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Hematological and Biochemical Markers in Glucose 6 Phosphatase Dehydrogenase (G6PD) Deficient Neonates

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Abstract

Objectives: This study aimed to analyze the association of hematological and biochemical markers with G6PD deficiency in neonates

Methodology: This comparative cross sectional study was conducted at The CITI Lab Rawalpindi from January 2024 to June 2025. After obtaining patient history we evaluated 127 patients. Laboratory analysis included complete blood count (CBC) and reticulocyte count using a Beckman Coulter Unicel DXH 800 analyzer, bilirubin estimation (total bilirubin, direct bilirubin and indirect bilirubin) and G6PD enzyme estimation was done using Cobas C311 Roche analyzer.

Results: 127 cases were analyzed and out of these12 neonates were G6PD deficient (9.4%). 2 (3.2%) out of 62 non hyperbilirubinemia neonates (control group) and 10(15.4%) out of 65 hyperbilirubinemic neonates (study group) were deficient. The peak bilirubin level in G6PD deficient group was (21.2 mg/dl) higher than that of the normal G6PD group (10.72 mg/dl). The average age of neonates was 6.93 days. 87 of the 127 neonates were male (68.5%) and the male to female ratio was 2.2:1. No significant difference was found between Hemoglobin (Hb), Mean corpuscular volume (MCV), Mean Corpuscular hemoglobin (MCH), Mean corpuscular hemoglobin concentration (MCHC) and Red blood cells (RBCs) in both the groups.

Conclusion: G6PD deficiency was seen in 9.4% neonates. The incidence of G6PD deficiency was higher in the study group as compared to the control group. G6PD deficiency prevalence was higher in males. G6PD deficiency was not associated with hemolysis in our study suggesting other mechanisms of hyperbilirubinemia exist in these neonates. Cut off value of indirect bilirubin of 17.5 mg/dl was suggested above which all the neonates should be screened for G6PD deficiency.

Keywords: Glucose 6 phosphate dehydrogenase (G6PD), Enzymopathy, Hyperbilirubinemia, Neonatal jaundice, Newborn screening.

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Introduction

Glucose 6 phosphate dehydrogenase (G6PD) deficiency is a common hereditary blood disorder affecting 500 million people worldwide.[1] It has X linked recessive inheritance pattern. It has frequency of 8% across malaria endemic countries and 5.3% within malaria eliminating countries[2] thus making it most common enzymopathy. It is prevalent in tropical and subtropical zones including Africa, the Mediterranean and Southeast Asia where its prevalence is about 25%.[3] G6PD gene is located on X chromosome[4] and spans 18 kb (Xq28). It is expressed in all tissues of the body. It has 1545 base pairs and encodes 13 exons and 12 introns.[5] G6PD deficiency is caused by approximately 140 mutations resulting in amino acid substitution.[6] These mutations result in decreased enzyme activity which may be due to decrease in enzyme stability or may be due to disruption in protein folding of enzyme[7]. It is accepted that regional and racial factors have a strong correlation with rate and type of mutations. G6PD is a cytosolic enzyme which catalyzes the first step of pentose phosphate pathway.[8] It is a rate limiting enzyme which results in the production of nicotinamide adenine dinucleotide phosphate (NADPH). NADPH keeps glutathione in reduced state. This reduced glutathione neutralizes reactive oxygen species (ROS) and thus protects cells from oxidative stress. In G6PD deficiency NADPH and reduced glutathione level drops resulting in oxidative damage that denatures hemoglobin and form Heinz bodies. These Heinz bodies are detected by the splenic macrophages which causes premature removal of these RBCs by spleen. Even in normal neonates' RBCs have a reduced life span and lower level of enzymes like glutathione peroxidase and in case of G6PD deficiency they are more prone to oxidative damage.

