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Prevalence of glucose 6 phosphate dehydrogenase deficiency among infants and children of Parakou, Benin.

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Abstract

Background: Glucose-6-phosphate dehydrogenase (G6PD) deficiency is one of the most common and widespread erythrocyte enzymopathy in the world. The aim of this study was to determine the prevalence and the factors associated with G6PD deficiency in infants and children in Parakou in 2014. Methods: The current cross-sectional descriptive study with analytical purpose was carried out from March to August, 2014 in the town of Parakou (Republic of Benin). Its target consisted of 510 infants and children aged between 1 to 60 months. They were selected by two - stages cluster random sampling. Results: The recorded prevalence of G6PD deficiency was 26.1% [CI95%: 22.4%-30.2%] and factors that were associated with that deficiency were health history of neonatal jaundice and fever (p=0.0137 and 0.0001 respectively). No relationship was established between G6PD deficiency and factors such as sex, ethnic group, consanguinity, previous hemolytic crisis, jaundice outside of neonatal period, and anemia. Among clinical signs, only fever and mucocutaneous pallor were significantly associated with the studied G6PD deficiency ($p \leq 0.0001$). Conclusion: Prevalence of G6PD deficiency is considerably high in Parakou. Therefore, the introduction of systematic neonatal screening is required.

Keywords: G6PD deficiency, prevalence, infants and children of Benin, Benin,G6PD deficiency screening

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Introduction

Deficiency of glucose-6-phosphate dehydrogenase (G6PD) is one of the most common and widespread human hereditary erythrocyte enzymopathies. It affects globally about 400 million people [1-5] and particularly African populations, Black Americans, South-East Asian populations, populations around the Mediterranean and the Far East [4, 6]. It is not truly frequent among the other populations, especially in Europe and North America [5]. Its transmission is recessive and associated with sex; it is due to mutations in the G6PD gene on the long arm of X chromosome (locus Xq28). Many mutations in the *G6PD* gene were described, with the appearance of more or less functional protein accounting for the variable severity of the disease. Normal G6PD is designated as type B. There are many types of variants of the mutated G6PD gene; among them, two types are commonly found as the cause of favism: type A- that is found in African populations and the Mediterranean variant that is most common in Caucasians, [3, 5].

G6PD deficiency is mainly expressed in male (XY) hemizygous subjects and in homozygous girls. Heterozygous girls are called carriers. In homozygous girls, the disease has the same translation as in boys. G6PD enzyme activity is higher in children than in adults [3, 5]**.** It is a cause of neonatal jaundice which may lead to kernicterus in neonates and death or cerebral palsy [1]. Among a large majority of subjects affected, deficiency is most of times asymptomatic and occurs only during attacks. Those attacks are catalyzed by an important oxidative stress: infection, intake of some foods or drugs, and exposure to some substances [6]. Sometimes, deficient subject experiences an acute hemolytic crisis which may lead to potentially life-threatening complications [6].

In Benin, like most developing countries, systematic neonatal or infantile screening is not yet implemented and thus, many parents are unaware of their children's health status although a blood test for that enzyme is available. Therefore, in Benin cohort, G6PD deficiency carrier is discovered when an acute hemolytic crisis occurs. Then, even after the crisis, some patients do not receive adequate health education. They simply believe that they are « allergic » to the factor which gave rise to that hemolytic

crisis, without knowing the risk they are exposed to due to ingestion of other foods or drugs. Moreover, this tiny comprehensive pathology is poorly mastered by general practitioners and paramedics. However, the latter are certainly in the best position to do screening tests for disease detection, by providing patients with basic health education about their enzymatic deficiency and essential advices to avoid iatrogenic crises through prescription of harmful drugs likely to cause life-threatening complications in deficient subjects.

Reviewing the literature, until now, no study had been conducted to determine G6PD deficiency prevalence in Benin. This was the reason why this study has been conducted. It aimed at investigating G6PD deficiency in infants and children aged between 1 to 60 months in the town of Parakou, Benin as regards its prevalence, symptoms, and associated factors in 2014.

