

# Screening of G6PD Deficiency in Newborns at a Tertiary Care Teaching Hospital in Assam, North East India: A Cross-sectional Study

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## ABSTRACT

**Introduction:** Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency is the most frequently encountered enzymopathy in humans. It is closely linked to neonatal jaundice, chronic non-spherocytic haemolytic anaemia, and acute haemolytic anaemia. G6PD deficiency is a common cause of jaundice among neonates.

**Aim:** To screen for G6PD deficiency among newborns at a tertiary care teaching centre in Assam.

**Materials and Methods:** This hospital-based cross-sectional study was conducted in the postnatal ward of the Department of Paediatrics at Medical College and Hospital, Dibrugarh, Assam, India, from June 2021 to May 2022. A randomly selected sample of 630 term neonates aged between 24 hours and 7 days of life was included in the study. A 2 mL blood sample was collected in an Ethylenediamine Tetra-acetic Acid (EDTA) vial from each

neonate, and G6PD enzyme activity was estimated using the Kinetic Method with a commercially available G-six kit. Newborns with G6PD enzyme activity values less than 6.4 U/g Haemoglobin (Hb) were considered G6PD deficient. Data, including age, sex, religion, Total Serum Bilirubin (TSB/NBIL), G6PD activity, and Hb of the neonates, were entered into pre-designed forms. The data were analysed using the Chi-square test.

**Results:** Among the 630 screened neonates, 48 were found to be G6PD deficient, of which the majority were males (34 males, 14 females), resulting in a male-to-female ratio of 2.4:1. The occurrence of G6PD deficiency was 7.62%. The mean G6PD enzyme activity in deficient neonates was  $4.53 \pm 1.17$  U/g Hb.

**Conclusion:** This study identified a significant occurrence of G6PD deficiency in newborns, including females. The mean G6PD enzyme activity in deficient neonates was significantly lower than in normal cases.

**Keywords:** Enzyme activity, Glucose-6-phosphate dehydrogenase deficiency, Jaundice, Neonate

## INTRODUCTION

G6PD deficiency (an X-linked disorder) is the most frequently encountered enzymopathy in humans, affecting approximately 400 million individuals extensively around the globe [1]. It is closely associated with three paramount clinical syndromes: neonatal jaundice, chronic non-spherocytic haemolytic anaemia, and acute haemolytic anaemia. G6PD deficiency is a frequent cause of jaundice among neonates. About 5% of neonates with G6PD deficiency experience jaundice within the first 24 hours of life, and their serum indirect bilirubin peaks between day 3 and day 5, frequently exceeding 20 mg/dL [2].

When jaundice first appears at the end of the first week, the peak may not occur until the second week. Early detection of G6PD deficiency is crucial because if left untreated, it can result in severe haemolysis and anaemia or dreaded complications of neonatal jaundice, such as kernicterus in newborns [2].

G6PD deficiency is an inherited disorder in which the body cannot produce enough G6PD enzyme, which is essential for the normal functioning of red blood cells. G6PD deficiency can be a root cause of haemolytic anaemia, typically occurring after the consumption of fava beans, medications, or infections. Chronic anaemia can occur in certain rare cases. A neonate with G6PD deficiency can live a healthy and robust life with the acquisition of accurate precautionary measures. It has been estimated that approximately 80% of preterm babies and 60% of term babies experience jaundice to a certain extent during the initial week of birth. However, the jaundice arising from G6PD deficiency appears on the first day of birth and is typically of tremendous pathological significance [3].

Detection of G6PD deficiency via neonatal screening is practicable and cost-efficient. It enables the administration of preliminary precautionary actions against grievous haemolysis, kernicterus, jaundice, etc., in the initial phase after birth (neonatal life) as well as other preventative considerations in subsequent phases of life. A practical guidance form can be provided to the family members of G6PD-deficient infants, recommending selective intake of food items and exclusion of certain chemicals and drugs if G6PD deficiency status is detected at the earliest opportunity [4].

Studies conducted by Pao M et al., Goyal M et al., and Iranpour R et al., found varying prevalence of G6PD deficiency among different parts of the world [5-7]. No such study was previously conducted in this part of Northeast India to determine the burden of G6PD deficiency among newborns. Therefore, this study plays a pivotal role in estimating the prevalence of G6PD deficiency among neonates in this part of the country and providing proper advice regarding food and drugs to be avoided to prevent haemolysis in affected children.

