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Original Research Article

Assessment of the prevalence of G6PD deficiency in RBCs of live new-borns born at tertiary care hospital

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ABSTRACT

Background: Glucose-6-phosphate dehydrogenase is one of many enzymes that help the body process carbohydrates and turn them into energy. The mechanism by which G6PD deficiency causes neonatal hyper bilirubin may be due to hemolysis, but other mechanisms like secondary impairment of bilirubin conjugation and clearance by the liver may play a role. Therefore, through this study authors attempt to study the need for a newborn screening program for G6PD deficiency because of high prevalence and high risk of incidence due to consanguineous marriages in India.
Methods: This study was a prospective cross-sectional study conducted among 350 consecutively born live new-borns in maternity wards and NICU of Krishna Institute of Medical Sciences and Hospital and Research Centre, Karad, Maharashtra during October 2016 to October 2017.
Results: The maximum numbers of newborns were in the age group of 0-10 hours (36.80%), followed by in 11-20 hours (21.80%). The mean age among newborns was 2.86±5.83 hours. Out of 350 cases females were 181 (51.71%) and males (48.29%) and female to male ratio was 1.07:1.
Conclusions: G6PD deficiency is one of the major causes of neonatal jaundice within 24 hours of life in new-borns. Hence, neonatal screening for G6PD deficiency could be an alternative to the haemolytic crisis prevention strategy in order to optimize affected young child care and prevention of crisis occurrence by avoiding taking contraindicated foods and drugs.

Keywords: G6PD deficiency, Haemolysis, Haemolytic anemia, Jaundice, New-born Prevalence, Screening

INTRODUCTION

Glucose-6-phosphate dehydrogenase is one of many enzymes that help the body process carbohydrates and turn them into energy. G6PD is a cytoplasmic enzyme in the hexose monophosphate pathway and catalyzes the conversion of nicotinamide adenine dinucleotide phosphate (NADP) to its reduced form, NADPH. NADPH maintains glutathione in its reduced form, which acts as a scavenger for dangerous oxidative metabolites. G6PD also protects red blood cells from potentially harmful by-products that can accumulate when a person takes certain medications or when the body is fighting an infection that would otherwise cause precipitation of hemoglobin (Heinz bodies) or damage the RBC membrane.[1]

Glucose-6-phosphate dehydrogenase (G6PD) deficiency, the most frequent disease involving enzymes of the hexose monophosphate pathway, is responsible for 2 clinical syndromes, episodic hemolytic anemia, jaundice and chronic non spherocytic hemolytic anemia. G6PD deficiency is the most common erythrocyte enzymopathy.[2]

India is currently undergoing an epidemiological transition and congenital malformations and genetic disorders are gradually replacing sepsis as the major cause of perinatal and neonatal mortality. Presently, they constitute the fourth commonest cause (9.2%) of neonatal mortality in urban areas. With almost 24 million children born annually in India, it is estimated that at least 390,000 children suffering from this disorder are born in the country every year. This is because consanguineous marriages are still fairly common in many parts of India.[2]

The diagnosis of red cell enzyme deficiency usually depends on the demonstration of decreased enzyme activity either through a quantitative assay or a screening test. There are several methods available for the diagnosis of G6PD deficiency. However, fluorescent spot test and dichlorophenol indophenol (DPIP) decolorization method were found to be useful and suitable for routine use.[3]

Therefore, through this study we attempt to study the need for a newborn screening program for G6PD deficiency because of high prevalence and high risk of incidence due to consanguineous marriages in India. Also, routinely administered vitamin K prophylaxis can cause hemolysis in G6PD deficient newborns and may prove potentially fatal, which can be prevented if babies are screened for G6PD deficiency.

METHODS

This study was a prospective cross-sectional study conducted among 350 consecutively born live newborns in maternity wards and NICU of Krishna Institute of Medical Sciences and Hospital and Research Centre, Karad, Maharashtra during October 2016 to October 2017.

After obtaining an informed consent was taken from the parents of the neonate after explaining in detail about the procedure of the study in their vernacular language. The study was approved by the institutional ethical committee of KIMSDU, Karad.