Study design and research methodology

The current study was a descriptive cross-sectional study with analytical purpose conducted from July 1 to August 30, 2014.

Ethical and professional considerations

This research work was approved by the institutional ethics committee of the Faculty of Medicine of Parakou.

Study setting

This research was conducted in Parakou city, Republic of Benin where the enrolled subjects were selected. The most familiar ethnic groups were Bariba (29.4%), Fon (18.7%), Dendi (15.4%), Yoruba (14.9%), Ottamari (5.4%), Yom and Lokpa (5.1%), Fulani (4.4%), Adja (2.9%), and others (3.8%). The biochemistry laboratory of the University Hospital of Borgou Department (CHUDB) was the site of blood samples handling. The equipment consisted of one spectrophotometer (microlab 300, vital scientific, Dieren, made in The Netherlands), one centrifuge (nuvefuge CN180, Nuve Ankara, Turkey) and one water bath (Digisytem Laboratory Instrument INC, Taiwan).The reagents used within the framework of this study were kits to determine G6PD activity (Randox, United Kingdom) and kits for measuring hemoglobin levels (Cromatest, Linear chemicals, Barcelona, Spain).

Participants

Infants and children aged between 1 to 60 months residing in the city of Parakou, Benin at least one year before the study and their caregivers agreed to participate in it, were enrolled. Infants and children who have benefitted from blood transfusion within the last three months prior to the day of sampling were excluded from this study.

Procedure

Sample size

Minimum sample size was set by complying with representation criteria and by considering the prevalence of G6PD deficiency in sub-Saharan Africa that is estimated to be 15% [2, 3]. We used Schwartz formula to determine the minimal sample size. The size obtained was increased by 10% to take into account non-responders. Accordingly, the calculated sample size was N'= 400+ (400*0.1) i.e. 510 children.

Type of sampling

We undertook two-stage random cluster sampling of WHO type in the three boroughs of Parakou city. Clusters consisted of 17 subjects. There were 30 clusters and cluster unit was one area of Parakou city.

Cluster selection

The sampling frame consisted of a randomly organized list of populations of infants and children aged between 1 to 60 months from 41 areas of Parakou, Benin. Among the city 41 areas, 30 were randomly selected. The survey interval was set with the formula: *k = Total population/ number of clusters* (k = 45646/30 i.e. k = 1521). One figure was drawn randomly between 1 and k. Seven was that figure. The figure 7 helped for location identification of the first cluster. The other clusters were identified by adding always the survey interval (1521) to figure 7. The list of the 30 clusters was accordingly defined.

Selection of households and children

For households' selection: the interviewer was posted in the centre of the area. He chose, by turning a bottle on the ground, a direction at random (the ones indicated by the bottle neck). The number of houses in the direction chosen was counted and numbered. A house was selected at random among the numbered houses. That chosen house by drawing of lots was the first visited. Then, one house out of two was visited throughout the limitations of the area. The operation was resumed as much as necessary until cluster size was reached. All the households where there was at least one infant or child aged between 1 to 60 months, were selected.

Variables

The judgment criterion was the presence or not of erythrocyte G6PD deficiency among infants and children that was detected through determination of G6PD enzymatic activity. The independent data were descriptive and socio demographic (age, sex, ethnic group, age of parents, and degree of consanguinity between both parents). According to the Canon law, a brother and his sister are first degree relatives; cousins are second degree relatives; children of first cousins are third degree relatives; from first to second degree there are an uncle and his niece. Individual and family health histories like sickle cell disease, neonatal jaundice, chronic abdominal pain (repeated cholelithiasis), and repetitive hemolysis (coca-cola urines) were also registered. The same option was valuable to any concept of anemia, hemolytic or jaundice crisis when contraindicated drugs were taken as well as clinical data such as fever, pallor, mucocutaneous

jaundice, splenomegaly, (back) abdominal pain, and appearance of urine were sought and documented.

