



## Study of the prevalence of G6PD deficiency in the RBCs of new borns

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### ABSTRACT

This study we attempt to search the need for a newborn screening program for G6PD deficiency because of high prevalence in some areas and no such study was done in part of Maharashtra. 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. The current study was a retrospective cross-sectional study conducted to determine the prevalence of G6PD deficiency in Red Blood Cells of born live neonates in tertiary care hospital. The research was performed at a tertiary care hospital with the 350 newborns, showing the age distribution of newborns revealed that the highest number of newborns was in the age group of 0-10 hours (96.29 percent). Of the 350 newborns, 181 (51.71 percent) were females and 169 (48.29 percent) were males and the ratio of females to males was 1.07:1. Many newborns had between 2500-3000 g (52.86 percent) of birth weight led by > 3000 g (28 percent) and < 2500 g (19.14 percent). The majority of newborns were Hindu by religion (67.14%) followed by Muslims (22.86%), Buddhists (6%), as well as other religions (4%). Most of the newborns came from 1st gravida (42.86%) and led by 2nd gravida (37.14%)



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## INTRODUCTION

### Glucose-6-phosphate dehydrogenase

G6PD (MIM 00305900) (EC 1.1.1.49) is an important housekeeping cytosolic enzyme present in erythrocytes, involved in the glycolytic pathway for glucose metabolism. It precipitates the first stage of the pentose phosphate pathway and transforms glucose-6-phosphate (G6P) to 6-phosphogluconate, coupled with lack of the co-factor Nicotinamide

adenine dinucleotide phosphate (NADP) to Nicotinamide adenine dinucleotide hydrogen phosphate (NADPH) (Arese and Flora, 1990).

The human G6PD monomer is a compound of 515 amino acids with a molecular weight of roughly 59 kilodaltons (kDa). The enzyme is found in both dimeric and tetrameric forms in RBCs. The dimeric type is prevalent at physiological pH. Every monomer has two domains: one, the N-terminal domain (residues 27–200) with a dinucleotide binding site (residues 38–44), and the other with a nine-barrel anti-parallel sheet. Both the domains are connected to the  $\alpha$ -helix comprising the retained eight-residue base molecule (residues 198–206) (Arese and Flora, 1990).

In 1986, cloned and sequenced G6PD enzyme. (Chen *et al.*, 1991) Above around 450 G6PD types were distinguished on the bases of enzyme kinetics, physicochemical attributes as well as other parameters. About 300 versions have been identified by the (WHO, 1967).

Parents of these babies can then be advised about

proper precautions and care to be taken and prevent the child from exposure of certain medicine such as sulfonamides, cotrimaxazole, chlomphenicol, nitrofurantoin, dapson, nalidixic acid; Antimalarials: such as primaquine, pamaquine, chloroquine, quinacrine; antihelmintics ; chemical : such as benzene, naphthalene (moth balls), vitamin k analogues, dimercaprol, phenylhydrazine; illness : such as diabetic ketoacidosis, hepatitis and sepsis Figures 1 and 2.

**Aims**

1. To research the incidence of G6PD deficiencies in RBCs of live newborns delivered in tertiary care hospitals.
2. To study how many G6PD deficiency babies have hyperbilirubinemia and require intensive phototherapy and/or exchange transfusion in early neonatal period.

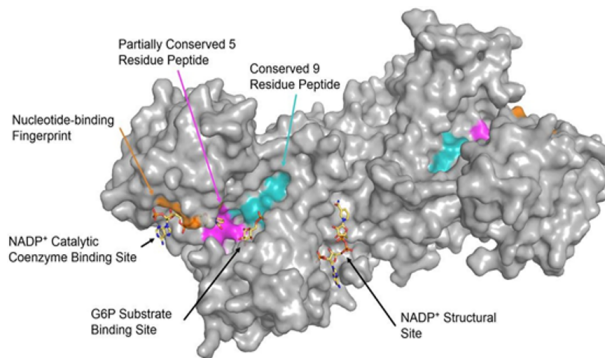
**Objectives of study**

1. In this tertiary care hospital, the prevalence of G6pd deficiency in the RBCs of live newborn babies is not known. This study is being carried out in this tertiary care hospital for the first time.
2. The babies who are found to have G6PD deficiency will be advised precautions regarding use of drugs (vitamin k), infection and other stressful situation to prevent hemolytic episodes.
3. Parental counseling will be done before and after the test