Hence, the present study was conducted to screen for G6PD deficiency in newborns at a tertiary care centre in Assam.

## MATERIALS AND METHODS

This hospital-based cross-sectional study was conducted in the postnatal ward of the Department of Paediatrics at Assam Medical College and Hospital, Dibrugarh, Assam. The study spanned a duration of one year from June 2021 to May 2022. Ethical clearance was obtained from the Institutional Ethics Committee (Approval No. AMC/EC/PG5685). Informed and written consent was obtained from the parents/guardians after a proper explanation of the study.

**Inclusion criteria:** The study population consisted of term neonates aged between 24 hours and day 7 of life.

**Exclusion criteria:** Neonates with congenital malformations, those requiring admission to the Neonatal Intensive Care Unit due to pathological causes other than neonatal jaundice alone, and neonates whose parents denied consent were excluded from the study.

**Sample size:** The sample size for the study was calculated using the formula  $n = z^2 pq / d^2$  ( $z = 1.96$ ;  $p = 0.015$ ,  $q = 1 - p$ ,  $d = 0.01$ ), considering a 95% confidence interval with an absolute precision of 1% and assuming that 1.5% of newborns have G6-PD deficiency, based on the study conducted by Goyal M et al. The calculated sample size was 568 [6]. Accounting for a non-response rate of 10%, the sample size was rounded off to 630 ( $568 + 10\%$  of  $568 = 568 + 57 = 625 \approx 630$ ).

## Procedure

**Data collection:** After enrolling the case, a detailed history and clinical examination of the neonate were performed. The findings were recorded in the pre-designed proforma. Samples for estimating G6PD activity were collected and tested. Hb and NBIL, along with fractions, were also tested for the cases. If the test came back positive, the results were informed to the parents, and a list of drugs to be avoided was handed to them.

**Sample collection:** Approximately 2 mL of blood sample was collected from each neonate from the peripheral veins on the dorsum of the hand, following proper aseptic and antiseptic measures. The sample was collected in an EDTA vial, and gentle inversion was done several times to prevent clot formation. The samples were then used within one hour of collection.

**Estimation of G6PD activity:** G6PD was assessed using the Kinetic Method [8] (Coral Clinical Systems, a division of Tulip Diagnostics (P) Ltd.). One mL of G6PD working reagent (L1) was mixed with 0.01 mL of whole blood and incubated for 5-10 minutes at room temperature. Then, 2 mL of starter reagent was added, mixed well, and incubated for five minutes at 30°C/37°C. The initial absorbance (A) was read, and the absorbance reading was repeated after 1, 2, and 3 minutes. The mean absorbance change per minute ( $\Delta A/\text{min}$ ) was calculated.

If G6PDH activity is very low, the per-minute change in absorbance rate will be very low. In such cases, another absorbance (A2) is taken exactly five minutes later after taking the initial absorbance (A1), and the mean absorbance per minute ( $\Delta A/\text{min}$ ) is calculated.

$$\Delta A/\text{min} = \frac{A_2 - A_1}{5}$$

## Calculation:

$$\text{G6PD activity (U/gHb)} = \Delta A \times \frac{4778}{\text{Hb (g/dL)}}$$

The reference range for normal G6PD activity is 6.4-18.7 U/g Hb [8].

Estimation of Total Serum Bilirubin (TSB/NBIL), unconjugated bilirubin (Bu), and conjugated bilirubin (Bc) was done using the end-point colorimetric [9] (dual wavelength) method with in-vitro BuBc Slides. The reference intervals for Bu, Bc, and TSB/NBIL in neonates are 0.6-10.5, 0-0.6, and 1.0-10.5, respectively [9].

Estimation of Haemoglobin (Hb) was done using the Sodium Lauryl Sulphate (SLS) detection method in Sysmex XN 550 [10]. The normal reference value for Hb levels in neonates is 14.0-24.0 g/dL [11].

