# G6PD deficiency: Study among 256 blood donors recruited at the blood transfusion center in Niamey

Daouda Alhousseini<sup>1\*</sup>, Mounkaila Boutchi<sup>1</sup>, Maïguizo<sup>2</sup> Seydou, Moumouni Sina Abdramane <sup>3</sup>, Sanogo Ibrahim<sup>4</sup>

1\* = Daouda Alhousseini, biochemestry, applied biologic science department, Science Faculty of Health – Niamey-Niger;

1 = MOUNKAILA Boutchi, Hematologist, applied biologic science department, Science Faculty of Health – Niamey-Niger;

2 = MAÏGUIZO Seydou, Biologist, National Centre for Blood Transfusion – Niamey-Niger; 3 = MOUMOUNI SINA Abdramane, MD Issaka Gazobi Maternity Niamey-Niger;

4 = SANOGO Ibrahim, U.F.R. of medicals sciences Abidjan. University Félix Houphouet Boigny – cocody.

Cote d'Ivoire;

Abstract — Glucose 6-Phosphate Dehydrogenase Deficiency (G6PD) is an inherited enzymopathy which is characterized by acute or chronic hemolysis attacks triggered by oxidative stress during infections, contact with chemical, during medication or foods. In a context of nutritional anemia and/or parasitic diseases involving pregnancy or major sickle cell crises, the presence of this enzymopathy poses additional challenges to the blood transfusion. To determine the prevalence of G6PD deficiency in Niamey, we undertook to conduct a prospective study in 256 blood donors at the National Blood Transfusion Center.

G6PD intra erythrocytic was determined by spectrophotometry on the hemolysate of washed red blood cells. Blood donors aged 18-65 years, having satisfied prior medical consultation and which have a greater or equal hemoglobin to 12 g/dl were selected as part of this study.

The average age of donors was 32 years old. The deficit is estimated at 15.7%, including 9.7% of the total deficit to 6% partial deficit. Subjects deficient male G6PD accounted for 87.5% against 12.5% for the female sex. The average activity of G6PD was  $2.9 \pm 0.4$  IU/g Hb in deficient subjects against 11.7  $\pm 0.3$  IU/g Hb for the non deficient. Means Hb of normal subjects, partially and totally deficient subjects were respectively 15.3 g/dl, 14.8 g/dl and 14.3 g/dl (P = 0.7). The family donation represented 61.5% and the donor parental consanguinity rate was 21%.

As G6PD deficiency was higher in the donor population, targeted screening blood bags could be considered when they are intended for certain categories of patients.

**Keywords** — Blood donors, G6PD deficiency, hemoglobin, Niamey

# I. INTRODUCTION

glucose phosphate deficit in deshydrogenase (G6PD) in a patient hit by this enzymopathy shows a state of morbid chronic hemolytic anemia. The acute hemolysis can entail a renal insufficiency by tubular necrosis and even a deadly shock state. In the newborn the clinical table can be the one of an fatal neonatal icterus due to the functional immaturity of the liver [1]. According to the WHO the frequency of the loss in value is estimated between 15 to 26% in sub-Sahara Africa [2]. In Niger anemia still remain an important reason of consultation and hospitalization requiring sometimes massive transfusions particularly in children and pregnant women [3]. The lack of blood product and the transfusing inefficiency if the donor is deficient in G6PD complicate the fragile situation of these patients. The objective of this survey is to determine the primacy of the deficit in G6PD in donors of blood at the national center of blood transfusion of Niamey.

# II. MATERIAL AND METHODS

# A. Material

- 1) *Type and period of survey:* It is a transversal prospective survey of a 5 months length, carried out from December 2012 to April 2013 at the National Center of Blood Transfusion (CNTS) of Niamey.
- 2) *The survey Population:* The survey population is made of regular or occasional blood donors, of the two sexes, aged 18 to 65 years that come of their own to the CNTS for a donation of blood.

- 3) *Sampling:* The sample is constituted progressively as donors come by themselves to the CNTS after a preliminary medical consultation has determined if the patient is capable to give blood. For this survey donors of which the rate of hemoglobin is superior or equal to 12g/dl have been selected.
- 4) *Variables of the survey:* The variables studied are the age, the sex, the origin, the matrimonial situation, the type of donation, the consanguinity, the rate of hemoglobin, the rate of reticulocytes and the activity of the G6PD intra blood cell.