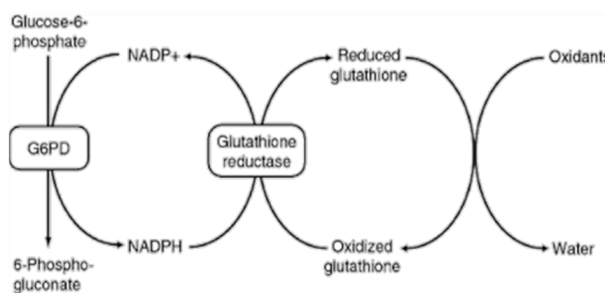
**Review of literature**

**Deficiency of Glucose-6-phosphate dehydrogenase**

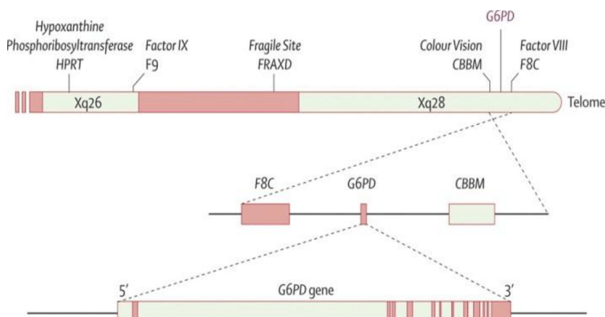
G6PD deficiency serves as an epitome of the various human enzymopathies identified currently. The earliest suspected reports of G6PD deficiency are from Pythagoras forbidding his students to eat favabeans (*Vicia fab*). (Howes *et al.*, 2013) Till the nineteen fifties, only three enzyme deficiencies were recognised in erythrocytes, namely Catalase, G6PD and Galactose-1-phosphate uridyltransferase. Out of these, only G6PD deficiency leads to haematological disorders, primarily haemolytic anaemia, that has brought this common deficiency into light. Today, deficiency of G6PD is the most severe clinically significant, hereditary erythroenzymopathy, present in approximately 400 million people throughout the world (Cappellini and Fiorelli, 2008).



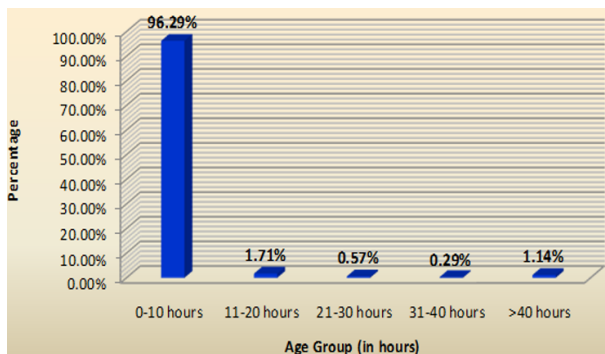
**Figure 1: G6PD Enzyme with Its Binding Sites**



**Figure 2: Pathway of G6PD and Glutathione Metabolism**



**Figure 3: Location of G6PD Gene (Gd)**



**Figure 4: Age distribution**

**Table 1: Percentage of G6PD deficiency in India**

Study	State	Percentage of G6PD deficiency
(Baxi <i>et al.</i> , 1963)	Maharashtra	13.64%
Baxi <i>et al.</i> (1961)	Maharashtra	15.74%
(Chatterjea, 1966)	India	4.62%
(Deshmukh and Shamra, 1968)	Maharashtra,	10%
(Kate <i>et al.</i> , 1978)	Maharashtra, West Bengal	7.55%
(Ghosh <i>et al.</i> , 1981)	West Bengal	4.28%
(Saha <i>et al.</i> , 1987)	West Bengal	0%
(Chhotray and Ranjit, 1990)	Orissa	13.20%
(Verma <i>et al.</i> , 1990)	Punjab	3.9%
(Jain, 1992)	Rajasthan	1.80%
(Kaeda <i>et al.</i> , 1995)	Madhya Pradesh	4.18%
(Kuruvilla <i>et al.</i> , 1998)	India	11.79%
(Joshi <i>et al.</i> , 2001)	Western India	22.59%
(Murhekar <i>et al.</i> , 2001)	Andaman and Nicobar Islands	3.45%
(Sukumar <i>et al.</i> , 2004)	India	10.49%
(Santhi and Sachdeva, 2004)	Haryana	9.56%
(Balgir, 2006)	Orissa	13.68%
(Gupte <i>et al.</i> , 2005)	Gujarat	21.78%
(Saraswathy and Aggarwal, 2005)	New Delhi	2.72%
(Dash <i>et al.</i> , 2005)	Mizoram	17.55%

