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ASSOCIATION OF NEONATAL HYPERBILIRUBINEMIA WITH GLUCOSE-6-PHOSPHATE DEHYDROGENASE ENZYME ACTIVITY WITH REFERENCE TO ABO-RH STATUS

Biochemistry

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ABSTRACT

Background: Neonatal hyperbilirubinemia manifests itself in first week of life and is an extremely common condition globally. Although the exact underlying pathophysiological mechanisms are not very well delineated, RBC glutathione store and hence the activity of glucose-6-phosphate Dehydrogenase, the key enzyme of Hexose Monophosphate shunt to provide reduced NADP is crucially implicated.

Materials & Methods: In this non interventional, cross-sectional, hospital based study, 200 randomly selected, clinically and biochemically confirmed hyperbilirubinemic neonates within their first 148 hours of life were selected as study subjects as per the inclusion and exclusion criteria. ABO grouping & Rh typing was done, serum total and direct bilirubin concentrations were assayed, and RBC G6PD activity measured.

Result: The study revealed 6% of hyperbilirubinemic neonates were having G6PD deficiency. But the hyperbilirubinemic neonates with G6PD deficiency were more prone to develop complicated disease so as to be managed by exchange blood transfusion. No specific blood group swas shown to be associated with hyperbilirubinemia although O positive group revealed a slightly higher predilection to G6PD deficiency. Consanguineous marriage was shown to be associated with G6PD deficiency.

Conclusion: Present study shows there is relationship between hyperbilirubinemia and G6PD deficiency and this enzyme deficiency is more commonly found in consanguineous marriage.

KEYWORDS

Bilirubin , Jaundice , Glucose 6 Phosphate Dehydrogenase , Neonate , Blood group

INTRODUCTION:

Neonatal hyperbilirubinemia is one of the commonest clinical conditions encountered in the first week of life. This is defined as elevation of serum total Bilirubin concentration above the 95th percentile for age. It involves nearly 8-11% of all neonates[1]. In most cases, the disease is self limiting, but some need admission and rarely, exchange transfusion.

Neonatal hyperbilirubinaemia is predominantly idiopathic in nature and does not lead to severe complications of unconjugated hyperbilirubinaemia like Kernikterus. But the risk of complications increase manifold when there is inherent RBC membrane disorders.

Glucose- 6- phosphate- dehydrogenase(G6PD) deficiency is the commonest enzymopathy worldwide, with an estimated global prevalence of 400 million people[2]. This X-linked recessive disorder is an independent risk factor for unconjugated hyperbilirubinaemia as it results in depletion of erythrocytic reduced glutathione store. This is the first enzyme of Hexose Monophosphate(HMP) shunt, which is the primary source of $NADPH+H^+$ within erythrocytes[3].

In this study, an attempt was made to find out any relationship between G6PD activity and severity of neonatal hyperbilirubinaemia. We also made an attempt to find out any possible relationship between neonatal hyperbilirubinaemia and ABO group & Rh type.

MATERIALS AND METHODS:

The study was done in the Department of Biochemistry with collaboration of the Department of Neonatal Intensive Care Unit (NICU), IPGMER & SSKM Hospital, Kolkata from January, 2017 to June, 2018. A total of 200 diagnosed hyperbilirubinemic neonates were taken for the study. Non polycythaemic newborns > 37weeks of gestation and uneventful labour, with birth weight ≥2.5 kg, with no foeto-maternal Rh incompatibility, were selected as the study group. Hemoglobin and serum total and conjugated bilirubin concentrations, G6PD activity in RBC lysate were estimated by spectrophotometric methods, and ABO & Rh typing were carried out by standard slide agglutination procedure. The results were analysed by statistical package for social sciences(SPSS) software.

Result and Analysis :

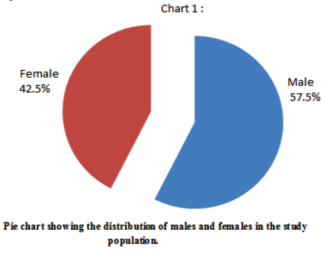

Chart 1: showing gender distribution of the subjects in study population (n=200).