# B. METHODS

- 5) **Blood sample:** The total blood (4ml) has been levied by venipuncture at the fold of the elbow on EDTA tube for the hematological parameter determination, the reticulocytes rate count and the dosage of the activity of the G6PD intra blood cell.
- 6) *Complete blood count:* The hematological parameters have been measured with the help of an automaton of numeration "Cell dyn 1800" type of Abbott diagnoses.
- 7) G6PD Dosage: The red blood cell is washed 3 times in physiological water before being hemolyzed. The activity of the intra erythrocytic G6PD is determined by spectrophotometry at 340 nm with the help of BIOREX Diagnostics Limited (UK) reagents on the gotten hemolysate. Analyses have been done according to instructions provided by the manufacturer. Normal activity of G6PD was defined as activity from 6.97 20.5 IU/g Hb, partial deficiency ranged from 2.21 6.90 IU/g Hb and total deficiency was defined as activity < 2.20 IU/g Hb (37°C).
- 8) *Reticulocytes count:* Reticulocytes are counted manually at the microscope on thin smears gotten after blood incubation with a 1% brilliant cresyl blue during 30 minutes.
- 9) **Data analysis:** Data have been collected on a card, and then analyzed by Epi- info version 3.5. The Chi- 2 test has been used for the comparison of proportions and the threshold of significativité has been fixed to p < 0.05.
- 10) *Ethics:* This survey got the approval of the national ethics committee and does not present any conflict of interest. Blood donors who participated in this survey gave their agreement on informed consent. No donor has been any neglect for refusing to participate in this study.

### **III.RESULTS**

This survey included 256 blood donors of whose age vary from 18 to 60 years with an average of 32 years old. The age group from 28 to 32 years was the more represented followed by those of 23 to 27 years and the one of 18 to 22 years with respectively 23.4%, 20.3% and 16.8%. The majority of donors were male with 81% against 19% for the female. Donors living in a conjugal home represented 59% against 38% for bachelors. The parental gift represented 61.5% against 38.5% for the voluntary gift. The inbred donors represented 21%.

The average rate of hemoglobin was  $14.7\% \pm 2g/dl$  for the all set of donors. In the same way the average rate of reticulocytes was  $84~000~/\mu l \pm 7000$ . However we notice that 6.9% had a rate of reticulocytes below  $25000/\mu l$ . The table I shows the sharing out of the hemoglobin rate. It shows that 39.5% of blood donors have a rate of hemoglobin between 12 and 14~g/dl.

Table I: Sharing out of patients by Hb rate

Hemoglobin rate (g/dl)	Number	Percentage
12 > Hb ≤ 14	101	39.5
$14 > Hb \le 16$	79	31
Hb > 16	76	29.5
Total	256	100

The deficit in G6PD has been observed in 40 out of the 256 donors (15.7%) of which 9.7% and 6% of donors had respectively a total and a partial deficit (table II).

Table II: Sharing out of donors according to the activity of the G6PD

G6PD activity	Number	Percentage
Total deficit	25	9.7
Partial deficit	15	6
Normal	216	84.3
Total	256	100

The activity of the G6PD of the deficient donors was an average of  $2.9 \pm 0.4$  UI/g Hb opposed to  $11.7 \pm 0.3$  UI/g Hb for the non deficient donors. The majority of deficient donors in G6PD was masculine approximately 35 cases (87.5%) opposed to 5 cases for the female (12.5%).

As indicated in table III, the average Hb rate of non deficient donors in G6PD was  $15.3g/dl \pm 1.6g/dl$ , the one of partially deficient donors in G6PD was  $14.8g/dl \pm 1.1$  g/dl, whereas the one of the completely deficient donors was  $14.3g/dl \pm 1.1g/dl$ . The difference observed between the 3 categories of donors is not meaningful (p=0.7). The rate of reticulocytes in donors having a total deficit was 76 600 /ul  $\pm$  4 600, the ones having a partial deficit was 89 600/ul  $\pm$  6000 whereas in the normal donors it was 98 600/ul  $\pm$  7000. In the same way the difference is not meaningful (P=0.44).