## STATISTICAL ANALYSIS

A Microsoft Excel worksheet was used to systemise the data collected in the investigation. Computer-based analysis was conducted using

the Statistical Package for Social Sciences (SPSS) version 20.0 software. The categorical values were summarised as percentages and proportions. Results for continuous measurements are presented as mean  $\pm$  standard deviation and compared using a student t-test. Discrete data are expressed as number (%) and analysed using the Chi-square test and Fisher's exact test. For all analyses, the statistical significance was set at a 5% level ( $p$ -value  $< 0.05$ ).

## RESULTS

In the present study, out of the 630 cases, 48 (7.62%) were tested G6PD deficient [Table/Fig-1].

Category	Number of cases (n)	Percentage (%)
G6PD deficient	48	7.62
Non G6PD deficient	582	92.38

**[Table/Fig-1]:** Prevalence of Glucose 6 Phosphate Dehydrogenase (G6PD) deficiency among study population.  
p-value- chi-square test

Among the 48 G6PD deficient cases, 34 (70.83%) were male and 14 (29.17%) were female. A total of 286 (49.14%) non-deficient cases were male, and 296 (50.86%) were female. The male-to-female ratio in G6PD deficient cases was 2.4:1.

Out of the 48 G6PD deficient cases, 36 (75%) had neonatal jaundice. Among the 582 non-deficient cases, 72 (12.37%) had neonatal jaundice ( $p < 0.05$ ). Thus, Neonatal Jaundice (NNJ) was significantly more common in G6PD deficient cases than in non-deficient cases [Table/Fig-2].

Parameters	Deficient (N=48) n (%)	Non deficient (N=582) n (%)	p-value
<b>Gender</b>			
Male	34 (70.83)	286 (49.14)	<b>0.0039</b>
Female	14 (29.17)	296 (50.86)	
<b>Religion</b>			
Hindu	42 (87.50)	552 (94.84)	<b>0.0263</b>
Muslim	2 (4.17)	18 (3.09)	
Christian	4 (8.33)	12 (2.07)	
<b>NNJ status</b>			
Present	36 (75)	72 (12.37)	<b>&lt;0.05</b>
Absent	12 (25)	510 (87.63)	

**[Table/Fig-2]:** Demographic profile and Neonatal Jaundice (NNJ) status in G6PD deficient and non-deficient cases.

G6PD activity was significantly lower in the G6PD deficient cases ( $4.53 \pm 1.17$  U/g Hb) than in the non-deficient cases ( $15.36 \pm 4.01$  U/g Hb). Additionally, the mean TSB/NBIL level of G6PD deficient cases ( $10.49 \pm 9.02$  mg/dL) was significantly higher than that of non-deficient cases ( $4.66 \pm 4.29$  mg/dL). The study also observed that the mean serum Bu level of G6PD deficient cases was ( $10.40 \pm 9.08$ ) mg/dL, while that of non-deficient cases was ( $4.65 \pm 4.29$ ) mg/dL [Table/Fig-3].

Parameters	Deficient	Non deficient	p-value
G6PD activity (U/g Hb)	$4.53 \pm 1.17$	$15.36 \pm 4.01$	<b>0.0001</b>
Mean TSB level (mg/dL)	$10.49 \pm 9.02$	$4.66 \pm 4.29$	<b>0.0001</b>
Mean unconjugated Bilirubin (Bu)	$10.40 \pm 9.08$	$4.65 \pm 4.29$	<b>0.0001</b>
Conjugated Bilirubin (Bc)	$0.03 \pm 0.03$	$0.03 \pm 0.06$	0.4837
Haemoglobin (Hb)	$13.62 \pm 1.28$	$16.87 \pm 2.63$	<b>0.0001</b>

**[Table/Fig-3]:** Mean G6PD activity, mean Total Serum Bilirubin (TSB) level and mean unconjugated Bilirubin (Bu) level among deficient and non-deficient group.

## DISCUSSION

In this study, out of 630 cases, 48 neonates were detected to have G6PD deficiency, which accounts for 7.62% of the total. According to a study conducted by Goyal M et al., in a hospital in New Delhi, India [6], the occurrence of G6PD deficiency among the study subjects was found to be 1.5%. Another study conducted in New Delhi, India by Pao M et al., included 2,497 neonates, out of which 50 were found to be G6PD deficient, resulting in an incidence of 2.0% [5]. In the study conducted by Verma M et al., in Punjab, India, 1,000 babies were screened for G6PD deficiency, and 39 cases (3.9%) were found to be G6PD deficient [12]. Another study conducted by Boskabadi H et al., found that the occurrence of G6PD deficiency among 1,139 jaundice babies was 5.2% [13].