Data collection

Data were collected from the infants and children's mothers through a direct structured interview using a questionnaire. A medical examination was carried out and consisted of a set of questions to search for individual or family health history suspecting G6PD deficiency as stated above. Physical examination helped assessing general condition, discoloration of the mucous membranes, presence or not of jaundice in bulbar conjunctiva, palmar pallor, and abdominal pain. Three teams composed of three persons were used to collect the data; among them there was one nurse who was responsible for collecting venous blood samples from enrolled infants and children.

Collection, transport and storage of blood samples

Blood samples were collected in the different areas selected by superficial venipuncture on EDTA tubes and kept chilled in a cooler equipped accumulator. Then, they were transported to the CHUDB biochemistry laboratory within the next two hours. Erythrocyte G6PD activity was determined on blood samples stored at +4 °C on the day of collection.

Biological manipulations

Red cell washing was done for collected 0.2 mL of blood in 2 mL of saline at 0.9% and then the resulting mixture was centrifuged at 3000 rpm during 10 minutes. These packed red blood cells were washed three times. Hemoglobin concentration in the blood samples was estimated by means of Drabkin's method (1946) [7] with Chromatest kits. Erythrocyte G6PD activity was evaluated by kinetic micro-method with Randox kits and considered as G6PD deficient when G6PD activity was below 6.97 UI/gHb/L according to the standards related to the reagent used.

Data processing and analysis

The data collected were captured and analyzed with EPI INFO 3.5 version software. The quantitative data were expressed as averages with their standard deviations and the qualitative ones as percentages. Frequencies were compared with PEARSON or FISCHER's khi- 2 test, depending on the case. Student's t test was used to compare averages. Difference was considered statistically significant when p value was below 5%. The stability and strength of associations were determined by the prevalence ratio and their confidence interval at 95%.

Results

Sociodemographic and economic characteristics

By the end of the study, 510 infants and children were selected. In that sample, more than half were boys (52.5%) with male to female sex-ratio of 1.1:1. Their ages ranged between 1 to 60 months with an average of 28.0 ± 17.1 months. In 77.6% of enrolled cases, the infants and children lived in a monogamous family. Among the 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%). The Bariba (39.8%) were the ethnic group most represented followed by the Dendi (25.5%); **Table (I)** shows the socio demographic characteristics of the study participants.

Clinical and paraclinical characteristics of the study population

Analysis of the obtained data revealed that 57.1% suffered from neonatal jaundice, 25.7% had individual history of hemolysis. **Table (II**) shows simultaneous presence of febrile condition distributed according to individual health history; all the individual health histories were significantly associated with the diagnosis of an infection (fever) (p =0.0001).

In our study, the previous intake of contraindicated drugs and foods did not result neither in hemolysis nor jaundice.

Clinical characteristics of the study population

Two clinical signs were mostly common in the respondents, especially fever (17.8%) and mucocutaneous pallor (12.3%). Anemia prevalence in the study population was 11.6%. The distribution of clinical and para-clinical signs among enrolled infants and children is shown in **Table (III)**.

Prevalence of G6PD deficiency in the study population

Table I. *Distribution of infants and children included in the study on G6PD deficiency according to sociodemographic and economic characteristics, Parakou 2014*

	Number (n=510)	Percentage (%)
Sex		
Male	268	52.5
Female	242	47.5
Age (months)		
≤ 30	307	60.2
> 30	203	39.8
Consanguinity		
Yes	104	20.4
No	406	79.6
Children's ethnic group		
Bariba	203	39.8
Dendi	130	25.5
Nago/Yoruba	45	8.8
Fon	40	7.8
Lokpa	17	3.3
Fulani	5	1.0
Others	70	13.7
Religion of the mothers		
Muslim	389	76.3
Christian	120	23.5
Others	001	0.2

Table II. *Distribution of presence of fever according to clinical health history of the infants and children included in the study on G6PD deficiency in Parakou (2014).*

