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# Glucose-6-Phosphate Dehydrogenase Deficiency in Icteric Newborns at the Essos-Yaoundé-Cameroon Hospital

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## Abstract

**Background:** Glucose-6-phosphate dehydrogenase deficiency is an enzymopathy characterized by insufficient production of reduced glutathione (GSH), a molecule known for its antioxidant role. This lack of GSH leads to a deficit in the elimination of peroxide ions from the red blood cells, causing thereby hemolytic accidents, which can be fatal if not properly managed. In neonates, the clinical picture is most often that of neonatal jaundice. **Objectives:** This study aimed to determine the place of G6PD deficiency as a cause of neonatal jaundice at Essos Hospital Centre. **Methods:** We conducted a prospective descriptive study over three months. Blood samples taken from newborns aged 0 to 28 days were analyzed in the medical analysis laboratory of the Essos Hospital Centre in Yaoundé. We carried out a determination of the enzymatic activity of G6PD, a blood count and the determination of the bilirubin level. The results obtained were analysed using R statistical software version 4.1.1. Linear regression analyses were used to assess correlations between the variables of interest. **Results:** Sixty-nine icteric neonates constituted our study population, with a total of 40 boys (58%) and 29 girls (42%) with a sex ratio of 1.37 in favour of boys. The prevalence of G6PD deficiency in icteric children was 50.72%. The mean hemoglobin was 15.3 ± 3.08 g/dL and the mean red blood cell count was 4.52 ± 1.01 × $10^6$/$mm^3$. The mean total bilirubin was 122 ± 48.3 mg/L with a maximum of 308 mg/L and the mean free bilirubin was 104 ± 46.6 mg/L with a maximum of 292 mg/L. Furthermore, after linear regression analysis, we obtained a positive and significant correlation between G6PD enzymatic activity and hemoglobin level ($r = 0.33$; $p \leq 0.001$), G6PD red blood cell level ($r = 0.26$; $p < 0.001$) and bilirubin level ($r = 0.19$; $p \leq$

0.001). **Conclusion:** Neonatal jaundice in G6PD-deficient children is a real public health problem and the prevention of hemolysis in children requires an early diagnosis of the enzyme disorder and good follow-up of the children.

## Keywords

G6PD Deficiency, Neonatal Icterus, Hemolysis

## 1. Introduction

Glucose 6-phosphate dehydrogenase (G6PD) is an enzyme that catalyses the first stage of the pentose pathway and allows the reduction of Nicotinamide Adenine Dinucleotide Phosphate (NADP) in the red blood cell to NADPH, coenzyme, which is essential in the fight against oxidative aggression via the production of reactive oxygen species [1]. Currently, it is estimated that there are 400 million G6PD deficient individuals in the world (11.9%), making it the most widespread erythrocyte enzymopathy in the world, ahead of pyruvate kinase deficiency, estimated at 1/20,000 [2]. These deficient subjects are essentially from Africa, Asia, India, the Middle East and the Mediterranean Basin with proportions estimated at 31.0% in Burkina Faso, 22.5% in Congo, 15.7% in Mali and 24.2% in Nigeria [3]. The prevalence of deficiency in Cameroon is estimated at 7.9% [4].

G6PD deficiency manifests itself primarily as induced hemolysis accompanied by unconjugated bilirubin jaundice. In neonates, physiological jaundice may worsen as a result of G6PD deficiency, and in severe cases lead to irreversible neurological complications resulting in acute or chronic encephalopathy and irreversible sequelae [5]. Studies show a high prevalence of neonatal jaundice. A study in Saudi Arabia showed a prevalence of neonatal jaundice of 25.22% [6]. A study in the USA showed an incidence of neonatal jaundice of 29.3/1000 neonates at the University Teaching Hospital; a study in Kenya showed a prevalence of jaundice of 29.9%. High prevalences of G6PD deficiency in icteric newborns are also observed. A study in Mauritania showed a prevalence of deficiency in icteric newborns of 18% [7] [8].

Neonatal screening for G6PD deficiency is not routine but is necessary for a newborn developing hyperbilirubinemia in the first 24 hours of life or infants with a history of neonatal jaundice. This work is part of the effort to improve the management of neonatal jaundice caused by G6PD deficiency.

However, these programs are not practiced in our context because of the high costs and inadequate technical facilities. This study will make it possible to update the state of the art on G6PD deficiency, and also to raise awareness among the population and health actors on the need to take it into account in the evaluation and management of icteric children.

