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Study on screening for glucose 6 phosphate dehydrogenase deficiency in newborn babies at our hospital

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ABSTRACT

Background: Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common human enzyme deficiency in the world. G6PD deficiency is an X-linked disorder affecting mostly African, south east Asian and middle-eastern population. In India, there are 13 biochemical variants of G6PD being reported so far out of which Mediterranean type is most common in the caste groups.

Aim and Objectives:to find out the incidence of G6PD deficiency in neonates delivered at our tertiary care hospital and to assess the contribution of G6PD deficiency in causing Neonatal jaundice.

Materials and Methods: The study was a prospective hospital based type undertaken among the 500 babies delivered at our hospital from. The objective of this study was to find out incidence of G6PD deficiency in babies delivered in our Hospital and to assess its contribution in causing neonatal jaundice. A detailed antenatal history, consanguinity, geographical area of mother, birth order, mode of delivery, gestational age, birth injuries and blood group were recorded in each baby by interviewing the mother after taking consent in prescribed proforma. The following laboratory investigations were carried out, G6PD screening test (fluorescent spot test), Baby blood group and Rh typing, Mother blood group and Rh type and If jaundice present total, direct and indirect bilirubin testing was done. If the sample (cord blood) shows G6PD deficiency by fluorescent spot method, quantitative assay was done.

Discussion and Conclusion: This study was conducted to detect incidence of G6PD deficiency in neonates and as acontributory factor in neonatal hyperbilirubinemia. The result of this study shows that incidence of G6PD deficiency in neonates delivered at our hospital is zero. Since incidence is zero in present study, G6PD deficiency is not a major cause forneonatal jaundice in our hospital.

Key-words: Glucose-6-phosphate dehydrogenase (G6PD) deficiency, newborn babies, fluorescent spot assay and neonatal jaundice.

INTRODUCTION:

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most commonhuman enzyme deficiency in the world [1]. G6PD deficiency is an X-linked disorderaffecting mostly African, south east Asian and middle-eastern population [2,3]. In India,there are 13 biochemical variants of G6PD being reported so far out of whichMediterranean type is most common in the caste groups. Orissa variant is mostprevalent in tribals of India. Kerala-Kalyan is the 3rd common variant [4]. Incidence ofG6PD deficiency in different geographical areas of India varies from 0 to 27% [5].

G6PD is the first enzyme of the pentose phosphate pathway and catalyzes theconversion of glucose-6-phosphate to 6-phosphogluconolactone (G6P), with theconcomitant reduction of nicotinamide adenine dinucleotide phosphate (NADP) to its reduced form (NADPH). NADPH protects cells from oxidative stress. Glucose-6-phosphate dehydrogenase deficiency causes a spectrum of diseases including neonatalhyperbilirubinemia, acute hemolysis. This deficiency can cause hemolytic anemia,usually after certain medications.Homozygotes and heterozygotes can be symptomatic, although the diseasetypically is more severe in persons who are homozygous for the deficiency.

The conversion of nicotinamide adenine dinucleotide phosphate to its reducedform in erythrocytes is the basis of diagnostic testing for the G6PD deficiency. Thisusually done by fluorescent spot test. With the right precautions, a child with G6PDdeficiency can lead a healthy and active life [6]. Hence this study was done to know theincidence of G6PD deficiency in neonates delivered at our tertiary care hospital and itscontribution in causing neonatal jaundice.

AIM AND OBJECTIVES:

1. To find out the incidence of G6PD deficiency in neonates delivered at our tertiary care hospital.
2. To assess the contribution of G6PD deficiency in causing Neonatal jaundice.

MATERIALS AND METHODS:

The study was a prospective hospital based type undertaken among the 500babies delivered at our hospital from September 2019 to August 2020. The objective ofthis study was to find out incidence of G6PD deficiency in babies delivered in our Hospital and to assess its contribution in causing neonatal jaundice.

A detailed antenatal history, consanguinity, geographical area of mother,birth order, mode of delivery, gestational age, birth injuries and blood group wererecorded in each baby by interviewing the mother after taking consent in prescribedproforma.

Study design: Prospective hospital based study.

