Print ISSN: 2958-3675 | Online ISSN: 2958-3683
International Journal of Medical and Pharmaceutical Research
Journal Home Page: https://ijmpr.in/
Vol-1 Iss-1, JUL-DEC 2020
Original Article Open Access

Clinical Profile and Etiological Factors of Neonatal Jaundice from A Rural Area of Bangladesh

Dr. Nityananda Baruri[1]; Dr. Goutam Kumar Lasker[2]; Dr. Manoj Kumar Malakar[3]; Dr. Gopal Chandra Roy[4]

[1]Assistant Professor, Department of Pediatric, Khulna Medical College Hospital, Khulna, Bangladesh.
[2]Registrar, Department of Pediatric, Khulna Medical College Hospital, Khulna, Bangladesh
[3]AssistantRegistrar Child Health, Khulna Medical College Hospital, Khulna, Bangladesh.
[4]OSD, DGH, Attachment in Medical Officer, Department of Pediatric, Dhaka Medical College Hospital, Dhaka, Bangladesh

ABSTRACT

Background: Neonatal jaundice, characterized by the yellowing of a newborn's skin and eyes due to elevated bilirubin levels, is a common condition affecting infants worldwide. Understanding the clinical profile and etiological factors associated with neonatal jaundice is essential for effective management and prevention strategies. Neonatal jaundice is a common cause of newborn hospital admission. The risk factors, characteristics and outcomes related to neonatal jaundice in Bangladesh has have been studied so far.

Aim of the study: The study aimed to study the clinical profile and underlying etiological factors leading to neonatal jaundice in this rural Bangladesh.

Methods: This prospective observational study was conducted in the neonatology ward at Khulna Medical College and Hospital, Khulna, Bangladesh. The study was conducted from January 2020 to December 2020. A total of 98 neonates were admitted to our post-natal ward during the specified period.

Result: A total of 98 participants were enrolled and analyzed. The gestational age distribution revealed that 88.78% of the study population was aged more than 37 weeks, 8.16% were in the 34-36-week range and 3.06% were in the 30-34-week range. Among the babies, 63% were male, and 37% were female. The distribution of birth weights showed that 3.06% weighed 1000-1500g, 5.10% weighed 1501-2000g, 8.16% weighed 2001-2500g, 58.16% weighed 2501-3000g, and 25.51% weighed more than 3000g. The etiology of neonatal jaundice in the study revealed that 43.88% had physiological jaundice, 24.49% had ABO incompatibility, and 8.16% had Rh incompatibility or idiopathy.

Conclusion: This study concludes that physiological jaundice is our hospital's most common cause of neonatal jaundice. This is followed by ABO incompatibility, sepsis, Rh incompatibility and idiopathic cases.

Keywords: *Etiology, Neonatal jaundice, Clinical Profile, Prevalence*

***Corresponding Author**
Dr. Nityananda Baruri
Assistant Professor (MBBS, DCH), Department of Pediatric, Khulna Medical College Hospital, Khulna, Bangladesh

INTRODUCTION

Neonatal jaundice is estimated to occur in 60% of term newborns in the first week of life, and < 2% reach total serum bilirubin (TSB) levels of 20 mg/ dL [1,2]. In rare instances, the TSB reaches levels that can cause kernicterus, a condition characterized by bilirubin staining of neurons and neuronal necrosis involving the brain's basal ganglia primarily and manifested in athetoid cerebral palsy, hearing loss, dental dysplasia, and paralysis of upward gaze [3]. Risk factors recognized to be associated with severe hyperbilirubinemia in newborns have jaundice in the first 24 hours of life. Glucose-6-phosphate dehydrogenase (G6PD) deficiency, ABO incompatibility, low birth weight, and sepsis are the common causes of neonatal jaundice in Asian and Southeast Asian regions. However, there is a group of babies whose cause of neonatal. Jaundice has yet to be found. Genetic factors and unknown environmental factors may also play a role in the prevalence of neonatal jaundice [4]. Glucose 6-phosphate dehydrogenase (G6PD) deficiency is the most important disease of the hexose monophosphate pathway. G6PD is a linked recessive disease where enzyme deficiency causes clinical manifestations ranging from neonatal jaundice to chronic nonspherocytic anemia and drug-induced hemolysis [5]. Neonatal jaundice is a relatively common cause of morbidity in Bangladesh. However, little information is available on patterns of neonatal jaundice. The unique Care Baby Unit (SCABU) in BIRDEM is a neonatal intensive care unit (ICU) that has been running for the last 13 years, where seriously ill babies are referred to. One study in SCABU observed that the incidence of neonatal jaundice was 23.5%, and among them, about 17% required exchange transfusion