## 2. Material and Methods

### 2.1. Type of Study and Population

We conducted a descriptive cross-sectional study over a period of 3 months

from July 1 to September 19, 2021, in the Neonatology Department of the Essos Hospital Centre of Yaounde with a capacity of 40 newborns. The study population consisted of neonates in the neonatology department with clinical signs of jaundice. Sampling was consecutive and children with parental consent were included in the study. A questionnaire designed by the authors on the basis of the literary journal was designed to collect the socio-demographic (gestational weight, sex, day of birth, etc.); and clinical-biological characteristics of the newborns including clinical data on knowledge of the condition, genetic and family history of the condition, and some phenoypic manifestations of the genetic condition.

### 2.2. Selection Criteria

The study population consisted of CHE consulting neonates with clinical signs of jaundice. Sampling was consecutive. Included in the study were those aged 0 to 28 days, with clinical signs of jaundice, and whose parental consent was obtained; Children whose sample obtained was non-compliant (hemolysis, not labelled) were also excluded.

### 2.3. Sampling and Biological Analyses

A volume of one milliliter of blood was collected in pediatric EDTA (Ethylene Diamine Tetra Acetic) and dry tubes from the neonates included in the study. Each tube of blood sample collected was sent to the Hematology and Biochemistry laboratory within one hour for analysis.

### 2.4. Biochemical Analysis of Blood Samples

The biochemical analyses consisted of the determination of G6PD activity and the determination of direct (conjugated) bilirubin and total bilirubin. The determination of enzyme activity was done spectrophotometrically and the absorbance reading at a wavelength of 340 nm according to the Cypress reagent recommendation.

The determination of bilirubin was done by the colorimetric spectrophotometer method and the reading at a wavelength of 560 nm.

### 2.5. Hematological Analysis of the Samples

The study of hematological parameters was carried out on a MINDRAY BC-2800 hematology machine with nineteen (19) parameters. The blood count was systematically accompanied by a blood smear to assess the quality of the blood cells and vital staining to classify cases of anemia and determine their origin. The slides stained with May Grunwald Giemsa and brilliant cresyl blue were read under a binocular Olympus microscope. The results obtained were interpreted according to the usual WHO values.

### 2.6. Data Processing and Statistical Analysis

The data collected were recorded in Microsoft Excel 2016. Statistical analysis of

the data was done using the statistical tool R version 4.1.1. The studied variables were gestational weight, age, hemoglobin level, free and conjugated bilirubin level, G6PD activity, and red blood cells count. The proportion of G6PD deficiency in icteric children was determined. Qualitative variables were presented as frequency while quantitative variables were presented as mean ± standard deviation. Comparison of proportions was done with the chi-square test when the expected number of children was greater than 5 and Fisher's test otherwise. Chi-square test was used to assess the association between both parameters. The Student's test was used to compare the mean between the different groups in our study population. Linear regression analysis was used to determine the relationship between G6PD deficiency and the variables studied. Pearson correlation tests were performed between G6PD level, hemoglobin and bilirubin. All these tests were done at a risk threshold of $\alpha = 5\%$.

### 2.7. Ethical Considerations

This study was approved by the Institutional Committee of Ethics and Research of the Université des Montagnes. (AUTHORISATION N˚. 2021/159/UdM/PR/CIE). Authorization to collect data and analyze samples from eligible patients has been obtained from the Essos Hospital Center in Yaoundé (N˚. 03954/AR/MINSANTE/DHL/CM). Before starting the study, participants were given a newsletter about the objectives of the study, its benefits and risks. For eligible participants, we obtained parental consent through their signature. The confidentiality of the research results was respected by the use of a unique code for each patient.

## 3. Results

### 3.1. Socio-Demographic and Clinico-Biological Characteristics of Icteric Newborns

During our study, 198 newborns were recorded; 69 icteric newborns constituted our study population, of which 35 (50.72%) were deficient. The prevalence of jaundice was 34.85%. The mean gestational age of these newborns was 37.1 ± 3.3 weeks; in G6PD-deficient newborns, 37.3 ± 3.42 weeks and 37.0 ± 3.40 weeks in those without G6PD deficiency (p = 0.782). The following Table 1 presents the socio-demographic characteristics of the children registered during the study period.

In our study population, 63.8% were 3 days old (n = 44), 31.9% were 2 days old (n = 22), and 4.35% were 4 days old (n = 3). The distribution of the population shows a male predominance (n = 40 for 22 G6PD deficient versus n = 29 for 13 G6PD deficient females). The sex ratio was 1.38 in favor of men with 58% of men (40/69) vs. 42% of women (29/69)). In addition, the average birth weight of the newborns was 3.02 ± 0.81 kg for an average height of 47.7 ± 5.13 cm. 68.1% of the newborns were born at term (n = 47) against 31.9% who were premature (n = 22). 2 (2.90%) newborns were transfused.