Exclusion criteria

- Babies who are not born at tertiary care hospital, Babies who were stillborn or expired, who are born preterm/ IUGR/ SGA/ congenital anomalies/ sick babies or babies who received blood transfusion or undergone exchange transfusion and babies whose parents have not given consent were excluded from the given study.

Details of maternal parameters like name, age, hospital details, type of delivery, any risk factors, past obstetric history, any medical and surgical illness were recorded in proforma. Details of neonate like date of delivery, sex, birth weight, history of previous babies having hemolysis or blood transfusion and examination details were recorded.

G6PD screening test: G6PD test kit qualitative method (96MB100-10(N)25963(O)10X0.5ml) manufactured by Arkray healthcare Pvt. Ltd were used for diagnosing G6PD deficiency.

Blood sample were collected from healthy newborns born at tertiary care hospital over a period of one and half year. Cord blood was not used for estimation due to the reference that a study done in Saudi Arabia showed that G6PD levels in the RBCs of cord blood had less amount of G6PD, thus there are chances of false negative results. Thus, to eliminate this bias we used venous blood of newborns within 48 hours of life. 2ml of whole blood sample was collected by intravenous route in EDTA bulb for analysis of qualitative G6PD estimation by decolorization of 2,6 dichlorophenol indophenols. Babies who came G6PD positive were further screened for hyperbilirubinemia and other investigations done were: CBC with Retic count, Peripheral smear with supravital stain, Bilirubin total, direct and indirect, liver function test, Direct coombs test and urine routine and microscopy.

Stastical analysis

The data was analyzed with the help of SPSS software version 21. The data was represented using tables and charts. Appropriate statistical tests were applied to find out the significance between the differences. Observations with p-value less than 0.05 were considered as significant.

RESULTS

The maximum numbers of newborns were in the age group of 0-10 hours (36.80%), followed by in 11-20 hours (21.80%). The mean age among newborns was 2.86±5.83 hours. Out of 350 cases females were 181 (51.71%) and males (48.29%) and female to male ratio was 1.07:1 (Figure 1).

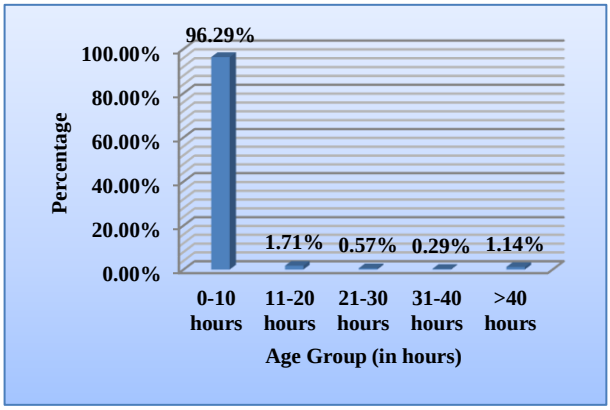

Figure 1: Age distribution.

The majority of newborns had birth weight 2500-3000 grams (52.86%) followed by >3000grams (28%) and <2500 grams (19.14%) (Figure 2).

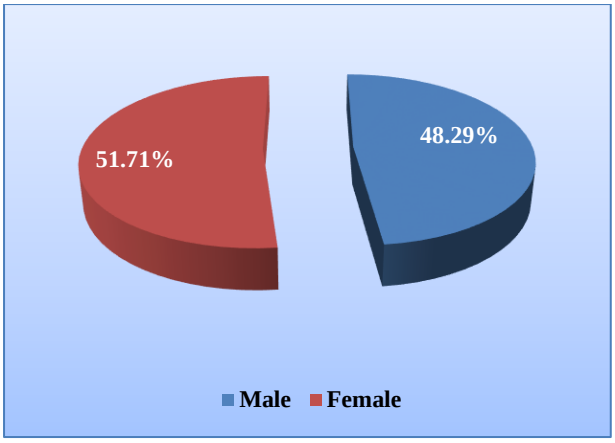

Figure 2: Sex Distribution.