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Depending upon the enzyme activity WHO has categorized the disease in five classes: Class I very severe deficiency (< 1% enzyme activity), Class II severe deficiency (1-10% enzyme activity), Class III moderate deficiency (10-60% enzyme activity), Class IV Normal (60-150% enzyme activity) and Class V (>150% enzyme activity).[9] Due to X linked inheritance; G6PD deficiency is more prevalent in males as compared to females. In females there are two copies of G6PD genes if one gene is absent females are heterozygous (carriers) however if both genes are absent females become homozygous and suffer from G6PD deficiency. In heterozygous females G6PD activity can be normal, intermediate or deficient due to random X chromosome inactivation so some heterozygotes may be G6PD deficient whereas others may not be affected.[6]

Most patients with G6PD deficiency are asymptomatic but G6PD deficiency can manifest itself in three forms (i) neonatal jaundice (ii) acute hemolytic anemia (AHA) and (iii) chronic non spherocytic hemolytic anemia (CNSHA). CNSHA is rare and AHA is triggered by fava beans, infections or drugs and can be life threatening or chronic. G6PD deficiency is the most prevalent cause of neonatal jaundice (NJ). There is significant association of G6PD deficiency with neonatal hyperbilirubinemia. The likelihood of developing hyperbilirubinemia is 2.4 times greater in neonates with G6PD deficiency compared to those with normal G6PD levels.[10] About one third of babies are born outside hospitals in Pakistan otherwise the real percentage of G6PD deficiency and hyperbilirubinemia will be much higher than observed. Jaundice starts in utero but becomes apparent on second or third day. Sometimes neonatal hyperbilirubinemia can lead to debilitating complication of kernicterus. Phototherapy or exchange transfusion may be required to prevent this. Anemia is not always present in these cases and jaundice is due to combination of increased bilirubin production due to hemolysis and physiological underdevelopment of neonatal liver function or the co inheritance of Gilbert's syndrome. G6PD deficiency is a risk factor for severe jaundice causing kernicterus and it still plagues much of the world population especially in lower income countries.

The frequency of G6PD deficiency among the asymptomatic newborns in Pakistan is 10.1%[11] whereas the frequency is found to be 31.6% in neonates presenting with indirect hyperbilirubinemia.[12] The association between G6PD deficiency and Neonatal jaundice is recognized but data correlating bilirubin level with hematological parameters in local setting is limited. The aim of this study was to investigate the effect of G6PD deficiency on bilirubin level in neonates and to analyze the correlation of RBCs indices with total, direct and indirect bilirubin so as to advise the physicians that G6PD screening should be considered in case of neonatal jaundice so we can properly allocate resources to combat this problem. It is important that the cut off values of indirect bilirubin for the screening of G6PD deficiency must be evaluated.

Methodology

This comparative cross sectional study was conducted at The CITI Lab Rawalpindi from January 2024 to June 2025 after ethical approval from institutional review board. Sample size is calculated with formula $n=z^2p\ (1-p)\ /\ e^2$ where p: prevalence of G6PD deficiency in neonates = 10.1[11] Z: significant value at 95%, Confidence interval = 1.96 e: margin of error= 5%. A total of 127 neonates were included in the study. The study included all infants of both genders that presented to us for G6PD deficiency testing from all collection points. Verbal consent was taken from parents while taking history and assurance was given that there would be no monitoring burden on them. Patient history about age at presentation, gender, fever and history of blood transfusion were taken. Two groups were made neonates with total bilirubin level less than 15 mg/ dl were placed in control group and those with bilirubin higher than 15mg/dl were placed in study group. 62 cases were paced in control group and 65 cases were placed in study group.

Exclusion criteria

(I) Neonates with conjugated hyperbilirubinemia on presentation (when direct serum bilirubin level is more than 20% of the total serum bilirubin level)

(II) Neonates who had received blood transfusion within 3 months

(III) Neonates with reticulocyte count more than 5%.

Four ml of blood sample was collected, 2ml in EDTA and 2 ml in gel sample. CBC and reticulocyte count was analyzed on automated hematology analyzer (Beckman Coulter Unicel DxH 800). Total, direct and indirect bilirubin and G6PD was performed on Roche analyzer Cobas C311. Automated enzyme assay was done. Hemolysate was prepared and G6PD quantitative enzyme assay was measured from the rate of absorbance change measured at 340nm to quantify the rate of NADPH produced. The reference range for normal G6PD is 7.9 to 16.3 U/g Hb. G6PD deficiency was diagnosed when an enzyme level was < 7.9 U/g Hb. G6PD was done within 24 hours of blood collection. Data was analyzed using Statistical package for Social Sciences (SPSS) version 27. The comparison between neonates with normal G6PD level and with G6PD deficiency was done. To check the normality of data Kolmogorov Smirnov and Shapiro Wilk test was done. The statistical difference in parameters between the groups was calculated using Independent samples T test for normally distributed variables and Mann Whitney U test for variables that were not normally distributed. To check if G6PD deficiency is significantly different between normal bilirubin group and raised bilirubin group Chi square test was used and p value was obtained. The relationship between G6PD level and various risk factors such as age, Hb, RBC, MCV, MCH, MCHC and bilirubin levels etc. was estimated by deriving odds ratio (OR) and confidence intervals from logistic regression models. All presented P values were two tailed, P values were recorded and those with values <0.05 were considered statistically significant.