Table III: Hematological parameters according to the activity of the G6PD

	Total deficiency	Partiel deficiency	Normal
Number	25	15	216
Average rate of Hb (g/dl)	14,3	14,8	15,3
Average rate of reticulocytes (/µl)	76 600	89 600	98 600

# IV.DISCUSSION

The average age of blood donors of the sample was 32 years old. Close to 60.5% of donors are of 18 to 32 years. The age group of 28 to 32 years represents 23.4% of our number and corresponds to the most active part of the Nigerien population. Some similar results have been found by Mountaga [4] in 2008 in Mali, with an average age of 33.14 years in their survey. The similarity of these results could be explained by certain homogeneity of the populations of these countries. The young adults represents the most important group among donors. It is explained by the fact that it is the most active age group therefore the most capable to donate blood. It is also in this age group that the feminine population is more active to the procreation, which explains that the majority of donors of our set (81%) are of male. Our results are very close to those found by Mountaga [4] that were 83.9% of male donors. These results corroborate surveys done elsewhere showing high proportions of male donors in relation

to those of female donors. Indeed according to a survey performed by Bhatgwat et al [5] in Bahrain. 96% of blood donors of their sample were male. These results are close of those of Fatemeh et al [6] that found 97.7% of male donors with an average age of 32.6 years. Besides the question of procreation the blood donation by women is also limited by the multiple constraints.

The family donation was the main source of blood at the National Center of Blood Transfusion in Niamey. Indeed 61.5% of people that came by themselves for a blood donation were relatives of the patient and 59% of donors lived in a conjugal home. Some similar situations had been evoked by Diawara [7] in Mali with 61% of family donors. The family donation yet not advisable is probably due to difficulties that the CNTS has to establish donors loyalty. The limited resources of the CNTS don't allow leading an active sensitization. Indeed an active sensitization targeted in direction of the young notably girls could make evolve the donation of blood toward a faithful one.

In our survey the rate of prevalence of the deficit in G6PD in donors of blood was 15.7%. Some comparable results have been found in a similar survey in donors of blood to the CNTS in Mali by Diawara [7] with 16.2% of deficit in all forms. The prevalence of this genetic enzymopathy varies according to the populations or ethnic groups. In children of 1 to 15 years in Nigeria, the deficit prevalence of G6PD found by Olatundun et al [8] was 15.3% while it remains lower (6,4%) according to Okebe et al [9] in the same category of age of Gambian school.

By analyzing the results of some studies on G6PD in Niamey some discrepancy was found according the category of the population. Indeed, while the study on the cord blood of newborns was reported 11, 80% of cases deficit [10], that carried out among major sickle cell disease [11] found a prevalence of 7.08%. Such a drop in sickle cell disease could be explained by a selection made from the small childhood infections in a health environment barely satisfactory.

The average rates of hemoglobin in patient partially and completely deficient have been compared to those of normal patients using Khi test and the difference is not significant. These results are similar those gotten by Shanthala Devi et al [12] that find 15.8 and 14.8 mg/dls respectively in the non deficient and in the deficient subjects.

Although the variation of the hemoglobin rate observed in the three groups of individuals is not meaningful such prevalence among donors would make the transfusion difficult in this region of malaria and all kinds of infections. Besides medicines used to fight these infections could be at the origin of oxidative stress susceptible to aggravate anemia. Numerous studies made case of hemolyse with hyperbilirubinemia after a transfusion of

deficient blood in G6PD particularly in new premature born [13], [14]. Such hemolyse becomes especially important when the transfused blood was stored during a certain time in the refrigerator between +4 and +6°C. Indeed one knows that the refrigerated storage of deficient blood in G6PD entails a deterioration of the constituent of the red blood cells more quickly such as the decrease of the activity of the ways of pentoses and gluthation reductase with as consequence the acceleration of its destruction [15]. While valuing the RBC survival, Brewer et al. [16] showed that the G6PD deficient red blood cells half-life is shorter than the one of normal red blood cells, even in the absence of administering an oxidizing medicine.

# V. CONCLUSION

This survey revealed the size of the deficit in G6PD in the blood donors' population in Niamey. It would be desirable to set a targeted screening at the time that the blood is destined to the vulnerable recipients as the new born and the major sickle cell patients to prevent hemolytic crises susceptible that complicate the precarious prognosis of these children.