**Table 2: Newborns distribution according to Religion**

Religion	No. of new born	Percentage
Hindu	235	67.14
Muslim	80	22.86
Buddhist	21	06.00
Others	14	04.00
Total	350	100

**Table 3: Distribution according to anemia**

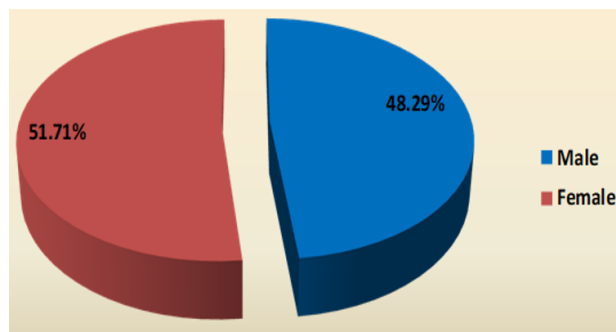
Hemoglobin (Hb in gm%)	No. of new born	Percentage
<17.1	63	18.00
17.1- 20.9	275	78.57
>20.9	12	3.43
Total	350	100

**Table 4: Distribution according to presence of Hyperbilirubinemia**

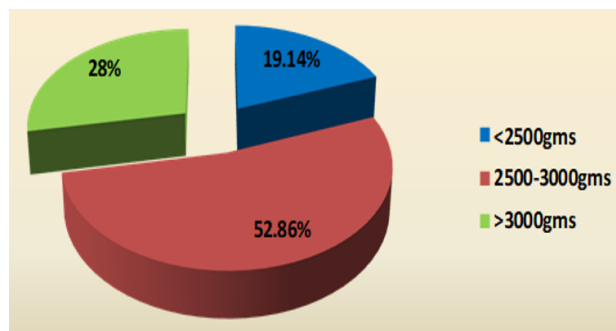
Hyperbilirubinemia	No. of new born	Percentage
Present	03	00.86
Absent	347	99.14
Total	350	100

**Table 5: Investigations in hyperbilirubinemic newborns who presented with jaundice within 48 hours of life**

Newborns with hyperbilirubinemia Within 48 hrs of life	Hemoglobin (in gm%)	Packed Cell volume	Total leucocyte count	Platelets	Reticcount
Baby 1	16.5	43.3	6500	210000	2.34
Baby2	14.8	48	10800	109000	3.12
Baby3	17.2	52.6	33800	180000	2.93



**Figure 5: Sex Distribution**



**Figure 6: Birth weight among newborns**

**Occurrence of G6PD deficiency in India**

(Baxi *et al.*, 1963) in the year 1963 reported the first deficiency of G6PD in the India occurrence from the Parsi populace of Mumbai.

(Mukherjee, 2017) in year 2017, investigated the prevalence of G6PD deficiency in Eastern India’ population. Total 250 patients (186 male and 64 female) were included in this study. They were subsequently categorized into various subgroups and analysed.

Quantitative estimation of G6PD enzyme was done with the help of commercially available kit. It is a sigma process and a modification of Kornberg and Horecker and Löhr and Waller’s spectrophotometric methods. It involves measurement of the rate of increase in reduction of NADP to NADPH and its spectrophotometric absorbance at 340 nm serves to quantify enzymatic activity. Deficiency of G6PD was found in overall 10 % of the populace in this part of

the country. Prevalence was higher in tribal community compared to non tribal community.

**G6PD Gene (Gd)**

Gd is around 18 kb long. The gene is located on the distal long arm of the X- (locus q28) and comprises 13 exons and 12 introns, the length of which ranges from 12 bp to 236 bp. The first exon does not have a coding chain, and the intron between exons 2 and 3 is incredibly long, stretching to 9,857bp. The sequence of the whole gene has been investigated and compiled. (Martini *et al.*, 1986), Figure 3.

**Deficiency of G6PD diagnosis**

**Deficiency of G6PD screening programmes for neonates:**

A number of screening programs are used to identify G6PD deficient patients which cannot be identified by routine observations and physical examinations. Screening test methods are extremely helpful for the processing of testing vast quantities. Clinical and biochemical aspects of neonatal screening are essential for successful diagnosis and subsequently management. To identify the affected infants before the development of clinical signs and reducing morbidity correlated with deficiency of G6PD, newborn screening is recommended (Beutler, 1994).