The prevalence of G6PD deficiency among newborns those who were tested were 0%. The majority of newborns were Hindu by religion (67.14%) followed by Muslims (22.86%), 6% newborns were Buddhist and 4% of other religion. It was observed that the majority of newborns were born out of Non-consanguineous marriage (85.43%) followed by 3rd degree consanguinity (12.28%). The 1st degree consanguinity was present among 1 (0.29%) patient. It was observed that the majority of newborns were Hindu by religion (67.14%) with maximum number of third-degree consanguinity (16.59%) and second-degree consanguinity (2.97%) followed by Muslims (22.86%) in whom third degree consanguity was around (5%), 6% newborns were from Buddhist and 4% from other religion. The majority of newborns were born out of Non-Consanguineous marriage (85.43%) followed by 3rd degree consanguinity (12.28%). The 1st degree Consanguinity was present among 1 (0.29%) patient (Table 1).

Table 1: Newborns distribution according to religion and consanguinity.

Religion	Consanguinity				Total
	No	1º	2º	3º	
Hindu	188	01	07	39	235
Muslim	76	00	00	04	80
Buddhist	21	00	00	00	21
Others	14	00	00	00	14
Total	299	01	07	43	350

Majority of newborns in 2-3 gravidity (51.43%) followed by primi gravid mothers (42.86%) (Table 2). Majority of newborns had normal hemoglobin 275 (78.57%) while 63 newborns (18%) had anemia. (Hb<17.1gm%) while 12 newborns had polycythemia (Hb>20.9).

Table 2: Distribution according to gravidity.

Gravida	No. of new born	Percentage
1	150	42.86
2	130	37.14
3	50	14.29
4	10	2.86
5	08	2.29
6	01	0.29
7	01	0.29
Total	350	100

Table 3: Distribution of study subjects according to various parameter.

Parameters		No. of new born	Percentage
Hyperbilirubinemia	Present	03	00.86
	Absent	347	99.14
	Total	350	100
History of drug intake	Oxidant drug (Vitamin K)	341	97.43
	Oxidant drug (Vitamin K) and Non-oxidant drug (Inj ampicillin, inj gentamycin)	07	02.00
	No drugs	02	00.57
	Total	350	100
Time (Hours) of life of newborns	<24	02	66.67
	24-48	01	33.33
	Total	03	100
Diagnosis of hyperbilirubinemia	Rh incompatibility	01	33.33
	ABO incompatibility	01	33.33
	Unknown cause	01	33.33
	Total	03	100
Exchange transfusion	Required	01	33.33
	Not required	02	66.67
	Total	03	100

It was observed that the majority of newborns had history of drug intake such as vitamin k (99.42%). Two babies who were not given any drug, because parents did not give consent. Majority of newborns had history of drug intake of oxidant drug vitamin K (97.43%) followed by oxidant drug (Vitamin K) and Non-oxidant drug (Inj ampicillin, Inj gentamicin) (2%).

Two babies who were not given any drug, because parents did not give consent (Table 3). Non-oxidant and oxidant drug given or taken during pregnancy may trigger hemolysis in G6PD deficient newborns. It was observed that the majority of mothers 341 (97.43%) had no history of oxidant drug intake while other 9 (2.57%) mothers had positive history of oxidant drug intake (Table 3).

It was observed that the majority of newborns had no hyperbilirubinemia (99.14%) while 3 (0.86%) newborns had presence of hyperbilirubinemia. Majority of newborns had hyperbilirubinemia (66.67%) within 48 hours of birth and no newborn had positive family history of G6PD/haemolytic disease and also no parents or close relatives had H/O episodic or repetitive blood transfusion for anemia or any other hemolytic diseases or no parents had migrated from G6PD prevalent area. Among 3 newborns with hyperbilirubinemia Rh incompatibility, ABO incompatibility and physiological jaundice was observed in 1 (33.33%) newborn each respectively (Table 3).