According to the study conducted by Kosaryan M et al., G6PD enzyme deficiency was found in 6.1% of the newborns [14]. Iranpour R et al., conducted a study in Isfahan, Iran, in which 2,560 newborns were enrolled, and the incidence of G6PD deficiency was found to be 3.2% [7]. Elella SA et al., conducted a cross-sectional study in Egypt and found that G6PD deficiency was present in 4.3% of the total newborns enrolled in the study [15]. However, in a descriptive observational study conducted by Bisoi S et al., in West Bengal, 16 (14.67%) out of 109 newborns were found to be G6PD deficient, which was much higher than the previously mentioned studies [16]. Pahlavanzadeh M et al., conducted a study in Iran, showing a similar result of a high occurrence of G6PD deficiency (18.1%) [2].

In this study, the majority (75%) of G6PD-deficient neonates had neonatal jaundice during their hospital stay. Out of all the icteric neonates found in this study, 33.33% were G6PD deficient. In a study conducted by Pao M et al., the incidence of hyperbilirubinemia among G6PD-deficient neonates was 32% [5]. Verma M et al., conducted a study in Punjab, India, where 48.7% of G6PD-deficient neonates presented with neonatal hyperbilirubinemia [12]. A similar result was found in another study conducted by Bisoi S et al., where 5 (31.25%) out of 16 G6PD-deficient newborns had hyperbilirubinemia, and 23.8% of the babies who developed severe jaundice were G6PD deficient [16]. In contrast to these studies, the present study shows an increased incidence of hyperbilirubinemia among G6PD-deficient newborns. However, in a study conducted by Iranpour R et al., in Iran, it was found that 7.5% of neonates with significant jaundice were G6PD deficient [7].

In this study, out of the 48 G6PD-deficient cases, 70.83% were males, and 29.17% were females. Thus, a male preponderance was seen among the deficient cases with a male-to-female ratio (M:F) of 2.4:1. A similar male preponderance was found in the study conducted by Iranpour R et al., where 67 (84.8%) cases were males, and 12 (15.2%) cases were females out of 79 G6PD-deficient cases,

with a male-to-female ratio of 5.5:1 [7]. Another study conducted by Elella SA et al., reported that 76.5% of the G6PD-deficient cases were males, and 23.5% were females, with a male-to-female ratio of 3.2:1 [15]. In the study conducted in West Bengal by Bisoi S et al., out of 16 G6PD-deficient cases, 10 (62.5%) cases were male, and 6 (37.5%) cases were female, with a male-to-female ratio of 1.6:1 [16]. Pahlavanzadeh M et al., reported a study in which out of the G6PD-deficient neonates, 78.9% were male, and 21.1% were female, with a male-to-female ratio of 3.7:1 [2].

However, a study conducted in Karnataka, India by Ramadevi R et al., revealed that out of 405 deficient cases, 198 (48.8%) were male and 207 (51.1%) were female, resulting in a male-to-female ratio of 0.9:1 [17]. Despite G6PD deficiency being an X-linked recessive disease that commonly occurs in males, the authors found female individuals with the deficiency, likely due to homozygous females or random X inactivation. This corresponds to a 25% occurrence of homozygous (affected) female children, 25% heterozygous (carrier) female children, 25% heterozygous (affected) males, and 25% normal male children when the father is G6PD deficient (affected) and the mother is a heterozygous (carrier) of G6PD deficiency.

The study reported a mean G6PD enzyme activity of (4.53±1.17) U/g Hb in G6PD deficient cases, whereas non-deficient cases exhibited a mean G6PD activity of (15.36±4.01) U/g Hb. This significant difference indicates lower G6PD activity in G6PD deficient cases compared to non-deficient cases. Similar findings were reported in a study by Iranpour R et al., where deficient patients had a mean G6PD enzyme activity of 3.22±1.8 U/g Hb, while normal G6PD patients showed a mean activity of 35.12±12 U/g Hb [7]. Another study conducted by Kosaryan M et al., in Iran reported a mean G6PD enzyme activity of 3.22±1.8 U/g Hb among G6PD deficient newborns [14].