Although a cure does not exist for this condition, recognizing the presence of G6PD deficiency is a significant contributor to reduction in newborn mortality and morbidity and prophylactic avoidance of triggers in adults. This is enough of an impetus to start preventive measures through screening programmes.

(Khneisser *et al.*, 2007) reported significant decrease (95%) in G6PD deficiency related hospitalization rate in screened Lebanese newborn males as compared to those unscreened.

**Association of G6PD deficiency with other diseases**

The strong incidence of G6PD polymorphic variants in many racial groups and cultures raises the risk of interaction with certain pathological disorders.

Hemoglobinopathies such as Sickle Cell Disease (SCD) and Thalassemia is usually observed associated with the deficiency G6PD. G6PD impaired people with Gilbert's Syndrome are more prone to develop neonatal jaundice and may suffer hyperbilirubinemia later on in life (Kaplan and Hammerman, 2000).

Various other disorders have also been speculated to be associated with G6PD deficiency.

1. Significant number of patients with gallstones exhibit G6PD deficiency with one or more hemolytic episodes.
2. In another study, primaquine-sensitive black people exhibited significantly higher cholesterol level compared with a control groups. It was also noted that the level of esterified cholesterol in serum fell sharply during acute drug induced hemolysis in the G6PD deficient males, although it remained unchanged in the normal subjects (Chen *et al.*, 1991).
3. Severely G6PD deficiency can contribute to insufficient leukocyte activity. resulting in chronic granulomatous disease. Reduced NADPH oxidase function was found in granulocytes, leading to impaired NET formation. Defective NET (neutrophil extracellular trap) formation has thus far been only observed in patients with the NADPH oxidase deficiency chronic granulomatous disease, who require antibiotic and antimycotic prophylaxis to prevent life-threatening bacterial and fungal infection.
4. Reports indicate predisposition of G6PD deficiency patients to a higher risk of hypertension.
5. In non hematologic tissues, low G6PD levels may be important in the development of cataracts.
6. Heyman, showed a significantly increased association between prevalence of diabetes in the 45–65 year-old age-group among patients with G6PD deficiency.
7. Higher incidence of toxoplasmosis has been recorded in child with deficiency of G6PD in Oman.
8. G6PD deficiency has association with diseases of Angiotensin- converting enzyme (ACE) gene.
9. Reduced G6PD levels in patients of vitiligo.

10. A variety of human tumors (breast, prostate, colon and stomach) in individuals who are not G6PD-deficient, it is apparent that G6PD activity in malignant cells tends to be higher relative to benign cells. This phenomenon has been known for 30 years, and is most probably related to a higher rate of cell division.

11. G6PD deficiency may contribute some protection against certain cancers.

12. It has also been reported that mortality due to cardiovascular diseases reduces in G6PD deficient patients.

13. It has also been suggested that deficiency of G6PD poses some

### Advisory and regulatory consideration for G6PD deficient patients

Avoiding oxidative stressors is the primary therapy for G6PD deficiency patients like the Precipitating drugs, broad beans, naphthalene - found in moth-balls and henna etc triggering /inducing the hemolysis. Although G6PD associated hemolysis usually is short-lived, most of these authors have emphasized on diagnosis of the trigger, proper control and early treatment in neonatal jaundice as very important (Mehta *et al.*, 2000).

(Youngster *et al.*, 2010), while worrying about the conflicting advices to the patients, causing uncertainty and crisis due to lack of consensus between physicians about the medication in the event of G6PD deficiency related haemolytic anemia, in such cases it was concluded to prohibit only seven of the currently used drugs: dapsone, nitrofurantoin, toloumchloride (toluidine blue), rasburicase, primaquine, methylthionium chloride (methylene blue) and phenazopyridine. A very detailed list of drugs or triggers to be avoided in G6PD deficiency cases has been provided.

(Elyassi and Rowshan, 2009) opined that in the management for pain and anxiety in G6PD deficient patients, medications for eg, benzodiazepines, codeine/codeine derivatives, propofol, morphine and ketamine are secure and have not been proven to induce hemolytic seizures, could be used.

### MATERIALS AND METHODS

This study was conducted in maternity wards and NICU of Krishna Institute of medical sciences and hospital and research centre, Karad, Maharashtra.

### Study population

350 consecutively born live newborns, born at tertiary care hospital.