Sample size: 500 babies delivered in our hospital in the duration of one year.

Sample size was obtained through sample size calculated provided by creative research system through online survey system. To determine sample size for 2000 babies delivered in one year with confidence level of 95% and confident interval of 5, requiring sample size was 322. For more convenient sample size is rounded to 500.

Inclusion Criteria: Babies delivered at our hospital.

Exclusion Criteria: none

Blood Sample Collection and Biochemical Investigations: The following laboratory investigations were carried out,

1. G6PD screening test (fluorescent spot test)
2. Baby blood group and Rh type
3. Mother blood group and Rh type
4. If jaundice present

a. Indirect bilirubin
b. Direct bilirubin

c. Hemoglobin
d. DCT(direct Coomb's test)
e. Retic count

If the sample (cord blood) shows G6PD deficiency by fluorescent spot method, quantitative assay was done.1ml cord blood of all babies collected in EDTA vials immediately after birth. 10 microliters of collected blood is added to 100 microliter reagent mixture,a spot is made on a Whatman No.3 filter time. The sample is incubated at room temperature and further spots made at 5,10,15, and 20 minutes respectively. The spots are allowed to dry and examined under a long wave UV lamp (Fluorescent Spot Test Method). In normal samples, the first spot fluoresces slightly with increasing in fluorescence in the remaining spots indicating reduction of NADP to NADPH+H. G6PD deficient sample did not show fluorescence in any of the spots. Babies found to have G6PD deficiency were followed, if they develop jaundice standard management protocol of the hospital is employed.

Criteria for some of the variables are: Cord blood samples fluoresce under fluorescent microscopy were consideredas normal G6PD activity. Preterm babies had been defined as those neonates havinggestational age < 37 weeks, and low birth weight (LBW) babies were those whosebirth weight <2.49 kg. Serum bilirubin levels were obtained when newbornsdeveloped jaundice.

Descriptive statistics:
The descriptive procedure displays univariate summary statistics for several variables. In a single table and calculates standardized values (Z score). Variables were ordered by the size of their means (in ascending or descending order), alphabetically, or by the order in which we select the variable (the default)

Cross tabs procedure:
The crosstabs procedure forms two-way and multiway tables and provide a variety of tests and measure of association for two-way tables. The structures of the table and whether categories are ordered determine what test or measure to use. All the statistical methods were carried out through the SPSS for windows.

RESULTS:

In the present study, 500 babies delivered in our hospital, were included for screening for G6PD deficiency by fluorescent spot method, all of them were found have normal activity.

Table 1: Incidence of G6PD deficiency in neonates delivered in our hospital

Number of babies	Method	Number of babies with G6PD deficiency	Incidence
500	Fluorescent spot method	0	0

Table 2: Sex of the baby versus G6PD screening

Sex of the	Frequency	Percentage	G6PD (normal)	G6PD (deficient)

baby				
Male	266	53.2	266	0
Female	234	46.3	234	0
Total	500	100	500	0

Table 3: Gestational Period versus G6PD screening

Gestational period	**Frequency**	**Percentage**	**G6PD (normal)**	**G6PD (deficient)**
Term	469	93.3	469	0
Pre-term	31	6.2	3.1	0
Total	500	100	500	0

Table 4: Birth Weight versus G6PD screening

Birth Weight (Kgs)	**Frequency**	**Percentage**	**G6PD (normal)**	**G6PD (deficient)**
<2.49	92	18.4	92	0
2.50-2.99	152	30.4	152	0
3.00-3.49	221	44.2	221	0
3.50+	35	7.0	35	0
Total	500	100	500	0

Table 5: Consanguineous marriage versus G6PD screening

	Frequency	**Percentage**	**G6PD (normal)**	**G6PD (deficient)**
NCM	467	93.4	467	0
CSM	33	6.6	33	0
Total	500	100	500	0

Table 6: Jaundice versus G6PD screening

	Frequency	**Percentage**	**G6PD (normal)**	**G6PD (deficient)**
Jaundice	42	9.1	42	0
Non-Jaundice	477	89.9	477	0
Total	500	100	500	0