	Total (N)	Previous fever		p-value
		Number (n)	Percentage (%)	
Neonatal jaundice				
Yes	40	2	5.0	**<0.0001**
No	470	454	96.8	
Health history of jaundice				
Yes	12	1	8.3	**<0.0001**
No	498	455	91.5	
Health history of hemolysis				
Yes	18	10	55.6	**0.0001**
No	492	446	90.8	

Table III. *Distribution of clinical and paraclinical signs among the population of infants and children, selected within the framework of the study on G6PD deficiency in Parakou in (2014).*

Clinical signs	Number (n)	Frequency (%)
Anemia (Hb <11g/dL)	59	11.6
Physical signs		
Fever	29	05.7
Dark urine	1	0.19
Mucocutaneous pallor	20	03.9
Mucocutaneous jaundice	1	0.19
Abdominal pain	2	0.39

Among the 510 infants and children registered, 133 had G6PD deficiency, i.e. a prevalence of 26.1% [95% CI: 22.4%-30.2%].

Socio demographic and clinical factors associated with G6PD deficiency

There was no significant difference between prevalence of G6PD deficiency of children (above 30 months of age) and in infants (under 30 months), (p = 0.5282); **Table (IV).** Also, there was no significant relationship between sex distribution and G6PD deficiency (p = 0.9823). The relationship between ethnicity and G6PD deficiency was not statistically significant (p = 0.7235), Chi^2 = 4.770. The difference in prevalence observed among infants and children aged between 1 to 60 months born from a consanguineous marriage and those born from a non consanguineous marriage, was not significant (p = 0.4712), Chi^2 = 0.5191.

There was no statistical significant relationship between fever and G6PD deficiency (p = 0.6483). Regarding health history of cases, only jaundice was significantly associated with G6PD deficiency (p = 0.0137). The association between G6PD deficiency and anemia was not significant (p = 0.0894).

Discussion

This study conducted in the population was based on a WHO type of two - stages random cluster sampling in the three boroughs of Parakou city. This type of sampling allowed us to argue that the research work results may be extrapolated to the population of infants and children aged from 1 to 60 months in the city of Parakou. The technique used helped to determine the enzymatic activity of erythrocyte G6PD without the ability to say if the deficiency was partial or total. It should have been associated with the identification of genetic variants but it enabled us to determine the prevalence of G6PD deficiency among infants and children (1 to 60 months) in the city of Parakou ; Benin that was 26.1% [95% CI: 22.4%-30.2%]. It also helped us to identify history of neonatal jaundice as one of the associated factors with G6PD deficiency.

Enrolled infants and children's age ranged between 1 to 60 months with an average of 28.0 ± 17.1 months. The age group mostly represented infants (age below 30 months); 60.2%. The neonatal target would have been easier to cover, especially in hospitals but it would not have enabled us to extrapolate the results obtained to newborns in general or to infants and children under the age of 5 years in the population. Reviewing the literature, none has been found that targeted population of infants and children aged from 1 to 60 months. The different reported researches were rather focused on newborns, like those of Dembélé in Bamako (Mali) in 2008 [8] and Guellouz in Tunisia in 2010 [9]. In 2007 in Burkina Faso, Ilboudo and *al.* [10] had rather worked on primary school children. In 2005 Diawara [11] investigated prevalence of G6PD deficiency in blood donors in Mali.

The male predominance observed in our sample is similar to the distribution based on population sex in Parakou during the 3rd General Population and Housing Census (RGPH 3) with male to female sex ratio of 1.1:1. This allows us to say that our sample could be reasonably regarded as a representative sample of the population of Parakou.