[6]. Identifying infants at risk of developing severe hyperbilirubinemia and early intervention have reduced morbidity and mortality associated with bilirubin encephalopathy. The study aimed to study the clinical profile and the underlying aetiological factors leading to neonatal jaundice in this rural Bangladesh.

METHODOLOGY & MATERIALS

This prospective observational study was conducted in the neonatology ward at Khulna Medical College and Hospital, Khulna, Bangladesh. The study was conducted from January 2020 to December 2020. A total of 98 neonates were admitted to our post-natal ward during the specified period. Out of these 98 newborns were jaundiced (Serum bilirubin >10 mg/dl). So a total of 98 cases were enrolled for the study. Babies attending outpatient departments only were excluded from the study. Informed consent was obtained from the parents.

- **Inclusion criteria**:

Neonates with jaundice were admitted to the neonatology ward during the study period, with serum bilirubin of more than 10mg/dL.

- **Exclusion criteria**:

Neonates with jaundice are not admitted to the neonatology ward, only attending the outpatient department. Neonates with jaundice opted for discharge against medical advice. Parents are not willing to participate in this study.

Clinical methods ascertained jaundice. This was confirmed with the help of biochemical tests. Serum bilirubin was estimated by the Van den Bergh method. A detailed history was taken. A thorough physical examination was done, and the relevant investigations were carried out. General data were documented, including age, birth weight, age at detecting jaundice, breastfeeding status, and family history. Further investigations were not carried out on babies with physiological jaundice. Blood grouping and Rh typing of baby and mother were done. Cord blood bilirubin and haemoglobin, direct Coomb's test (DCT), and bilirubin monitoring were done whenever there was a setting for Rh incompatibility. In case of ABO incompatibility, DCT was done, and bilirubin was monitored. Other investigations like haemoglobin level, peripheral smear, and reticulocyte count were done. If these tests showed features of hemolysis and no blood group incompatibility, a G6PD assay, a sickling test, haemoglobin electrophoresis, and an osmotic fragility test were performed where appropriate. Neonates suspected of having sepsis were investigated by complete blood count, septic screen, and blood and urine cultures.All data were presented in a suitable table or graph according to their affinity. A description of each table and graph was given to understand them clearly. All statistical analysis was performed using the statistical package for the social science (SPSS) program and Windows. Continuous parameters were expressed as mean±SD and categorical parameters as frequency and percentage.

RESULT

This is a prospective study; a total of 98 were enrolled and analyzed in this study. The table shows the gestational age distribution of the study; most, 88.78% of the study population were aged more than 37 weeks, 8(8.16%) patients were from the age group 34-36 weeks, and only 3.06% of them were from the age group 30-34 weeks. 63% of babies were male and 37% female (Figure 1). The table shows the distribution of birth weights and corresponding frequencies and percentages. Three births weighed 1000-1500g, accounting for 3.06% of the total. Five births were in the 1501-2000g range, representing 5.10% of the total. The 2001-2500g range had eight births, accounting for 8.16%. The largest category, with 57 births, was in the 2501-3000g range, making up 58.16%. Lastly, there were 25 births with weights more fabulous than 3000g, representing 25.51% of the total. Table 3 shows the neonatal jaundice etiology of the study; most 43.88% of the babies had physiological jaundice, 24(24.49%) babies had ABO incompatibility, and each Rh incompatibility, Idiopathy had the same percentage of 8.16%.

Table 1: Distribution according to gestational age (N=98).