Table 7: Causes of Jaundice Versus G6PD Deficiency

Cause of Jaundice	**Number of Babies**
Rh Incompatibility	4
OA Incompatibility	6
Infants of Diabetic Mother	2
Sepsis	3
Exaggerated physiological jaundice	27
G6PD Deficiency	0
Total	42

DISCUSSION:

In the present study, 500 babies delivered at our hospital were screened todetect incidence of G6PD deficiency in neonates by semiquanatitive assay. All thebabies were found to have normal G6PD activity.In study done by Mritunjay et al,[7] out of 2479 babies screened for G6PDdeficiency by a semiquanatitive assay,50 neonates were found to be G6PD deficiencywith incidence being 2%.Ramin Iranpour et al[8] screened 2501 babies, by a semiquanitative assay, outof which 79 neonates were found to have G6PD deficiency, incidence being 3.2%.

In the present study, out of 500babies, 262 babies (53.2%) were males and 234 were females (46.8%). All the babieswere found to have normal G6PD activity.In study done by Mritunjay et al [7] out of 2479 babies screened for G6PD deficiency,1343 were males and 1136 were females. 2.83% males and 1.05% females were foundto be G6PD deficient.Ramin Iranpour et al[9] screened 2501 babies, 1307 were males and 1194 werefemales. Out of which 67 males (5.1%) and 12 (1%) females were found to be G6PDdeficient.

In present study, out of 500 babies screened 469 were term(93.8%) and 31were preterm (6.2%). All the babies were found to have normal G6PD activity.Out of 2501 babies screened by Ramin Iranpour et al, [9] 2291(91.6%) babieswere term and 202 (8.4%)were preterm babies.68 term babies(2.9%) and 11 pretermbabies(5.4%) were found to have G6PD deficient.Sukumal et al[10] screened 109 babies out of which, 86(78.7%) were term and23(21.3%) were preterm babies.10 term babies(11.6%) and 6 preterm(26.08%) werefound to be G6PD deficient.

In present study 500 babies were screened, out of which 467 babies were bornto non-consanguineous marriage (93,4%) and 33 babies born to consanguineousmarriage (6.6%). All the babies were found to be with Normal G6PD activity. In the study by Sukumal et al, [10] out of 109 babies screened, 5(4.6%)were bornto consanginous married couple and 1(20%) was found to be G6PD deficient. Out of104 (95.4%) babies born from non-consanguineous marriage, 15 babies(14.42%) wereG6PD deficient.

In present study, allthe babies found to have normal G6PD activity. Regional distribution of G6PD deficiencystudied by Bhasin et al, [11] showed 0% deficient in Karnataka, 0.03% in Andhrapradesh,and 0.07% in Tamilnadu.Study by Vandana Rai et al, [12] showed the frequency of G6PD deficiencyamong Indian population as a whole ranges from 0 to 27%. G6PD-deficientfrequency is comparatively higher in North and West Indian zones, whereas in SouthIndia it is uniformly low except in Andhra Pradesh and Tamil Nadu.Hakim et al screened 186 babies in Kerala, all were showing normal G6PDactivity.

In present study, out of 500 babies screened for G6PD deficiency, 43 babies developedJaundice. All of them had normal G6PD activity.Sukamol Bisol screened 109 babies, out of which 21 babies developed jaundice and 3babies found to be G6PD deficient with P value of > 0.05 which is not significant.

CONCLUSION:

In the present study, 500 babies delivered in our hospital were screened to detect incidence of G6PD deficiency in neonates. The screening was done by fluorescent spot test. Out of 500 babies screened, 266 were males and 234 are females. Screened, 69 babies were term and 31 were preterm. 500 babies were screened, out of which 467 babies born to NCM and 33 babies born to CSM.

Among 500 babies screened for G6PD deficiency, 43 babies developed Jaundice. The incidence of G6PD deficiency found to be zero out of 500 babies screened.

This study was conducted to detect incidence of G6PD deficiency in neonates and as acontributory factor in neonatal hyperbilirubinemia. The result of this study shows thatincidence of G6PD deficiency in neonates delivered at our hospital is zero. Since incidence is zero in present study, G6PD deficiency is not a major cause forneonatal jaundice in our hospital.

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