of which maximum cases were in babies with ABO incompatibility (6 cases) in which 2 cases had associated with septicemia. Rh isoimmunisation was seen in 3 cases, out of which 1 had associated septicemia. Thus, it was found that septicemia was a potentiating factor in causing severe neonatal hyperbilirubinemia in patients with blood group incompatibility. [26]

Hence, Clinicians need a systematic approach to identify the infants who may develop severe hyperbilirubinemia and keep them in follow up. Slightly higher incidence of septicaemia than the other causes in our study may reflect the lacunae in maintaining asepsis at natal and postnatal practices in developing countries.

Conclusions

This present study concluded that the neonatal jaundice should be identified and assessed in babies within 24-72 hours after birth, especially in preterm and low birth weight babies to avoid life threatening and disabling complication of kernicterus. Parental counselling and monitoring of baby is the most important from the management of neonatal jaundice for prevention from the incidence of progression to severe hyperbilirubinemia and complications.

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