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A comparative study of peak total serum bilirubin level in glucose-6-phosphate dehydrogenase deficient and glucose-6-phosphate dehydrogenase normal neonates in neonatal hyperbilirubinemia

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

Abstract: ***Introduction:*** *Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency is an inherited deficiency that may cause neonatal jaundice, as has been found in several countries and different ethnic groups.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. It is a genetically inherited sex-linked abnormality.* ***Methods:*** *The study was done on 994 clinically icteric babies without other risk factors for neonatal jaundice, 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, direct coomb's test, haemoglobin, blood smear examination, reticulocyte count, and G6PD status. Data was collected and adequate intervention was done depending on indication. Data was analysed by software SPSS version 20.* ***Results****- In G6PD deficient neonates (146), mean peak TSB level was 23.96 ± 4.173 mg/dl whereas in G6PD normal neonates, it was 19.93 ± 3.848 mg/dl. The P-value calculated is < 0.0001 which is extremely significant.* ***Conclusions:*** *The prevalence of severe NHB with G6PD deficiency among neonates in North East region is relatively high and babies with G6PD deficiency have a higher chance of severe hyperbilirubinemia and developing complications like kernicterus and poor neurodevelopmental outcome. Therefore, screening of newborns for G6PD deficiency need to be done in all newborns, so that G6PD deficiency can be identified and adequate intervention can be done timely.*

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I. Introduction

Glucose-6-Phosphate 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. Neonatal hyperbilirubinemia has been attributed to isoimmune incompatibility, low birth weight, prematurity, abnormal parturition, G6PD deficiency, infection, liver diseases, drugs and maternal causes.(1) Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common erythro-enzymopathy(2) affecting 10% of the world population. It plays a protective role against malaria(3).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.(4) G6PD deficiency is present in all the tribal groups studied from North-East India.(5)

In Assam, systematic neonatal or infantile screening is not yet implemented and thus, many parents are unaware of their children's health status. Without knowing the risk, they are exposed to ingestion of food or drugs which can aggravate hemolysis. Considering these consequences, risk of bilirubin encephalopathy and in severe cases death, especially in a resource limited state like Assam, the study was taken up.

Aim of the study-To compare the peak total serum bilirubin level in neonatal hyperbilirubinemia in G6PD deficient newborns and G6PD normal newborns admitted for neonatal jaundice in a tertiary care hospital in North-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 ABO incompatibility, Rh incompatibility, sepsis, prematurity, low birth weight, polycythemia, cephalhematoma, infant of diabetic mother and gastrointestinal obstruction etc. were excluded from the study. After taking necessary precautions and asepsis, relevant laboratory investigations were done 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 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) using integrated reagent cartridge.Results analyzed by computer based statistical package for the social science SSPS software version 20. P- values of less than 0.05 were considered statistically significant.

III. Results

A total of 994 babies without other precipitating factors, were admitted for neonatal hyperbilirubinemia. All 994 neonates were tested for G6PD enzyme deficiency. Out of 994 neonates tested for G6PD deficiency and 146(14.7%) were identified as G6PD deficient neonates. In the study, the major fraction of G6PD deficient neonates are predominated by male babies. Male constituted 80.14% and females 19.86% (Table.1 a&b)

Table.1(a & b) G6PD deficient males and females among NHB babies

G6PD status	Number of babies n=994	G6PD deficient 146(14.7%)	
G6PD deficient	146(14.7%)	Males	Females
G6PD normal	848(85.3%)	117(80.14%)	29(19.86%)

In this study, in G6PD deficient neonates, mean peak TSB level was 23.96 ± 4.173 mg/dl whereas in G6PD normal neonates, it was 19.93 ± 3.848 mg/dl. The p-value calculated is < 0.0001 which is extremely significant. When the peak TSB level was compared between in G6PD deficient male and G6PD deficient female neonates, it was 23.40 ± 4.11 mg/dl and 22.66 ± 4.11 mg/dl respectively. The P-Value is >0.05 (p= 0.0725) which is not significant. Fig 2(a and b)

Fig.2(a) Mean peak TSB level

G6PD status	**Peak TSB (mean ± SD)(mg/dl)**
Normal	19.93 ± 3.453
Deficient	23.25 ± 4.173
P- value	< 0.0001

Fig.2(b)Mean peak TSB level in G6PD deficient male and female babies

G6PD deficient neonates	**Peak TSB (mean ± SD)(mg/dl)**
Males	23.40 ± 4.158
Females	22.66 ± 4.064
P- value	< 0.0725

IV. Discussion

Since G6PD deficiency plays a protective role against malaria[(3)], it can explain its increased prevalence in this region. In this study, the peak TSB in G6PD deficient babies and G6PD normal babies admitted for neonatal jaundice was 23.96 ± 4.173 mg/dl and 19.93 ± 3.848 mg/dl. Peak TSB level was compared between in G6PD deficient male and G6PD deficient female neonates, it was found to be 23.40 ± 4.11 mg/dl and 22.66 ± 4.11 mg/dl respectively. The P-value is >0.05 (P = 0.0725) which is not significant. Singhal et al[(6)] in 1992, found peak TSB in G6PD deficiency 25.2 ± 9.7 mg /dl where as in idiopathic NHB babies , peak TSB was 16.9 ± 4.1mg/dl. Pao et al[(7)] in 2003, in a study done at Delhi, showed that mean maximum serum bilirubin level in G6PD deficient babies was 17.8 mg/dl. Enver Atay et al in 2005[(8)], in a study done in Turkey, found that mean peak TSB was 21.2 ± 3.7 mg/dl and 24.98 ± 5.9 mg/dl in G6PD normal and deficient groups, respectively.

Rahul Sinha et al[9] 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 and that of G6PD normal was 17.36 ± 2.8 mg/dl. The results found in the present study is comparable to most of other studies except to that of Pao et al[7]

V. Conclusion

The prevalence of severe NHB with G6PD deficiency among neonates in North East region is relatively high. Neonatal jaundice is the most common clinical presentation. The babies with G6PD deficiency have a higher chance of developing severe hyperbilirubinemia and therefore higher chances developing various complications like kernicterus which can cause death and neurodevelopmental handicap. G6PD is an X-linked condition[10] and males are therefore more commonly and more severely affected than females but when females are G6PD deficient ,the development of jaundice is almost equally severe . In later life, the disease can cause acute haemolytic crises on exposure to agents/drugs which trigger or sensitize the red cells leading to significant morbidity and mortality in childhood. So in a population with a high prevalence rate, early detection of G6PD enzyme deficiency by neonatal screening of all babies is desirable to take appropriate measures and interventions to prevent the complications of hemolysis and jaundice.

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