The mean hemoglobin levels were 14.8, 16.5 and 17.2, mean PCV levels were 43.3, 48 and 52.6, the mean total leucocyte count was 6500, 10800 and 33800 per cumm, mean platelet levels were 210000, 109000 and 180000 per cumm and mean reticulocyte count percentage was 2.34, 3.12 and 2.93 in baby 1, baby 2 and baby 3 respectively (Table 4). It was observed that, babies blood group were B^+ve, AB $^+$ve and B $^+$ve, mean total bilirubin levels were 24.8, 14.9 and 18.6 and mother's blood groups were O^-ve, O^+ve and B $^+$ve of baby 1, baby 2 and baby 3 respectively (Table 4). The DCT was positive in baby 1 and negative in other newborns. The hemoglobinuria was absent in all newborns with hyperbilirubinemia.

Table 4: Distribution of various haematological parameters among the babies presented with hyperbilirubinemia.

Parameters	Baby 1	Baby 2	Baby 3
Hemoglobin (gm%)	14.8	16.5	17.2
Packed cell volume	43.3	48	52.6
Total leucocyte count	6500	10800	33800
Platelets	210000	109000	180000
Retic count	2.34	3.12	2.93
Baby's blood group	B^+ve	AB^+ve	B^+ve
Mother's blood group	O^-ve	O $^+$ve	B^+Ve
Direct Coombs Test (DCT)	Positive	Negative	Negative
Hemoglobinuria	Absent	Absent	Absent
Total bilirubin	24.8	14.9	18.6
Indirect billirubin	24.0	14.5	0.40
Direct billirubin	0.80	17.9	0.70

Table 5: Relation of hemoglobin and hyperbilirubinemia among newborns.

Parameters		Hyperbilirubinemia within 48 hours of life		Total	P value
		Present	Absent		
Haemoglobin (gm%)	<17.1	01	12	13	χ^2=1.42, DF=2, P=0.23 Not significant
	≥17.2	02	335	337	
	Total	03	347	350	
Birth weight (grams)	<2500	02	65	67	χ^2=4.31, DF=2, P=0.09 Not significant
	2500-3000	01	184	185	
	>3000	00	98	98	
	Total	03	347	350	

It was observed that presence of hyperbilirubinemia was more prevalent in birth weight <2500grams but showed no statistically significant relation (P>0.05). It was observed that there was no statistically significant relation between hemoglobin and hyperbilirubinemia (P>0.05).

All newborns treated with phototherapy in hyperbilirubinemia and among 3 newborns; 1 (33.33%) patient required exchanges transfusion (Table 5).

DISCUSSION

Glucose-6-phosphate dehydrogenase (G6PD) is an enzyme with considerable role in the metabolism of red blood cells. The enzyme deficiency leads to impaired production of reduced glutathione and predisposes the red cells to damage by oxidative metabolites, causing acute or chronic hemolysis. G6PD deficient neonates may be asymptomatic or manifest clinically as hyperbilirubinemia or even kernicterus or with high

colored urine. Screening for G6PD deficiency and recognition of prevalence of the enzyme deficiency in individual communities have got definite place in investigating the case of neonatal jaundice.

The present study was a prospective cross-sectional study undertaken to study the prevalence of G6PD deficiency in RBCS of live newborns born at tertiary care hospital. In the present study, age distribution among newborns showed that maximum numbers of newborns were in the age group of 0-10 hours (96.29%), followed by in 11-20 hours (1.71%). In the present study we collected venous blood for screening G6PD deficiency. The mean age among newborns was 2.86±5.83 hours. Out of 350 newborns females were 181 (51.71%) to males (48.29%) and female to male ratio was 1.07:1.

Adedemy JD et al, studied the prevalence of glucose 6 phosphate dehydrogenase deficiency among infants and children of Parakou, Benin.[4] Out of 510 infants and children were selected more than half were boys (52.5%) with male to female sex-ratio of 1.1:1.

Mukherjee S et al, studied prevalence of glucose 6 phosphate dehydrogenase deficiency in eastern india, observed out of 250 newborns 74.4 % were males while 25.6 % were females.[5]

It was observed that the majority of newborns had birth weight between 2500-3000 grams (52.86%) followed by >3000grams (28%) and <2500grams (19.14%). The majority of newborns were Hindu by religion (67.14%) followed by Muslims (22.86%), Buddhist (6%) and other religion (4%). It was observed that the majority of newborns were of 1st gravidity (42.86%) followed by 2nd gravida (37.14%)

Adedemy JD et al, studied the prevalence of glucose 6 phosphate dehydrogenase deficiency among 510 infants and children included in the study, 104 were born from consanguineous parents (20.4%). Among them, 68 were second degree relatives (65.4%) and 36 were first to second degree (34.6%).[4] In the present study the prevalence of G6PD deficiency among newborns was 0%.