Based on ethnicity, the Bariba was the major ethnic group respectively followed by the Dendi, the Yoruba, and the

Table IV: *G6PD deficiency prevalence according to sociodemographic characteristics of mothers, infants and children included in the study and their clinical characteristics, Parakou (2014).*

	Total (N)	G6PD deficiency		p-value
		Number (n)	Percentage (%)	
Age (months)				
≤ 30	307	77	25.1	0.5282
> 30	203	56	27.6	
Sexe				
Male	268	70	26.1	0.9823
Female	242	63	26.0	
Ethnic groups				
Dendi	130	32	24.6	0.7235
Bariba	203	57	28.1	
Fulani	5	1	20.0	
Fon and Allied	40	13	32.5	
Lokpa	17	5	29.5	
Yoruba/ Nago	7	3	42.9	
Others	38	8	21.1	
Consanguineous marriages				
Yes	104	30	28.8	0.4712
No	406	103	25.4	
Clinical factors				
Fever as a trigger factor				
Yes	91	22	24.2	0.6483
No	419	111	26.5	
History of neonatal jaundice				
Yes	40	17	42.5	**0.0137**
No	470	116	24.7	
		G6PD deficiency		
Health history of hemolysis				
Yes	18	7	38.9	0.2075
No	492	126	26.6	
Anemia				
Yes	59	10	16.9	0.0894
No	451	123	27.3	
Other physical signs				**<0.0001**
Fever	29	9	31.0	
Dark urine	1	0	0	
Mucocutaneous pallor	20	11	0.5	
Abdominal pain	2	1	0.5	
Mucocutaneous jaundice	1	0	0	

Fon. Except the Fon who ranked fourth due to the lower participation rate in the first borough, ethnic distribution of children in our sample almost reflects the general ethnic distribution of the population living in Parakou.

Among the 510 infants and children involved in the survey, 104 were born to consanguineous parents, i.e. 20.4%; 68 of them were second degree relatives (65.4%), and 36 first to second degree relatives (34.6%). This could be attributed to the higher rate of consanguineous marriage in the town of Parakou, more specifically in the muslim population. However, in 79.6% of the cases, infants and children were born from a non-consanguineous marriage. This rate is similar to the one obtained by Diawara in blood donors in 2005 [11].

Neonatal jaundice, history of jaundice, and history of hemolysis were found respectively at 57.1%, 17.1%, and 25.7%. The recorded neonatal jaundice rate may be due to the higher rate of prematurity in our maternities and to prevalence of infection during the neonatal period [12, 13]. In the study target population, fever (17.8%) and

mucocutaneous pallor (12.3%) were the most common clinical signs.

G6PD deficiency was documented at a rate of 26.1%. This prevalence is close to the studies carried out in Yemen in 2009 by Al-Nood [14], in Nigeria by Williams et al in children aged 1 month to 15 years [15], in Burkina Faso by Ilboudo et al in primary school students in 2007 [10], and in Bagdad (Iraq) by Al Mendalawi in 2010 [16] which were 22.6%, 15.3%, 16.3%, and 19.2% respectively. Diawara in blood donors in 2005[11] and Traore in 2004 in children aged from 03 months to 20 years [17], found a prevalence of G6PD deficiency to be 16.2 % and 16.4% respectively. In our cohort, frequency is higher than the one found by Kaddari et al in 2004 at the Delafontaine hospital in Saint- Denis (France) [18], Castro et al in 2006 in the south of Brazil [19], Gahutu et al in 2011 on 749 children at the University teaching hospital of Butare (Rwanda) [20], Hassan et al in 2003 in Basra (South Iraq) [21], Badens et al (2000) on Timone's children in Marseille (France) [22], Benabadji in 1978 in the North of Algeria [23] with 7%, 7.9%, 9.6%, 12 .5%, 9.5%, and 3.2% respectively.

There was no significant difference in prevalence of G6PD deficiency between infants and children under 30 months and those above 30 months of age ($p = 0.5282$). This result may be due to the fact that it is a genetic disease which has nothing to do with age. Such result was comparable to those found by different authors [24, 25].