Gestational age (Weeks)	Frequency	Percentage
>37	87	88.78
34-36	8	8.16
30-34	3	3.06
Total	**98**	**100.00**

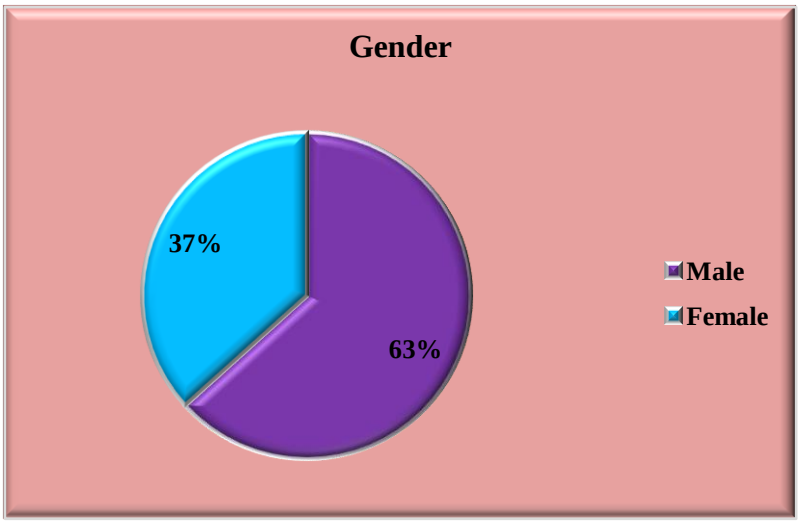

Figure 1: Gender distribution of the babies (N=98).

Table 2: Distribution based on birth weight (N=98).

Birth weight (gm)	Frequency	Percentage
1000-1500	3	3.06
1501-2000	5	5.10
2001-2500	8	8.16
2501-3000	57	58.16
>3000	25	25.51
Total	**98**	**100.00**

Table 3: Neonatal jaundice etiology (N=98).

Etiology	Number	Percentage
Physiological Jaundice	43	43.88
ABO incompatibility	24	24.49
Sepsis	3	3.06
Rh incompatibility	8	8.16
Idiopathy	8	8.16
Prematurity	3	3.06
Cephalhematoma	3	3.06
Breastfeeding	3	3.06
HaemolyticAnaemia	3	3.06
G6PD deficiency	0	0.00
Hypothyroidism	0	0.00
Total	**98**	**100.00**

DISCUSSION

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 resolves at the end of the first week after birth. However, when severe hyperbilirubinemia is present, there is a potential risk for acute bilirubin encephalopathy and kernicterus. This can lead to death in the first months, and infants still alive often suffer from mental retardation, movement, and balance disorders, seizures, hearing loss at high frequencies, and speech impairment. So, timely diagnosis and treatment of neonatal jaundice are crucial to prevent further complications. In this study, most babies with neonatal jaundice were of term gestation. Only 10% of babies studied were preterm. Bhutani et al. and Singhal et al. found a higher percentage of premature babies in their studies [7, 8]. Of 98 neonates studied, 63% were males, and only 37% were females. A higher incidence of significant hyperbilirubinemia in male babies as compared to female babies was found in various other studies [9]. Physiological jaundice was noted in 35% of babies in our study, which is the most common group. Normally some icterus appears on the second to the third day, reaching its maximum on the second to fourth day and decreasing on the fifth to seventh days, mainly due to liver enzymes not evolving enough. This jaundice is called physiologic jaundice. Various factors such as maternal diabetes, race, premature infant, medication use of the mother, male gender, cephalohaematoma, breastfeeding, weight loss, and delayed stools in the baby may be correlated with physiologic jaundice [10]. Since most of these are normal physiological findings, it also increases the overall contribution of