Table 1. Socio-demographic characteristics of icteric newborns at Essos Hospital Centre in Yaoundé between July and September 2021.

| Parameters | Deficit $N = 35$ | Normal $N = 34$ | Total $N = 69$ | p-value |
|---|---|---|---|---|
| **Gestational age (weeks), M ± SD:** | 37.3 ± 3,42 | 37.0 ± 3.40 | 37.1 ± 3.38 | 0.782 |
| **Age (days):** | | | | **0.031** |
| 2 | 7 (20,0%) | 15 (44.1%) | 22 (31.9%) | |
| 3 | 25 (71.4%) | 19 (55.9%) | 44 (63.8%) | |
| 4 | 3 (8.57%) | 0 (0.00%) | 3 (4.35%) | |
| **Sex:** | | | | 0.555 |
| F | 13 (37.1%) | 16 (47.1%) | 29 (42.0%) | |
| M | 22 (62.9%) | 18 (52,9%) | 40 (58.0%) | |
| **Gestational size (cm): M ± SD** | 47.6 ± 5.22 | 47.8 ± 5.10 | 47.7 ± 5.13 | 0.899 |
| **Gestational weight (kg): M ± SD** | 2.95 ± 0.77 | 3.09 ± 0.85 | 3.02 ± 0.81 | 0.491 |
| **Eventually:** | | | | 0.391 |
| No | 9 (25.7%) | 13 (38.2%) | 22 (31.9%) | |
| Yes | 26 (74.3%) | 21 (61.8%) | 47 (68.1%) | |
| **Transfused:** | | | | 0.239 |
| No | 35 (100%) | 32 (94.1%) | 67 (97.1%) | |
| Yes | 0 (0.00%) | 2 (5.88%) | 2 (2.90%) | |

M ± SD = Mean ± Standard Deviation.

### 3.2. Distribution of Our Study Population According to Hematological and Biochemical Parameters

The following Table 2 describes the biological parameters (hemogram profile, hemolytic and enzyme activity) in children during the study period.

From Table 2, G6PD activity in deficient children is 5.67 ± 2.99 U/ghb and 14.4 ± 4.70 U/ghb in healthy children ($p < 0.001$). In addition, hematological and hemolysis parameters did not show a significant difference although the results describe an elevation of hemolytic parameters (free, direct and total bilirubin) in children with a deficit of enzyme activity compared to normal children.

### 3.3. Correlation between Variables of Interest

The following Table 3 describes the correlation between variables of interest:

From Table 3, we note a significant and positive correlation between G6PD deficiency and HGB, ($r = 0.33$; $p < 0.001$); G6PD and RBCs ($r = 0.26$ and $p < 0.001$).

**Table 2.** Biological parameters of icteric newborns at Essos Hospital in Yaoundé between July and September 2021.

| Parameters | Deficit $N = 35$ | Normal $N = 34$ | Total $N = 69$ | p-value |
|---|---|---|---|---|
| Red blood cells ($10^6$/µL), M ± SD | 4.60 ± 0.93 | 4.43 ± 1.09 | 4.52 ± 1.01 | 0.486 |
| Haemoglobin (g/dL), M ± SD | 15.8 ± 3.13 | 14.7 ± 2.96 | 15.3 ± 3.08 | 0.121 |
| Hematocrit (%), M ± SD | 48.2 ± 9.71 | 44.7 ± 8.59 | 46.5 ± 9.27 | 0.119 |
| VGM (fL), M ± SD | 105 ± 9.32 | 101 ± 14.1 | 103 ± 12.0 | 0.198 |
| TCMH (pg/cellule), M ± SD | 34.7 ± 3.11 | 35.6 ± 11.4 | 35.1 ± 8.25 | 0.659 |
| CCMH (g/dL), M ± SD | 33.1 ± 1.53 | 32.9 ± 1.71 | 33.0 ± 1.61 | 0.694 |
| Total Bilirubin T (mg/L), M ± SD | 123 ± 55.5 | 120 ± 40.3 | 122 ± 48.3 | 0.814 |
| Direct bilirubin (mg/L), M ± SD | 13.2 ± 5.29 | 14.7 ± 10.9 | 13.9 ± 8.51 | 0.489 |
| Free bilirubin (mg/L), M ± SD | 107 ± 55.1 | 100 ± 36.2 | 104 ± 46.6 | 0.538 |
| G6PD (U/ghb), M ± SD | 5.67 ± 2.99 | 14.4 ± 4.70 | 9.98 ± 5.88 | <0.001 |

M ± SD = Mean ± Standard deviation.