As G6PD deficiency is recognized as a genetic anomaly associated with sex (X linked recessive disorder, a significant male predominance was expected to be found in this research. Interestingly, this was not the case here. The deficiency was observed in 26.0% of the female subjects compared to 26.1% of male subjects without a significant difference. Traore h ad found male predominance with 13.7% in the Malinke areas [17]; in 1975 Duflo et al in a hospital-based study carried out in Bamako, reported male predominance with 15.7% [26]. In 1998, Kaneko et al reported a male predominance in Vanuatu with 7.4% [27], as well as William et al in Nigeria [15] and Ilboudo in Burkina with respective prevalence estimated to 24.6% and 20.5% [10]. On the contrary, in another study, Traore found a female predominance with 20.7% compared to 12.1% for male sex [28]. On the other hand, Leslie et al also found no difference in G6PD deficiency prevalence among male and female subjects in the Hazara ethnic group in Afghanistan in 2013 [29]. As G6PD deficiency is carried by X chromosome, a higher frequency is normally expected in the male subjects. Given the higher frequency of prevalence, classical theory would be no more sufficient to explain the phenomenon observed in our population. Actually, whereas a higher frequency in male children was expected, the importance of the anomaly distribution does not take anymore into account those classical data.

Unlike the males who are hemizygous for this gene and can be either normal or G6PD deficient, the females, in spite of having two G6PD genes, could be either normal or deficient (homozygous or double heterozygotes), or intermediate (heterozygous). According to Sharma et al, Lyon hypothesis can explain some situations of lack of difference in prevalence between genders. Therefore, because of X chromosome inactivation, heterozygous females are mosaics and since X inactivation is nonrandom, there can be varying phenotypes in heterozygotes females, with normal, intermediate, or grossly deficient G6PD RBC activity [30]. On molecular basis, it is known that female subjects show mosaic phenotypes, showing normal, intermediate, or high degree G6PD deficiency depending on that lyonisation of X chromosome [30, 31]. In fact in females, only one X chromosome is active, the second one remains inactive as Barr corpuscula. This fact can explain that G6PD activity could be the similar in male and in female although it was supposed to be its double [31].

Prevalence of G6PD deficiency varies from one ethnic group to another [30]. Our study population covers the three Parakou's boroughs. In the region of Parakou lives a melting and cosmopolitan population with different ethnic groups from different geographic origins. The variability of G6PD deficiency between different ethnic groups could also have a compensative effect which neutralizes the gender difference in our situation but this mosaicism needs to be investigated and documented in our population.

Deficiency in the current study was proportionally distributed between the ethnic groups, except among the Yoruba where it was mostly common (42.2%). The difference was not significant between ethnic groups. Our results are identical to those of Dolo et al who conducted a study on deficiency frequency in three ethnic groups in Mali in 2014 [32] and to those carried out by William et al in Nigeria in children aged 1 months to 15 years in many ethnic groups, although the Yoruba were the largest group in Nigeria [15].

Given that G6PD deficiency is a hereditary disease, we focused on the type of marriage between the children parents, and we found that most of them were born from a non consanguineous marriage regardless the fact they are deficient or not. There was no difference in prevalence of G6PD deficiency between children born from consanguineous marriages or those who were not. In Bamako (Mali) [9] Dembélé et al found the same results. Moreover, the different authors reviewed did not study such aspect.

Fever was diagnosed from all the infants and children with health history of neonatal jaundice, jaundice after neonatal period, and hemolytic crisis. This outcome could mean that children probably had suffered from an infection that would have resulted in an oxidative stress and brought about all those signs directly or through the use of contraindicated antipyretics in such cases.

Conclusion

At the end of this research studied G6PD deficiency prevalence, it is obvious that G6PD deficiency is considerably common and is therefore represents a public health issue in Parakou, Benin. One infant and child aged 1 to 60 months out of four was found to be deficient. Interestingly, G6PD deficiency affected both boys and girls without significant sex predilection. The recorded deficiency was proportionally distributed among enrolled ethnic groups except in the Yoruba where it was relatively higher. Infection probably was the trigger factor of deficiency clinical manifestations. Consanguineous marriage has no relation with deficiency; the same applies to anemia. On the contrary, history of neonatal jaundice was significantly associated with G6PD deficiency. Finally, 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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