physiological jaundice in cases of neonatal jaundice. Bahl et al. reported that physiological jaundice contributed to the majority (63.8%) of cases studied [11]. This was followed by ABO incompatibility as the next leading cause of neonatal jaundice (21.8%). A Canadian study by Sgro et al. reported ABO incompatibility (51.6%) as the commonest cause of hyperbilirubinemia, similar to us, followed by G-6PD deficiency (20%), hereditary spherocytosis (7%), and sepsis/urinary tract infection (3%) [12]. Immune-mediated hemolysis and hyperbilirubinemia are usually related to Rh D and ABO incompatibilities and rarely due to other minor blood group incompatibilities, such as anti-C, anti-E, and anti-Kell [13]. ABO hemolytic disease generally occurs in infants of blood group A or B born to group O mothers. Although an ABO incompatibility situation exists in about 15% of pregnancies, only a fraction of infants born in this context develop significant hyperbilirubinemia, and often it is difficult to predict, even if not strongly predicted by Coomb's test [14]. The diagnosis of ABO hemolytic disease, as opposed to ABO incompatibility, is generally reserved for those with a positive Coomb's test and clinical jaundice within the first 12–24 h of life. Neonates having O-B incompatibility have been reported to develop hyperbilirubinemia within 24 h of life, more frequently than O-A incompatibility; however, the present study did not find such a difference [15]. Sepsis is known to cause hemolysis and hyperbilirubinemia, probably by increasing oxidative stress and damaging red blood cells susceptible to cell injury [16]. Incipient sepsis/ bacteremia has been reported as a rare cause of hyperbilirubinemia in the developed world; however, it accounted for 21% of our cases, reflecting poor perinatal care [17]. Most of our sepsis cases were culture-negative, probably because they were referred cases and had received prior antibiotics. The etiology of hyperbilirubinemia could not be ascertained in …% of our neonates. Many authors have also been unable to establish the etiology of hyperbilirubinemia in more than half of the cases in their series [18, 19]. It emphasizes the need for more thorough investigations to determine the cause and further studies to determine the role of environmental factors and genetic interactions, which may exaggerate hyperbilirubinemia when associated with other high-risk conditions [12]. Sepsis constituted 11% of the cases studied. This is in concordance with earlier studies which showed a similar trend. Sepsis was found to cause jaundice in 8% of neonates by Merchant et al. and 11.6% by Verma et al. [20, 21]. Rh incompatibilities were responsible for 8.16% of cases in this study. Bajpai PC et al. reported an incidence of 1.6% for Rh incompatibility, while Verma et al. found that it contributes to 9.8% of the cases [21, 22]. One case of haemolyticanaemia (3.06%) was later diagnosed as hereditary spherocytosis. This baby's mother suffered from hereditary spherocytosis and had undergone a splenectomy. We did not get any case of G6PD deficiency in our study. This could be a reflection of regional variation in the etiology of neonatal jaundice, and G6PD deficiency seems to be an uncommon problem in our area. G6PD deficiency was reported in 2.6% of neonates by Merchant et al. and 3.4% by Narang et al. [20,23]. Although the evidence of hemolysis and positive direct Coomb's test indicates significant immune-mediated hemolytic disease, they may not be helpful because of their poor sensitivity and specificity. Thus, the diagnosis of symptomatic immune-mediated hemolytic disease leading to hyperbilirubinemia should be considered in the context of blood group incompatibility that may be accompanied by a positive, direct Comb's test and evidence of hemolysis.

Limitations of the study:

Every hospital-based study has some limitations and the present study undertaken is no exception to this fact. The limitations of the present study are mentioned. Therefore, the results of the present study may not be representative of the whole of the country or the world at large. The number of patients included in the present study was less in comparison to other studies. Because the trial was short, it was difficult to remark on complications and mortality.

CONCLUSION AND RECOMMENDATIONS

Physiological jaundice is the leading cause of neonatal jaundice in our hospital, followed by ABO incompatibility, sepsis, Rh incompatibility, and idiopathic cases. Cephalhematoma, breastfeeding jaundice, and hemolytic anemia are less common causes. Parental counselling and baby monitoring are crucial for managing neonatal jaundice. While severe hyperbilirubinemia is rare, its associated complications are dangerous.

Funding: *No funding sources*
Conflict of interest: *None declared*

REFERENCES

1. Provisional Committee on Quality Improvement, Subcommittee on Hyperbilirubinemia. Practice parameter: management of hyperbilirubinemia in the healthy term newborn. Pediatrics. 1994 Oct;94(4):558-65.
2. Newman TB, Escobar GJ, Gonzales VM, Armstrong MA, Gardner MN, Folck BF. Frequency of neonatal bilirubin testing and hyperbilirubinemia in a large health maintenance organization. Paediatrics. 1999 Nov 1;104(Supplement_6):1198-203.
3. Maisels MJ. Jaundice: In Avery GB, Fletcher MA, MacDonald (ed). Neonatology: Pathology and management of the newborn.
4. Ho NK. Neonatal jaundice in Asia. Baillière's clinical haematology. 1992 Jan 1;5(1):131-42.
5. Srivatsa A, Bharti B, Singhi SC. Does nimesulide induce haemolysis in glucose-6-phosphate dehydrogenase deficiency? Acta paediatrica (Oslo, Norway: 1992). 2003 May;92(5):637-8.