Furthermore, the study found that the mean TSB/NBIL level (total serum bilirubin to non-bilirubin-bound immunoglobulin level) in G6PD deficient cases was (10.49±9.02) mg/dL, whereas non-deficient cases had a mean TSB/NBIL level of (4.66±4.29) mg/dL. This significant difference indicates higher TSB/NBIL levels in G6PD deficient cases compared to non-deficient cases. A study conducted by Pahlavanzadeh M et al., reported that the mean serum total bilirubin at hospitalisation was 18.43 mg/dL for G6PD deficient neonates and 11.86 mg/dL for normal G6PD neonates, showing a significant difference [2].

Thus, most of the studies discussed above (as shown in [Table/ Fig-4]) support the findings of the present study that G6PD enzyme activity in G6PD deficient neonates is significantly lower than in non-deficient cases [2,5-7,12-17]. The present study also confirms the common finding that G6PD deficiency is more prevalent in males.

	Place and year of the study	Sample size	Occurrence/ Incidence of G6PD	Prevalence of hyperbilirubinemia among G6PD deficient	Sex distribution (M=male; F=female)	Male: Female ratio	Mean G6PD enzyme activity in G6PD deficient (U/g Hb)	Mean TSB level in G6PD deficient (mg/dL)
Present study	AMCH, Dibrugarh; 2021-2022	630	7.62%	75.0%	M-70.83% F-29.17%	2.4: 1	4.53±1.17	10.49±9.02
Goyal M et al., [6]	MAMC and Lok Nayak Hospital, New Delhi; 2011-2012	6000	1.5%	-	-	-	-	-
Pao M et al., [5]	Indraprastha Apollo Hospital, New Delhi; 1998-2003	2479	2.0%	32.0%	-	-	-	-
Verma M et al., [12]	Christian Medical College, Ludhiana; 1990	1000	3.9%	48.7%	-	-	-	-
Boskabadi H et al., [13]	Ghaem Hospital, Mashhad, Iran; 2003-2009	1568	5.2%	-	-	-	-	-



Kosaryan M et al., [14]	Mazandaran, Iran; 2007-2010	115622	6.1%	-	-	-	3.22±1.8	-
Iranpour R et al., [7]	Isfahan, Iran; 2007	2560	3.2%	-	M-84.8% F-15.2%	5.5: 1	3.22±1.8	-
Elella SA et al., [15]	Menoufia governorate, Egypt; 2015	118829	4.3%	-	M-76.5% F-23.5%	3.2: 1	-	-
Bisoi S et al., [16]	IPGMER, Kolkata; 2009	109	14.67%	31.25%	M-62.5% F-37.5%	1.6: 1	-	-
Pahlavanzadeh M et al., [2]	Yazd, Iran; 2008-2009	105	18.1%	-	M-78.9% F-21.1%	3.7: 1	-	18.43
Ramadevi R et al., [17]	Karnataka; 1994	5140	-	-	M-48.8% F-51.1%	0.9: 1	-	-

**[Table/Fig-4]:** Comparison of findings in present study with other studies [2,5-7,12-17].

### Limitation(s)

Since the study was conducted in a hospital setting, the sample may not represent the general population, and the findings may not accurately apply to other populations. Additionally, the proper variant of G6PD deficient newborns could not be assessed in the study. Follow-up with the infants who tested positive for G6PD deficiency was not conducted, which prevents an assessment of the impact of the deficiency on their health outcomes and quality of life. Furthermore, potential confounding factors such as family history, ethnicity, or geographic location were not accounted for in this study, which could affect the prevalence of G6PD deficiency.

### CONCLUSION(S)

In the present study, most of the newborn babies with G6PD deficiency presented as neonatal hyperbilirubinemia. The levels of Hb were significantly lower in G6PD-deficient neonates. A significant number of females were also found to be G6PD deficient, which may be attributed to homozygous females or random X inactivation. Considering the importance of G6PD deficiency, it is crucial for clinicians to screen for this disorder and make the family members of affected neonates aware of the need to avoid certain food items, chemicals, and drugs when G6PD deficiency is detected early on. Given the high prevalence of G6PD deficiency in this population, universal screening of newborns is required in this region so that clinicians can prevent complications.