Results

Table I showed the age-wise distribution of neonates according to G6PD deficiency status. Most G6PD-deficient neonates were observed within the first two weeks of life, with equal frequency in the 1–7 days and 8–14 days age groups (41.7% each).

Table II showed the stratification of G6PD deficient neonates by total bilirubin level and the highest proportion of G6PD deficient neonates was found in moderate category reflecting that elevated bilirubin levels are more common among deficient neonates.

Table II: Distribution of G6PD-deficient neonates according to severity of hyperbilirubinemia.

Severity of Jaundice	Serum Bilirubin Range (mg/dL)	Number of G6PD Deficient Cases	(%)
Mild	5-12	2	16.7
Moderate	13-18	6	50
Severe	>18	4	33.3
Total		12	100

The descriptive comparisons between neonates with normal G6PD level and with G6PD deficiency was done and mean values were showed in table III. To check the normality of data Shapiro Wilk test was done. Hb, MCV and RBCs were normally distributed so Independent samples T test was used and p value was obtained. Age, MCH, MCHC, total bilirubin, direct bilirubin and indirect bilirubin were not normally distributed and Mann Whitney u test was used and p values were obtained.

Table III: Comparison of hematological and biochemical parameters between neonates with normal G6PD levels and G6PD deficiency.

	Normal G6PD	G6PD deficiency	P value
Age (days)	6.4	11.36	0.014
Hb (g/dl)	15.2	14.2	0.153
MCV (fl)	101.0	101.3	0.900
MCH (pg)	34.5	34.7	0.560
MCHC (g/dl)	34.2	34.2	0.323
RBC (million cells/mcl)	4.4	4.2	0.225
Total Bilirubin (mg/dl)	10.72	21.2	<0.001
Direct Bilirubin (mg/dl)	0.9	2.4	0.04
Indirect Bilirubin (mg/dl)	9.8	18.8	<0.001

Chi square test was used between the study group and the control group and it showed that G6PD deficiency was more common in hyperbilirubinemic neonates as shown in table IV. The p value of <0.05 was considered significant.

Correlation analysis of G6PD with other parameters was done and shown in table V. It showed that G6PD had significant positive correlation with age, total bilirubin,

Table I: Age-wise distribution of neonates according to G6PD deficiency status

G6PD	1-7 days	8-14 days	15-21 days	22-28 days	Total
Yes	05(41.7%)	05(41.7%)	01(8.3%)	01(8.3%)	12
No	83(72.2%)	18(15.7%)	08(7.0%)	06(5.2%)	115
Total	88	23	09	07	127

direct bilirubin and indirect bilirubin.

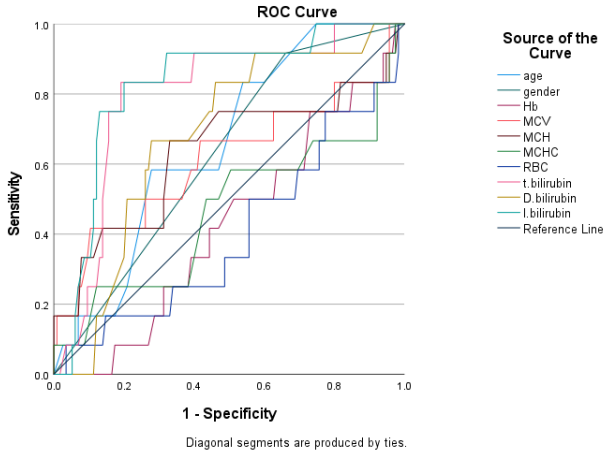
Table IV: Association of G6PD deficiency with hyperbilirubinemia among study and control groups using Chi-square analysis.			
Chi Square Test	Control group	Study group	P value
G6PD deficient neonates	2	10	<.004

Further Binary logistic regression analysis was done and shown in table VI. The table showed that Hb, age, gender, total bilirubin and indirect bilirubin has a significant impact on G6PD as P value was less than 0.05.