**Table 3.** Correlation between variables of interest.

| Parameters | R | p-value |
|---|---|---|
| G6PD and Hemoglobin | 0.33 | <0.001 |
| G6PD and red blood cells | 0.26 | <0.001 |
| G6PD and Free bilirubin | 0.19 | <0.001 |

r = Pearson correlation coefficient.

We also observe a significant and positive linear correlation between G6PD and free bilirubin.

### 3.4. Distribution of Our Study Population According to Immuno-Hematological Parameters

The following Table 4 shows the distribution of newborns according to their ABO blood group:

It appears from Table 4 that the most frequent blood type was group O with respectively 42.9% in deficient children and 50.0% in healthy children (0.847). In addition, the most common rhesus was rhesus positive with 97.1% in deficient children and 100% in healthy children.

The results previously described suggest a follow-up of the deficient patient and even more of the newborn by his physiological immaturity and the consequences that could follow in order to improve his future.

## 4. Discussion

This study aims to describe the role of G6PD deficiency as a cause of neonatal jaundice at the Essos Hospital Centre in Yaounde. During the course of our

**Table 4.** Distribution of icteric newborns between July and September 2021 according to their blood/rhesus group.

| Parameters | Deficit $N = 35$ | Normal $N = 34$ | Total $N = 69$ | p-value |
|---|---|---|---|---|
| **Blood type child:** | | | | 0.847 |
| A | 10 (28.6%) | 7 (20.6%) | 17 (24.6%) | |
| AB | 3 (8.57%) | 2 (5.88%) | 5 (7.25%) | |
| B | 7 (20.0%) | 8 (23.5%) | 15 (21.7%) | |
| O | 15 (42.9%) | 17 (50.0%) | 32 (46.4%) | |
| **Rhesus child:** | | | | 1.000 |
| Negative | 1 (2.86%) | 0 (0.00%) | 1 (1.45%) | |
| Positive | 34 (97.1%) | 34 (100%) | 68 (98.6%) | |

study, 198 newborns were recorded, of which 69 (34.85%) presented clinical jaundice. The most represented population was male with 40 (58%) against 29 girls (42%) with a sex ratio 1.38 in favor of men. These results are similar to those obtained by Mohammed Abdel Fattah [9] who worked in Egypt on 69 newborns, including 47 boys and 22 girls; they are also similar to those obtained by Wafaa Moustafa [10] in Egypt who worked on 202 newborns, including 144 boys and 58 girls. The mean age of our study population was 2.72 ± 0.54 days which is very similar to the results of Wafaa Moustafa [10] who obtained a mean age of 3.75 days.

50.72% of icteric newborns had a G6PD deficiency. Indeed, G6PD deficiency is an enzymopathy characterised by insufficient production of reduced glutathione, an antioxidant agent. Insufficient production of reduced glutathione leads to a deficit in the elimination of peroxide ions from the red blood cell, which is responsible for haemolytic accidents that can be fatal if not properly managed [11]. In neonates, the clinical picture is most often that of neonatal jaundice. However, the population of neonates without deficiency (49.28%) could be explained by the fact that there are other causes of neonatal jaundice such as alloimmunisation due to ABO-Rhesus incompatibilities, prematurity of the newborn and many others. These results are close to those obtained by Valaes [12] in Greece who found prevalence of 50% and far from those of Karimi *et al.* and Wafaa Moustafa *et al.* [10] who found respective prevalences of 9% and 8.9% in Egypt. This could be explained by their large population size of 202 newborns and by the fact that they included all newborns present during their study period.

Among G6PD deficient individuals, the most represented population was male newborns with 22 (31.88) deficient individuals compared to 13 (18.84) deficient individuals in the general population. The transmission of G6PD deficiency is hereditary. The deficiency is linked to an anomaly in the G6PD gene located on the X chromosome and is transmitted according to the laws of genetics. A mother (XX) passes on an X to each of her children and a father (XY) passes

on an X to each of his daughters and a Y to each of his sons. When both parents are normal (because all Xs are normal, *i.e.* not carriers of G6PD deficiency), they cannot pass on a deficiency they do not have to their children. For a mother "transmitting" the G6PD deficiency, *i.e.* having an abnormal X, and a normal X (heterozygous) and a normal father (his only X is normal): If the father has a G6PD deficiency, *i.e.* his only X is abnormal (hemizygous) and the mother is normal (her 2 X's are normal): all their boys will be normal and all their girls will be transmitters. It is essentially the women who transmit to their boys [1] [2]. As G6PD deficiency is an X-linked recessive disease, the expression is greater in males as they have only one X chromosome with no ability to suppress the expression of the defective gene. Heterozygous females with one defective and one normal gene may express the gene as a normal or mild deficiency and have the chance to escape detection by the usual screening tests or even by enzyme assay [13].