### REFERENCES

- Noori-Dalooi MR, Najafi L, Ganji SM, Hajebrahami Z, Sanati MH. Molecular identification of mutations in G6PD gene in patients with favism in Iran. *J Physiol Biochem*. 2004;60(4):273-77.
- Pahlavanzadeh M, Hekmatimoghaddam S, Ardestani MT, Ghafoorzadeh M, Aminorraaya MM. G6PD enzyme deficiency in neonatal pathologic hyperbilirubinemia in Yazd. *Iran J Ped Hematol Oncol*. 2013;3(2):69-72.
- Mitra S, Rennie J. Neonatal jaundice: Aetiology, diagnosis and treatment. *Br J Hosp Med*. 2017;78(12):699-704.
- Anderle A, Bancone G, Domingo GJ, Gerth-Guyette E, Pal S, Satyagraha AW. Point-of-care testing for G6PD deficiency: Opportunities for screening. *Int J Neonatal Screen*. 2018;4(4):34.
- Pao M, Kulkarni A, Gupta V, Kaul S, Balan S. Neonatal screening for glucose-6-phosphate dehydrogenase deficiency. *Indian J Pediatr*. 2005;72(10):835-37.
- Goyal M, Garg A, Goyal MB, Kumar S, Ramji S, Kapoor S. Newborn screening for G6PD deficiency: A 2-year data from North India. *Newborn screening for G6PD deficiency: A 2-year data from North India*. *Indian Journal of Public Health*. 2015;59(2):145-48.
- Iranpour R, Hashemipour M, Talaei SM, Soroshnia M, Armini A. Newborn screening for glucose-6-phosphate dehydrogenase deficiency in Isfahan, Iran: A quantitative assay. *Journal of Medical Screening*. 2008;15(2):62-64.
- G-Six Kit (Kinetic Method) Manufactured by Coral Clinical Systems-A division of Tulip Diagnostics (P) Ltd. [https://coralclinicalsystems.com/product-details.php?p\\_id=59](https://coralclinicalsystems.com/product-details.php?p_id=59).
- Instructions for use Vitros Microslide Vitros Chemistry and Integrated System Ortho Clinical Diagnostics. 2015;MP2-40\_EN;1 of 13,7 of 13.
- XN-550-Sysmex. <https://www.sysmex-ap.com/product/xn-550/>. (Accessed Apr 26, 2023).
- Pagana KD, Pagana TJ, Pagana TN. *Mosby's Diagnostic and Laboratory Test Reference*, St. Louis, Mo: Elsevier. 2019:600-50. <https://evolve.elsevier.com/cs/product/9780323675215?role=student>.
- Verma M, Singla D, Crowell SB. G6PD deficiency in neonates: A prospective study. *Indian J Pediatr*. 1990;57(3):385-88.
- Boskabadi H, Omidian M, Mafinejad S. Phosphate dehydrogenase deficiency in newborns with hyperbilirubinemia in Mashhad, Iran. *Maced J Med Sci*. 2010;3(4):383-87.
- Kosaryan M, Nasehi MM, Karami H, Parsaii MR, Mahdavi MR, Zakizadeh R, et al. Neonatal screening for G6PD deficiency in Mazandaran Province, Iran. 2007-2010. *IJBC*. 2011;3:113-16.
- Elella SA, Tawfik M, Barseem N, Moustafa W. Prevalence of glucose-6-phosphate dehydrogenase deficiency in neonates in Egypt. *Ann Saudi Med*. 2017;37(5):362-65.
- Bisoi S, Chakraborty S, Chattopadhyay D, Biswas B, Ray S. Glucose-6-phosphate dehydrogenase screening of babies born in a tertiary care hospital in West Bengal. *Indian J Public Health*. 2012;56(2):146-48.
- Ramadevi R, Savithri HS, Devi AR, Bittles AH, Rao NA. An unusual distribution of glucose-6-phosphate dehydrogenase deficiency of south Indian newborn population. *Indian J Biochem Biophys*. 1994;31(4):358-60.

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