IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 10 Ver. 2 (October. 2018), PP 21-23
www.iosrjournals.org

A comparative study of peak total serum bilirubin level in neonates with ABO incompatibility, Rh incompatibility and G6PD deficiency.

Dr. Mrinalini Das[1], Dr. Ashok Singh[2]
1(Associate Professor, Department of Pediatrics, GMCH, Guwahati, India)
2(Junior Resident, Department of Pediatrics, GMCH,Guwahati, India)
Corresponding Author:Dr.Ashok Singh

Abstract: Introduction: *Maternal-fetal ABO blood group incompatibility, in which the mother has blood group O and the newborn has blood group A or B, occurs in 15-20% of all pregnancies. The hemolytic process results from maternal anti-A or anti-B immunoglobulin G (IgG) antibodies crossing the placenta and attaching to the appropriate antigens on the neonatal red cells. Rh incompatibility develops when an Rh negative mother has a baby with an Rh positive father. It is characterized by the presence of IgG antibodies in maternal circulation, which causes hemolysis in the fetus by crossing the placenta and sensitizing red cells for destruction by macrophages in the fetal spleen with consequent hyperbilirubinemia. Glucose-6-PhosphateDehydrogenase(G6PD) deficiency is an inherited deficiencythat may cause neonatal jaundice, as has been found in several countries and different ethnic groups. G6PD isan enzyme essential for basic cellular functions including protection of red cell proteins from oxidative damage. G6PD deficiency is the most common red cell enzyme abnormality associated with hemolysis as well as withneonatal jaundice and also associated with kernicterus and even death. It is a genetically inherited sex-linkedabnormality.* ***Methods:*** *The study was done on 1619 clinically icteric babies without other risk factors forneonatal jaundice sepsis, prematurity, low birth weight, polycythemia, cephalhematoma,infant of diabetic mother and gastrointestinal obstruction etc., admitted in Neonatal Intensive Care Unit of GMCH, Guwahati over a period of 1 year.Investigations included direct and indirect serum bilirubin levels, blood group of mother and baby, directcoomb's test, haemoglobin, blood smear examination, reticulocyte count, and G6PD status. Data was collectedand adequate intervention was done depending on indication. Data was analysed by software SPSS version 20.* ***Results****-In the neonates with ABO incompatibility (272), mean peak TSB level was 20.5±4.6mg/dl., In the neonates with Rh incompatibility (158), peak TSB was21.9±6.5mg/dl. In G6PD deficient neonates (146), mean peak TSB level was 23.9 ± 4.1 mg/dl. The P-value calculated is < 0.0001 which is extremely significant.* ***Conclusions:*** *The prevalence of severe NHB with ABO incompatibility, Rh incompatibility and G6PD deficiency among neonates in North East region ishigh and babies with these factors have higher chances of severe hyperbilirubinemia anddeveloping complications like kernicterus and poor neurodevelopmental outcome. Therefore, screening ofnewbornsfor ABO incompatibility, Rh incompatibility and G6PD deficiency is needed to be done in all newborns, so that these babies can be identified andadequate intervention can be donetimely.*

Date of Submission: 24-09-2018 Date of acceptance: 10-10-2018

I. Introduction

Jaundice is a commonly reported condition in neonates.Jaundice typically results from the deposition of unconjugated bilirubin pigment in the skin and mucus membranes. Approximately 60% of term and 80% of preterm neonates become icteric in the first week of life. higher bilirubin levels are associated with neurological abnormalities, hearing loss, motor abnormalities.[1] Under certain circumstances, severe hyperbilirubinemia can cause complication, known as Kernicterus. Orth first described yellow staining of the brain, in 1875, later referred to by Schmorl as kernicterus.[2] Maternal-fetal ABO blood group incompatibility, in which the mother has blood group O and the newborn has blood group A or B, occurs in 15-20% of all pregnancies[3].Hemolytic disease develops in approximately 10% of such newborns and may be associated with clinically significant neonatal hyperbilirubinemia[4].The hemolytic process results from maternal anti-A or anti-B immunoglobulin G (IgG) antibodies crossing the placenta and attaching to the appropriate antigens on the neonatal red cells. Resultant heme catabolism causes an increased indirect bilirubin (IB) production, leading to neonatal jaundice.[5] Rh incompatibility develops when an Rh negative mother has a baby with an Rh positive father. It is characterized by the presence of IgG antibodies in

maternal circulation, which causes hemolysis in the fetus by crossing the placenta and sensitizing red cells for destruction by macrophages in the fetal spleen with consequent hyperbilirubinemia.[6] Dehydrogenase (G6PD) is an enzyme essential for basic cellular functions including protection of red cell proteins from oxidative damage. G6PD deficiency is the most common red cell enzyme abnormality associated with hemolysis as well as with neonatal jaundice and also associated with kernicterus and even death. Neonatal hyperbilirubinemia occurs in 2.5-6 % neonates in India[7] . It plays a protective role against malaria[8].One third of children with G6PD deficiency develop neonatal jaundice which when severe and if untreated could give rise to kernicterus, a well-known cause of death and neurodevelopmental handicap. G6PD deficiency is present in all the tribal groups studied from North-East India.[9]