Regarding the biological parameters, the mean hemoglobin value was 15.28 ± 3.08 g/dL with a minimum of 8.10 g/dL. The majority of the newborns did not have anemia, which could be explained by the fact that not all children had G6PD deficiency and therefore not all were prone to hemolysis. In our study population, some newborns had anemia. This could be explained by the fact that deficient subjects are exposed to hemolysis. These results are similar to those of Wafaa Moustafa [10] who obtained a mean hemoglobin level of 15.4 ± 3.05 g/dL with a minimum of 8.2 and a maximum of 23.1 g/dL, and similar to those of Joshi *et al.* where the mean hemoglobin level was 13.87 ± 3.59 g/dL with a minimum of 8 and a maximum of 19.4 g/dL in the neonate with hyperbilirubinemia.

The mean value of total bilirubin was 121.8 ± 48.2 mg/L with a maximum of 308 mg/L and that of free bilirubin was 103.7 ± 46.5 with a maximum of 292 mg/L thus reflecting hyperbilirubinemia. These high values of total and free bilirubin could be explained by the fact that in neonates the functional immaturity of the liver associated with G6PD deficiency promotes severe hemolysis, whose consequences are hyperbilirubinemia and kernicterus [2]. These results are lower than those of Mohammed Abdel Fattah who obtained a mean total bilirubin value of 231.09 ± 5.77 mg/L.

After linear regression analysis, we obtained a positive and significant correlation between G6PD enzyme activity and hemoglobin level ($r = 0.33$; $p \leq 0.001$), reflecting a close relationship between G6PD and hemoglobin. Indeed, the decrease in enzyme activity is accompanied by a drop in hemoglobin level proportional to the decrease in enzyme activity. Moreover, we also obtained a positive and significant correlation between G6PD enzyme activity and bilirubin level ($r = 0.19$; $p \leq 0.001$). Indeed, bilirubin from the high frequency of hemolysis, coupled with fetal immaturity in the neonate, is a consequence of the decreased activity of the enzyme.

Neonatal jaundice correlates closely with G6PD deficiency coupled with fetal immaturity in the newborn. Preventing hemolysis in deficient newborns requires a healthy lifestyle, mainly by avoiding all substances capable of stressing and lysing

the red blood cell.

**Limitation of the study.** The purpose of this study is to study the different causes of neonatal jaundice and in particular those related to G6PD deficiency. A longitudinal study on a larger population would indeed make it possible to demonstrate the real impact of this deficit on the biological parameters of the newborn and to evaluate other biological parameters characteristic of hemolysis in neonates deficient in G6PD.

## 5. Conclusion

At the end of this study, we note a prevalence of deficit of 50.72%. The prevalence of jaundice was 34.84%. Regarding biological parameters, the mean haemoglobin was 15.28 g/dL with a minimum of 8.10 g/dL and the mean red blood cell count was $4.52 \pm 1.01 \times 10^6/mm^3$ with a minimum of $1.89 \times 10^6/mm^3$. We also note a significant decrease in G6PD activity and hyperbilirubinemia in deficient children, unlike healthy children. Future studies will investigate other characteristic parameters of hemolysis in children during G6PD deficiency.

## Authors' Contributions

PYMT carried out the data collection, and analysis of the biological samples and drafted the manuscript. RDMT interpreted the data and contributed to the drafting of the manuscript. PTM contributed to the revision of the manuscript. MSN contributed to the revision of the manuscript. JLS contributed to the interpretation of the data, statistical analysis and drafting of the manuscript. PDCD contributed to the revision of the manuscript.

## Patient Consent for Publication

Written informed consent was obtained from parents/guardians, and assent by the minor was obtained.

## Ethical Considerations

This study was approved by the Institutional Ethics and Research Committee of the University of Mountains. (AUTHORISATION N˚2021/159/UdM/PR/CIE). An authorisation for data collection and analysis of samples from eligible patients was obtained from the Essos Hospital Centre of Yaoundé (N˚ 03954/AR/MINSANTE/DHL/CM). Before starting the study, a letter of information on the objectives of the study, its benefits and risks were given to the participants. For eligible participants, we obtained parental consent through their signatures. The confidentiality of the research results was respected by using a unique code for each patient.

## Data Availability

The data used in the study for this article are available close to the corresponding author.

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## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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