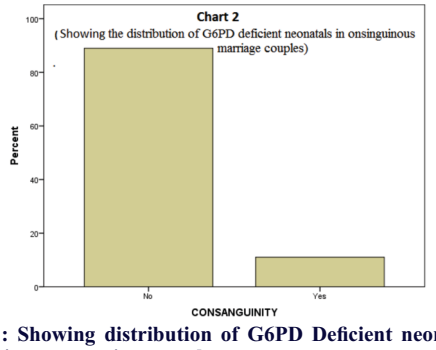

Chart 2 : Showing distribution of G6PD Deficient neonates in consanguineous marriage couple.

Table 1 (Showing distribution of case in respect to ABO Rh type)

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	2	1.0	1.0	1.0
	A-	2	1.0	1.0	2.0
	A+	26	13.0	13.0	15.0
	AB-	4	2.0	2.0	17.0
	AB+	22	11.0	11.0	28.0
	B-	5	2.5	2.5	30.5

	B+	59	29.5	29.5	60.0
	O+	80	40.0	40.0	100.0
	Total	200	100.0	100.0	

Table 1: Showing distribution of different blood groups in study population (n=200).

DISCUSSION:

Neonatal hyperbilirubinemia, due to Glucose-6-Phosphate Dehydrogenase (G6PD) enzyme deficiency leads to hemolysis in some cases after oxidative challenge[4]. Although in most cases it may be asymptomatic and usually presents as hyperbilirubinemia without any hemolytic disease.

The present study was done in the Department of Biochemistry and Neonatal Intensive Care Unit, Institute of Post Graduate Medical Education and Research, SSKM Hospital, Kolkata from January 2017 to June 2018 to detect the incidence of Glucose-6-Phosphate Dehydrogenase deficiency in hyperbilirubinemic neonates.

In our study, out of 200 hyperbilirubinemic neonates, 12 neonates were found to be having G6PD deficiency (6%). This was consistent with a similar study by Marzban et al, where the frequency of G6PD deficiency in 244 icteric neonates of Tehran, Iran was found to be 5.7%. A mean of 57.50% male and 42.50% female neonates were found to be hyperbilirubinemic out of the total population. This result was found to be consistent to another study done by Dholakia et al.

It was found that in this study population, there was insignificant relationship between G6PD deficiency and different blood groups. However, it was found that O+ blood group had a slight predilection towards this enzyme deficiency.

The study shows a significant relationship between G6PD deficiency and consanguineous marriage($p<0.05$).

Our study also revealed that out of the total study population, 4 neonates (2%) were in need of exchange blood transfusion. Out of the 4 neonates who required exchange transfusion, 2 neonates were found to be G6PD deficient.

SUMMARY AND CONCLUSION:

Glucose- 6- phosphate- dehydrogenase is one of the most important enzymes which helps in maintaining the integrity of erythrocytes by maintaining reduced glutathione reserve.

G6PD defiency is one of the commonest inborn errors of metabolism in eastern parts of India as well as parts of Africa, Asia, Mediterranean region and Middle East. Being an X-linked recessive disease, it is more common in male neonates compared to females.

Severe neonatal hyperbilirubinemia is most serious consequence of G6PD defiency[6]. Approximately, 60% of the term and 80% of preterm neonates became icteric in the first week of life[5]. In most cases, it is self-limiting in nature but relatively less number of neonates require exchange blood transfusion. Such type of severe neonatal hyperbilirubinemia is often associated with G6PD deficiency than other neonatal hemolytic diseases.

In our study, 4 neonates with hyperbilirubinemia ended up in need of exchange blood transfusion, out of which 2 were G6PD deficient.

Consanginous marriages have also been found to have a positive relationship with G6PD deficiency. However, no significant relationship was found when tested in different blood groups, but it was noticed that O positive blood group shows a higher prediction to G6PD deficiency, although statistical result were insignificant($p>0.05$).

Hence we conclude that G6PD activity should be measured routinely in hyperbillurubinimic or severly icteric neonates considering his/her age, sex and maturity of life, so that further complications can be avoided in time and for the better future of those neonates.

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