Table V: Correlation analysis between G6PD levels and demographic, hematological, and biochemical parameters.		
	Correlation coefficient ®	P value
Age	0.216	0.014
gender	0.129	0.144
Hb	-0.132	0.136
MCV	0.034	0.698
MCH	0.051	0.562
MCHC	-0.087	0.325
RBC	-0.108	0.219
Total bilirubin	0.356	<0.001
Direct bilirubin	0.173	0.049
Indirect bilirubin	0.357	<0.001

Table VI: Binary logistic regression analysis showing the impact of hematological and biochemical variables on G6PD deficiency.		
	P Value	Odds Ratio
Hb	<0.001	0.022
Age	0.008	1.039
gender	<0.001	2.95
MCV	0.074	1.425
MCH	0.212	2.009
MCHC	0.431	1.613
Total bilirubin	0.027	0.085
Direct Bilirubin	0.092	6.8
Indirect Bilirubin	0.016	16.5

The roc curve for different variables for predicting G6PD deficiency was done. Total bilirubin, direct bilirubin and indirect bilirubin were above the reference line, indicating meaningful discrimination. Indirect bilirubin is the best parameter in predicting the deficiency with AUC of 0.825. The optimal cut off of indirect bilirubin for predicting G6PD deficiency was 17.5 mg/dl with 83.3% sensitivity and 80% specificity based on the youden index (YI) Figure 1



Discussion

The most common causes of neonatal jaundice include hemolytic disorders, G6PD deficiency, sepsis, low birth weight and idiopathic causes (impaired function of glucoronyl transferase enzyme). G6PD deficiency which is an important risk factor for hyperbilirubinemia and kernicterus in neonates has different prevalence between geographic regions and ethnic groups. In Pakistan G6PD deficiency is prevalent in Baluchistan and Khyber Pakhtunkhwa population due to consanguineous marriages and common origin of these ethnic groups.

Several factors like imbalance between production, conjugation, elimination of bilirubin, environmental factors and ethnicity are responsible for developing hyperbilirubinemia. G6PD is responsible for producing hydrogen peroxide not only in RBCs but in all type of cells in body. In RBCs G6PD deficiency is responsible for oxidative stress leading to hemolysis. In G6PD deficient individual's enzyme activity is not only low in RBCs but also in other cells like kidney cells, liver cells, leucocytes and platelets but no other cell is completely dependent on G6PD and there are some alternatives to generate products of pentose phosphate shunt. However, when the deficiency is too severe the bactericidal function of neutrophils and monocytes becomes impaired leading to recurrent infections. Other non-hematological conditions include autoimmune disorders, and potential impacts on liver, cardiovascular and immune function and sometimes conditions become even more complex due to severe RBCs breakdown. Our study showed prevalence of G6PD deficiency in neonates was 9.4% which was close to studies done in Quetta (10.06%) [11] and Karachi (8.3%) [13]. Similar results were observed by

Noor B et al showing prevalence of 11.7% in all neonates and 29.3% in jaundiced neonates[14]. The prevalence of G6PD deficiency was higher in studies done by Khan I et al (16.8%) [15], Paopongsawan P et al (14.4%)[16], khan MS et al (12.8%)[17], Shah MA et al (13.2%)[18], Rani A et al (31.6%) [12]. Studies done in Iraq (16.5%) [19], Ethopia (24.6%) [20] and Bahrain (42%) [21] also showed increased prevalence of G6PD deficiency. On the other hand studies done by Jha RK et al (7.6%) [22], Pimpakan T et al (6.5%)[4], Yang YK et al (3.8%)[23] and Al-Bedaywi RRR et al (2.51%)[3] showed low prevalence of G6PD deficiency. Studies done by Rani A et al, Shah MA et al, Kassahun W et al and Isa HM et al showed prevalence of G6PD in jaundiced neonates[12, 18, 20, 21] whereas Khan I et al [15] showed prevalence of G6PD in jaundiced male neonates which might be the reason for high prevalence of G6PD in these studies as both male gender and high bilirubin increases the risk of G6PD deficiency.