**Study design**

Prospective cross-sectional study

**Sample size**

350 consecutively born live newborns in tertiary care hospital during two years period.

Formula applied for calculation of sample size  $n=4pq/l^2$  Where  $p=4.5\%$ ,  $q=95.5\%$ ,  $l= 2.5\%$  error  $(4*4.5*95.5)/(2.5*2.5) =276$

Minimum 276 newborns should be studied.

**Duration of study**

From October 2016 to March 2018

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.

**Need of the study**

1. Deficiency G6PD is transferred from one or both of the parents' genes to the child's gene.
2. These children cannot be identified by routine observation and clinical examination. Only screening can help to identify these children.
3. G6PD deficiency is the major cause of acute hemolysis in children. Only early detection and precaution can save mortality and morbidity in children.
4. Early detection can also avoid recurrent blood transfusion and kernicterus in newborns.
5. Many countries have also established and included screening programme for G6PD and it is cost effective as well.

**Sample collection**

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.

2 ml of whole blood sample was collected by intravenous route in EDTA bulb for analysis of qualitative G6PD estimation by decolourisation of 2,6 dichlorophenol indophenols.

Blood is collected of newborn babies within 48 hours of life.

Babies who came G6PD positive were further screened for hyperbilirubinemia and other investigations done were

1. CBC with Retic count
2. Peripheral smear with supravital stain
3. Bilirubin total, direct and indirect
4. Liver function test
5. Direct coombs test
6. Urine routine and microscopy

**OBSERVATIONS AND RESULT**

The Figure 4 shows age wise distribution of newborns. The average number of newborns in the age group was 0–10 hours (96.29 percent), followed by 11–20 hours (1.71 percent). The average age of newborns was  $2.86 \pm 5.83$  hours.

The Figure 5 shows gender wise distribution of newborns. In 350 cases the the number of women were 181 (51.71 percent) and male were 169 (48.29 percent) and the ratio of female to male was 1.07 : 1.

The Figure 6 demonstrate the dispensation of new births child in accordance of birth weight. It was found that the birth weight of newborns was 2500–3000 g (52.86 percent), followed by > 3000 g (28 percent) and < 2500 g (19.14 per cent).

The Tables 1 and 2 shows newborns distribution according to religion. It was reported that the majority of newborns were Hindus by religion (67.14 per cent) followed by Muslims (22.86 per cent), 6 percent of newborns were Buddhists and 4 percent of other religions.

The Table 3 shows distribution of newborns according to haemoglobin levels. It was found that the majority of newborns had natural hemoglobin at 275 (78.57 per cent) and 63 newborns (18 per cent) had anemia. ( $Hb < 17.1$  gm%) although 12 newborns had polycythemia ( $Hb > 20.9$ ). As per Nelson textbook, describing the natural amount of hemoglobin in newborns 1-2 days of life as  $19 \pm 1.9$  gm/dl.

The Table 4 shows distribution of newborns in accordance to presence of hyperbilirubinemia. Hyperbilirubinemia leads to elevated bilirubin levels in the blood and is distinguished by anaemia, yellowish skin discoloration, sclera, mucous membranes, and nails for that particular birthweight and day of life according to Bhutani nomogram. It was found that the majority of newborns had no hyperbilirubinemia (99.14 percent) whereas the

incidence of hyperbilirubinemia was present in 3 newborns (0.86 percent).

The above Table 5 shows investigations in newborns with hyperbilirubinemia. It was observed that, mean hemoglobin levels were 16.5, 14.8 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 per cumm in baby 1, baby 2 and baby 3 respectively. The mean platelet levels were 210000, 109000 and 180000 per cumm in baby 1, baby 2 and baby 3 respectively. The mean reticulocyte count percentage was 2.34, 3.12 and 2.93 in baby 1, baby 2 and baby 3 respectively.

## CONCLUSIONS

The present study had no G6PD deficient newborn. Effective diagnosis and avoidance is the main approach for effective treatment and maintenance of G6PD deficiency. If the clinical and hematological results indicate deficiency of G6PD, quantitative spectrophotometric analysis of the function of the RBCs enzyme should be verified. Deficiency of G6PD is the main causes of neonatal jaundice within 24 hours of conception. Therefore, neonatal screening for G6PD impairment may be an addition to the hemolytic crisis management approach for improving impacted young children's treatment and disaster reduction by eliminating contraindicated food and medications.

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Nil

## Conflict of Interest

I hereby declare that there is no conflict of interest related to this manuscript.

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