Mukherjee MB et al, studied glucose-6-phosphate dehydrogenase (G6PD) deficiency among tribal populations of India and observed that the prevalence varied from 1.4 to 31.4 per cent in western India comprising of Rajasthan, Gujarat, Dadra and Nagar Haveli and Maharashtra.[2] Among the tribal groups studied, a very high frequency of G6PD deficiency was observed in the Gamits (31.4%), Dhankas (20.4%), Warlis (19.6%), Dhodias (17.8%), Bhils (16.3%) and Garasiyas (15.2%). Bhils from Nasik district of Maharashtra have a very low frequency (1.4%).

Paneliya CKB et al, studied the incidence of G6PD deficiency amongst the ethnic groups from January 2013 to January 2014 in cord blood of all babies born at Nazareth Hospital, Meghalaya; during the study period, G6PD deficiency was seen in 43 out of 2400 newborns (1.8%). G6PD deficiency was 1.9% in assamese newborns and 4% amongst Bihari newborns.[6]

In the present study, it was observed that the majority of newborns had history of drug intake of oxidant drug vitamin K (97.43%) followed by oxidant drug (Vitamin K) and Non-oxidant drug (Inj ampicillin, Inj gentamycin) (2%). Two babies were not given any drug because parents did not give consent. It was observed that the majority of mothers 341 (97.43%) had no history of oxidant drug intake while other 9 (2.57%) mothers had positive history of oxidant drug intake. Oxidant drug given or taken during pregnancy can trigger hemolysis in G6PD deficient fetus and newborns. The distribution of newborns according to hemoglobin levels showed that the majority of newborns had normal hemoglobin 275 (78.57%) while 63 newborns (18%) had anemia (Hb<17.1 gm%) while 12 newborns had polycythemia (Hb>20.9 gm%).

In a study by Adedemy JD et al, on prevalence of Glucose 6 Phosphate Dehydrogenase deficiency; anemia prevalence in the study population was 11.6%.[4]

In present study it was observed that the out of 350 newborns majority of newborns had no hyperbilirubinemia (99.14%) while 3 (0.86%) newborns had presence of hyperbilirubinemia.

In the study by Narang A et al (1997), out of 260 hyperbilirubinemic neonates 43 (16.5%) were found to be G6PD deficient and in the study by Dholakia A et al, (2012), found that out of 150 hyperbilirubinemic neonates, 16 (10.6%) were G6PD deficient.[7,8]

In the present study it was observed that, newborns with hyperbilirubinemia had mean hemoglobin levels of 14.8, 16.5 and 17.2 in baby 1, baby 2 and baby 3 respectively. The mean PCV levels were 43.3, 48 and 52.6 in baby 1, baby 2 and baby 3 respectively. The mean total leucocyte count was 6500, 10800 and 33800 in baby 1, baby 2 and baby 3 respectively. The mean platelet levels were 210000, 109000 and 180000 baby 1, baby 2 and baby 3 respectively. The mean reticulocyte count was 2.34, 3.12 and 2.93 in baby 1, baby 2 and baby 3 respectively. It was observed that, hyperbilirubinemic babies blood group were B^{+}ve, AB^{+}ve and B^{+}ve in baby 1, baby 2 and baby 3 respectively. Mother's blood groups were O^{-}ve, O^{+}ve and B^{+}ve of baby 1, baby 2 and baby 3 respectively. The DCT was positive in baby 1 and negative in other newborns. The urine hemoglobinuria was absent in all newborns with hyperbilirubinemia. It was observed that, mean total bilirubin levels were 24.8, 14.9 and 18.6 in baby 1, baby 2 and baby 3 respectively.