In our study comparison between neonates with normal G6PD level and with G6PD deficiency was done and significant difference between age, total bilirubin, direct bilirubin and indirect bilirubin was seen. However no significant difference was found between Hb, MCV, MCH, MCHC and RBCs. Our findings are similar to studies done by Shahzeen et al [13] and Akhter et al[24] in which no significant difference was observed in Hb and MCV between the two groups. In contrast to our study significant difference between Hb and HCt was seen between G6PD normal and deficient neonates by Isa HM et al. [21] Rani A et al and Yang YK et al also showed decreased Hb level in G6PD deficient group as compared to control group suggesting hemolysis in G6PD deficient neonates.[12, 23]

We applied Chi square test between study group and control group and it showed that there was statistically significant association between hyperbilirubinemia and G6PD deficiency in our sample (p=<.001). In our study G6PD deficiency was seen in 3.2% (2/62) of control group and 15.4% (10/65) of the study group. Table III showed that G6PD deficient infants were at higher risk for moderate and severe hyperbilirubinemia. This observation is in contrast to study done by Wisnumurti DA et al [25]which showed no correlation of bilirubin with G6PD deficiency but most of the other studies showed correlation between bilirubin level and G6PD deficiency.[12, 15,18,20,21]

Correlation analysis of G6PD with other parameters showed that G6PD had significant positive correlation with age and bilirubin (total, direct bilirubin and indirect bilirubin). Further Binary logistic regression analysis showed that Hb, age, gender, total bilirubin and indirect bilirubin had a significant impact on G6PD. The odds ratio of age was 1.039 and as the age of patient increases the chances of being diagnosed with G6PD deficiency increases. The odd ratio of gender confirms that males are 2.95 times more prone to have G6PD deficiency as compared to females due to its X linked recessive inheritance pattern. Similar results were seen by Rani et al and Noor et al suggesting G6PD deficiency is common in male neonates.[12, 14] The odd ratio of Hb is less than 1 indicating a strong negative association with G6PD (OR=0.022). It means G6PD will be associated with anemia although Hb of both neonates with normal G6PD level and with G6PD deficiency in our study were not statistically different. Studies done by Rani et al, Isa et al and Yang et al showed lower Hb in G6PD deficient neonates.[12, 21, 23] Our results were similar to those obtained by Kassahun et al in Ethopia.[20] Indirect bilirubin odds ratio is 16.5 showing a strong positive association indicating G6PD is much more likely to occur when indirect hyperbilirubinemia is present. In contrast total bilirubin showed an odd ratio of less than 1 suggesting when direct and indirect bilirubin are added in research total bilirubin did not contribute independently to predict G6PD status.

ROC curve analysis based on YI showed that we can use indirect bilirubin value of 17.5 mg/dl as cut off value for predicting G6PD deficiency with 83.3% sensitivity and 80% specificity. So from our data we conclude that we should be alert to screen neonates for G6PD deficiency when neonates have indirect bilirubin of more than this cutoff as WHO has recommended routine screening for G6PD deficiency in neonates when the prevalence of G6PD deficiency in males is 3-5%.[4]

Limitations: Some of limitations of our study were small sample size, absence of molecular analysis in G6PD deficient neonates. In Pakistan, one third babies are born outside hospitals and a selection bias may be at play obscuring the true picture of the extent of problem.

Conclusion

In Pakistan G6PD deficiency spectrum comprises of silent carriers, mild symptoms and acute life threatening hemolysis. All G6PD deficient neonates do not develop jaundice raising the question if certain variants are more at risk of developing jaundice or if other risk factors also play a role. The disease is under reported in the country because it remains asymptomatic until an oxidative trigger is given. G6PD deficiency can cause kernicterus in neonates therefore early detection and intervention are simple and cost effective. The existing data in Pakistan is highly variable and not sufficient. No national screening program exists. WHO has recommended routine screening for G6PD deficiency in neonates but Pakistan is a resource limited country and screening all neonates will not be practically possible therefore we suggest a cutoff point of indirect bilirubin of 17.5 mg/dl above which all neonates should be screened but large scale studies are needed to assess that cutoff value. The screening in neonates will also increase parental awareness about the disease and its triggering factors so that they can improve the quality of life by avoiding them.

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