Aim of the study-To compare the peak total serum bilirubin level in neonates with ABO incompatibility, Rh incompatibility and G6PD deficiency in a tertiary care hospital inNorth-East India.

II. Materials and Methods

The study was done in Neonatal intensive care unit, Gauhati Medical college and Hospital, Guwahati, Assam, India during the period of 1 year from July 2016 to June 2017**.** The study was approved by the institute's Ethical Committee. The study was a hospital based observational study. All the healthy term newborns that have completed 37 weeks of gestation, admitted for neonatal jaundice in Neonatal Intensive care Unit (NICU), GMCH, Guwahati, were included in the study. Babies with other factors causing neonatal jaundice like sepsis, prematurity, low birth weight, polycythemia, cephalhematoma,infant of diabetic mother and gastrointestinal obstruction etc. were excluded from the study. The babies with more than one risk factors simultaneously were also excluded. After takingnecessary precautions and asepsis, relevant laboratory investigations like complete blood count,blood group typing of the neonates and mothers, direct coomb's test, peripheral blood smear, reticulocyte count,sepsis screen, serum bilirubin level with fraction by appropriate standard methods and G6PD status was done by using (GBK-G6PD kit(ARKRAY Healthcare Pvt. Ltd.). Total serum bilirubin was estimated from clotted blood by automatic analyser(VITROS® 5600 integrated system from Ortho Clinical Diagnostics 1001 U.S. 202 Raritan, NJ 08869) usingintegrated reagent cartridge. Results were analysed by computer based statistical package for the social science SSPSsoftware version 20. P- values of less than 0.05 were considered statistically significant.

III. Results

A total of 1619 babies without other precipitating factors, were admitted for neonatalhyperbilirubinemia. In this study is was found that 272 neonates were admitted for ABO incompatibility,158 for Rh incompatibility and146 were identified as G6PD deficient neonates.

Fig.2 Causes of NHB

Factors	No. of Babies
ABO incompatibility	272
Rh incompatibility	158
G6PD deficiency	146
Others	1043
Total	1619

In the neonates with ABO incompatibility (272), mean peak TSB level was 20.5±4.6mg/dl., In the neonates with Rh incompatibility (158), peak TSB was21.9±6.5mg/dl. In G6PD deficient neonates (146), mean peak TSB level was 23.9 ± 4.1 mg/dl.

Fig.2 Mean peak TSB level in babies with ABO incompatibility, Rh incompatibility and G6PD deficiency.

Factors	Mean peak TSB (mg/dl)
ABO incompatibility	20.5±4.6 mg/dl
Rh incompatibility	21.9±6.5 mg/dl
G6PD deficiency	23.9 ± 4.1 mg/dl
Others	19.6 ± 3.4 mg/dl

IV. Discussion

Singh et. al.[10] in 2016 in a study in 2016 found peak TSB level of 22.7 ± 8.97mg/dl in babies with ABO incompatibility. SinemAkgül et. al.[11] in 2013 in a study found peak TSB 20.2±5.7 mg/dl in babies with ABO incompatibility. In another study done by Shao-Wen Cheng et. al.[12] in 2012, the peak TSB level in ABO incompatibility was 23.3±3.2 mg/dl. In the present study, the peak TSB in babies with ABO incompatibility was 20.5±4.6mg/dl.

In a study done by Yi-Hao Weng et. al.[13] in 2008, the peak TSB level in Rh incompatibility was 25.8 ± 3.5 mg/dl. In a study done bySaitALTIKAT,et. al.[14] in 2012 found the peak TSB level of 18.94 ± 4.88 mg/dl. In the present study, the peak TSB in babies with Rh incompatibility was 21.9±6.5mg/dl.