6. Hasan HS, Fateha US, Abdullah AH, Azad K. Neonatal jaundice. InExperience at BIRDEM proceedings of the 4th national conference and scientific seminar of Bangladesh neonatal forum 2004.
7. Bhutani VK. Evidence-based issues regarding neonatal hyperbilirubinemia. Paediatrics review. 2005;114:130-53.
8. Singhal PK, Singh M, Paul VK, Deorari AK, Ghorpade MG. Spectrum of neonatal hyperbilirubinemia: an analysis of 454 cases. Indian pediatrics. 1992 Mar 1;29(3):319-25.
9. Effiong CE, Aimaku VE, Bienzle U, Oyedeji GA, Ikpe DE. Neonatal jaundice in Ibadan. Incidence and etiologic factors in babies born in hospital. Journal of the National Medical Association. 1975 May;67(3):208.
10. Liu AH, Covar RA, Spahn JD, Leung DY, Kliegman RM, Behrman RE, Jenson HB, Stanton B. Nelson textbook of pediatrics. Childhood Asthma, Elsevier, New Delhi. 2004
11. Bahl LA, Sharma RA, Sharma JA. Etiology of neonatal jaundice at Shimla. Indian pediatrics. 1994 Oct;31(10):1275-8.
12. Sgro M, Campbell D, Shah V. Incidence and causes of severe neonatal hyperbilirubinemia in Canada. Cmaj. 2006 Sep 12;175(6):587-90.
13. Annagür A, Altunhan H, Konak M, Hasan KO, Rahmi ÖR. Role of subgroup incompatibility in newborn jaundice requiring exchange transfusion. European Journal of General Medicine. 2014 Jun 28;11(2):2-6.
14. Ip S, Chung M, Kulig J, O'Brien R, Sege R, Glicken S, Maisels MJ, Lau J, Subcommittee on Hyperbilirubinemia. An evidence-based review of important issues concerning neonatal hyperbilirubinemia. Pediatrics. 2004 Jul;114(1):e130-53.
15. Kaplan M, Hammerman C, Vreman HJ, Wong RJ, Stevenson DK. Hemolysis and hyperbilirubinemia in antiglobulin positive, direct ABO blood group heterospecific neonates. The Journal of pediatrics. 2010 Nov 1;157(5):772-7.
16. Kaplan M, Eidelman AI. Post factum imposition of exchange transfusion criteria: in defence of neonatologists. Acta Paediatrica. 2011 Apr;100(4):479-81.
17. Maisels MJ, Kring E. Risk of sepsis in newborns with severe hyperbilirubinemia. Pediatrics. 1992 Nov;90(5):741-3.
18. Sgro M, Campbell D, Shah V. Incidence and causes of severe neonatal hyperbilirubinemia in Canada. Cmaj. 2006 Sep 12;175(6):587-90.
19. Ipek IO, Bozaykut A. Clinically significant neonatal hyperbilirubinemia: an analysis of 646 cases in Istanbul. Journal of tropical pediatrics. 2008 Jan 21;54(3):211-3.
20. Merchant RH, Merchant SM, Babar ST. A study of 75 cases of neonatal jaundice. Indian pediatr. 1975.
21. Verma M, Chhatwal J, Singh D. Neonatal hyperbilirubinemia. The Indian Journal of Pediatrics. 1988 Nov;55(6):899-904
22. Bajpai PC, Misra PK, Agarwal M, Engineer AD. An etiological study of neonatal hyperbilirubinaemia. The Indian Journal of Pediatrics. 1971 Nov;38:424-9.
23. Narang A, Gathwala G, Kumar P. Neonatal jaundice: an analysis of 551 cases. Indian pediatrics. 1997 May 1;34:429-32.