# **COMPETING INTERESTS:**

The authors declare they have no competing interests relevant to the manuscript

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# REFERENCES

- [1] Monchy D, Babin FX, Srey CT, Ing PN, Von Xylander S, LY V, Busch Hallen J. Deficit in G6PD: Fréquence dans un groupe d'enfants d'âge préscolaire d'une région centrale du Cambodge. Med Trop 2004; 64: 355-358
- [2] Glucose 6 phosphate dehydrogenase deficiency. WHO working group. Bull World Health Organ 1989; 67: 601-611.
- [3] Garba M, Yayé B, Boutchi M, Idi N, Alio PA, Nayama M, L'anémie sévère per gravidique et du post partum à la maternité Issaka Gazobi: Étude prospective à propos de 207 cas sur 5 mois. Journal de la Société de Biologie Clinique, 2012; n° 016; 101-106
- [4] Mountaga Tall. Provision in blood of the hospital somine dolo of mopti. Thèse méd. Université de Bamako-Mali. 2008
- [5] Bhatgwat GP, Bapat JP. G6PD in Bahraimi Blood Donors. Bahrain Medical Bulletin, december 1987 vol.9, n°3.
- [6] Fatemeh Emamghorashi, Farhang Hoshmand, Abdolrahman Mohtashamifar. Screening glucose for 6 phosphate dehydrogenase deficiency in blood donors Asian J Transfus Sci. 2010 Jan; 4(1): 31-33.

- [7] Diawara A. Déficit en G6PD chez les donneurs de sang du CNTS de Bamako. Thèse pharm. Université de Bamako-Mali: 2005
- [8] Olatundun Williams, Daniel Gbadero, Grace Edowhorhu, Ann Brearley, Tina Slusher, Troy C. Lund, Glucose 6 phosphate Dehydrogenase Deficiency in Nigerien Children. PLOS ONE July 2013; 8(7): 1-8
- [9] Joseph Okebe, Alfred Amambua-Ngwa, Jason Parr, Sei Nishimura, Melissa Daswani, Ebako N Takem et al. The prevalence of glucose-6-phosphate dehydrogenase deficiency in Gambian school children. Malaria Journal 2014, 13:148
- [10] Mounkaila B, Daouda A, Garba RM, Aridouane D. Neonatal glucose-6-phosphate dehydrogenase (G6PD) deficiency in Niamey. International Journal of Biotech Trends and Technology (IJBTT) 2016; 13(1): 12-14
- [11] Mounkaila Boutchi, Amadou Idé Ibrahim, Alhousseini Daouda, Sabo AM Zeinabou and Ali Touré Ibrahim. Sickle Cell Anemia and Glucose-6-phosphate dehydrogenase (G6PD) deficiency: Impact on Biological and Clinical Parameters. International Journal of Biotech Trends and Technology (IJBTT) 2016; 15(1): 13-17
- [12] Shanthala Devi AM, Helen R, Vanamala A, Chaithra V, Karuna R. Screening for G6PD Deficiency in Blood Donor Population. Indian J Hematol Blood Transfus 2010 26(3):122-123
- [13] Samanta S, Kumar, P, Kishore Sai SUNIL, Garewal, G, Narang, A. Donor Blood Glucose 6-Phosphate Dehydrogenase Deficiency Reduces the Efficacy of Exchange Transfusion in Neonatal Hyperbilirubinemia. Pediatrics 2009; 123(1): 96-100
- [14] Mohammed K. Alabdulaali, Khaled Mr. Alayed, Abdulaziz F. Alshaikh, and Shihab A. Almashhadani. Prevalence of glucose 6 phosphate dehydrogenases deficiency and sickle cell milks among blood donors Riyadh in. Asian J Transfus Sci. 2010; 4(1): 31-33.
- [15] Francis RO, Jhang JS, Pham HP, Hod EA, Zimring JC, and Spitalnik SL. Glucose 6 Dehydrogenase-Deficiencies phosphate in Transfusion Medicine: The Unknown Risks. Vox Blood. 2013; 105(4): 271-282.
- [16] Brewer GJ, T AR, Kellermeyer RW. The hemolytic effect of primaquine. XII. Shortened erythrocyte life span in primaquine-sensory Negroes male in the absence of drug administration. J Lab Clin Med. 1961 58: 217-24.