In this study, the peak TSB in G6PD deficient babies admitted for neonatal jaundice was 23.9 ± 4.1 mg/dl. Singhal et al.[15] in 1992, found peak TSB in G6PD deficiency 25.2 ± 9.7 mg /dl. EnverAtay et. al.[16] in 2005, in a study done in Turkey, found that mean peak TSB of 24.98 ± 5.9 mg/dl in G6PD deficient babies. Rahul Sinha et al[17]in 2016 in a study at Jodhpur, Rajasthan, found the mean maximum serum bilirubin level in the G6PD deficient group was 25.17 ± 5.60 mg/dl.The results found in the present study is comparable to most of other studies. It was found that TSB levels of the hyperbilirubinemia newborn infants with ABO and Rh incompatibility and G6PD deficiency were higher than TSB levels of the other hyperbilirubinemia newborn infants.

V. Conclusion

In present study, it is seen that the babies with ABO incompatibility, Rh incompatibility and G6PD deficiency have a higher level of total serum bilirubin and thus have propensity to undergo longer duration of phototherapy and more chances of undergoing exchange transfusion and thus are at higher risk of morbidity and consequent mortality. The neurodevelopmental outcome of such babies also needs to be evaluated further. So, there is a need of strengthening the peripheral health setups by making them aware of these risk factors namely ABO incompatibility, Rh incompatibility and G6PD deficiency as important causes of neonatal jaundice, particularly in this geographically and demographically prone population which will be helpful in to take appropriate measures and interventions on time to prevent the development of complications of severe neonatal jaundice

References

[1]. Pathologic hyperbilirubinemia. Available at: https://www.utmb.edu/pedi_ed/core/neonatology/pag e_30.htm
[2]. Thor WR Hansen. Chief Editor: Ted Rosenkrantz, MD; Neonatal Jaundice, 2016. http://emedicine.medscape.com/article/974786-overview hyperbilirubinemia. Indian J med Res. 1989; 306-13
[3]. Yigit S, Gursoy T, Kanra T, et al. Whole blood versus red cells and plasma for exchange transfusion in ABO haemolytic disease. Trans Med 2005; 15: 313-318.
[4]. Yaseen H, Khalaf M, Rashid N, Darwich M. Does prophylactic phototherapy prevent hyperbilirubinemia in neonates with ABO incompatibility and positive Coombs' test? J Perinatol 2005; 25: 590-594.
[5]. Kaplan M, Na'amad M, Kenan A, et al. Failure to predict hemolysis and hyperbilirubinemia by IgG subclass in blood group A or B infants born to group O mothers. Pediatrics 2009; 123: 132-137.
[6]. McIntosh N, Stenson B. The newborn. In: McIntosh N, Helms PJ, Symth RL (eds.) Forfar &Arneil's Textbook of Pediatrics. 6th ed. Edinburgh: Churchil Living Stone; 2003p.177-391
[7]. .Madan N, Sundaram KR, Bhargava SK ,Sood SK. Glucose-6-phosphate dehydrogenase deficiency and neonatal
[8]. Luzzatto L, Bienzle U. The malaria/G-6-P.D. hypothesis. 3. *Lancet* 1979; *1* : 1183-4
[9]. *People of India: an introduction*. Calcutta, India: Anthropological Survey of India; 1992
[10]. Singh, Sharad K. et al. Clinical Epidemiology and Global Health , Volume 4 , Issue 2 , 95 - 100
[11]. SinemAkgül et al The Turkish Journal of Pediatrics 2013; 55: 506-509
[12]. Shao-Wen Cheng,,et al, Chang Gung Med J vol. 35 No. 2, March- April, 2012
[13]. Yi-Hao Weng,et al, Chang Gung Med J vol. 32 No. 4, July- August 2009
[14]. ALTIKAT, Sait, Halil and S Kuru. "Research for the effects of rh and aboincompatibility in newborn infants on total bilirubin levels within the first 24 hours in and around kütahya." (2012).
[15]. Singhal PK, Singh M, Paul VK, Deorari AK, Ghorpade MG. Spectrum of neonatal hyperbilirubinemia: An analysis of 454 cases.IndianPediatr 1992; 29: 319-325
[16]. EnverAtay, Abdulkadir Bozaykut, IlkeOzahiIpek; Glucose-6-phosphate Dehydrogenase Deficiency in Neonatal IndirectHyperbilirubinemia, Journal of Tropical Pediatrics, Volume 52, Issue 1, 1 February 2006, Pages 56–5
[17]. Sinha R, Sachendra B, Syed VS, Nair L, John BM. To study the prevalence of glucose 6 phosphate dehydrogenase(G6PD) deficiency in neonates with neonatal hyperbilirubinemia and to compare the course of the neonatal jaundice in deficient versus non deficient neonates. J Clin Neonatol2017;6:71-4

Dr. Mrinalini Das, "A comparative study of peak total serum bilirubin level in neonates with ABO incompatibility, Rh incompatibility and G6PD deficiency. "IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 17, no. 10, 2018, pp 21-23.