In a study by Paneliya CKB et al, showing relation between G6PD deficiency and neonatal jaundice

observed the mean total bilirubin levels was 18.3±3 mg/dL among G6PD deficient and 17.4±1.8 mg/dL among non G6PD deficient babies.[6] They concluded that there was a higher frequency of neonatal jaundice amongst babies who were G6PD deficient compared to non G6PD deficient babies, though the severity of jaundice was similar.

It was observed that the 3 newborns had hyperbilirubinemia (66.67%) within 48 hours of birth. The hyperbilirubinemia develops at 08, 12 and 42 hours of birth among newborns. In the present study, among 3 newborns with hyperbilirubinemia, Rh incompatibility, ABO incompatibility and unknown cause of jaundice was observed in 1 (33.33%) patient each respectively. In the 350 newborns studied, presence of Hyperbilirubinemia was more prevalent in birth weight <2500 grams but showed no statistically significant relation (P>0.05) between birth weight and bilirubin level.

Dholakia A et al, in their study reported that the ABO Incompatibility and Prematurely were associated with 32.6% and 30.6% G-6-PD deficiency neonates respectively.[8]

Mondal M et al, studied glucose-6-phosphate dehydrogenase deficiency in neonatal jaundice observed that out of a total of 176 newborns with hyperbilirubinemia, 138 newborns (78.40%) had birth weight of 2500 gm or more and 38 (21.60%) were less than 2500 gm.[9] Among those with birth weight ≥2500 gm., 19 babies (13.76%) had G6PD deficiency. The association of birth weight and hyperbilirubinemia was not significant. (P>0.05)

It was observed that there was no statistically significant relation between hemoglobin and hyperbilirubinemia (P>0.05). The distribution of newborns according to treatment with phototherapy in hyperbilirubinemia showed that all hyperbilirubinemic newborns were treated with phototherapy. In a study by Mondal M et al, in Sammilani Medical college during the period from November 2010 to December 2010, out of 176 newborns, the 24 neonates with G6PD deficiency required phototherapy for duration of more than 24 hours.[9] Two babies (8.33%) required phototherapy for a period of 24 to 48 hours. Eight (33.33%) needed phototherapy for 48 to 72 hours. It was observed that among 3 newborns who had hyperbilirubinemia;1(33.33%) newborn required exchanges transfusion in addition to phototherapy.

In a study by Mondal M et al, among 176 babies presented with early neonatal jaundice 23 required double volume exchange transfusion (one required exchange transfusion twice). 11(45.83%) neonates in the G6PD deficient group required exchange transfusion.[9]

In the study by Kuruvilla et al, only one out of 25 G6PD deficient baby (n=212) required an exchange transfusion; in all other cases jaundice improved with phototherapy alone.[10]

G6PD deficiency is never total in humans, it is embryonically lethal. Women who are heterozygous (Gd+/Gd-) carriers of severely deficient allels, can have almost normal G6PD levels due to a selective advantage of cells expressing the normal allele. According to Minucci et al, most of the G6PD mutations are asymptomatic and the total number of mutations could be probably much higher than that reported.[11] Minucci et al, opined that large population screening studies will determine, in the future, the exact prevalence of all G6PD mutations in the general population.[11]

There is also need for a large screening programmed, especially in malaria endemic zones, where due to natural selection of population, there seems to be a higher incidence of glucose 6 phosphate dehydrogenase enzyme deficiency. Hence, the study helps to start screening program all over state and prevent recurrent hemolysis and anemia in neonates and children. This will help in decreased admission for blood transfusion and leads to decrease morbidity and mortality among neonates and children. The present study helps to educate the parents about proper care and prevention among newborns of G6PD deficiency.

CONCLUSION

The present study had no G6PD deficient new-born. Early detection and prevention are the key strategy for the successful management and control of G6PD deficiency. If the clinical and hematological findings raise the suspicion of glucose 6 phosphate dehydrogenase deficiency, the disorder should be confirmed by quantitative spectrophotometric measurement of red blood cell enzyme activity. G6PD deficiency is one of the major causes of neonatal jaundice within 24 hours of life in new-borns. Hence, neonatal screening for G6PD deficiency could be an alternative to the hemolytic crisis prevention strategy in order to optimize affected young child care and prevention of crisis occurrence by avoiding taking contraindicated foods and drugs.

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