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Prevalence of Glucose-6-Phosphate Dehydrogenase Deficiency in a Population of Nigerian Women Resident in Akure

David Morakinyo Sanni[1, *], Oluwasegun Victor Omotoyinbo[2], Damilola Folajimi[1]

[1]Department of Biochemistry, Federal University of Technology, Akure, Nigeria

[2]Department of Biological Sciences, Wesley University, Ondo, Nigeria

Email address:

moraksanni@yahoo.co.com (D. M. Sanni)

[*]Corresponding author

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Abstract: Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common human enzymopathy in Sub-Saharan Africa and is particularly prevalent in historically malaria-endemic countries. This work is a preliminary study on the prevalence of G6PD deficiency in both pregnant and non-pregnant women carried out on 100 subjects consisting of 50 pregnant women and 50 non-pregnant women as well as toidentify predictors of G6PD deficiency by analyzing other parameters. Presence of G6PD, total protein, albumin, total bilirubin, aspartate transaminase, alanine transaminase and alkaline phosphatase were determined in all the subjects using spectrophotometric method. It was observed that the overall prevalence of G6PD deficiency was found in 10% of pregnant women considered. The albumin, total bilirubin, aspartate transaminase, alanine transaminase, total protein and alkaline phosphatase were not significantly different in G6PD deficient pregnant and non-pregnant women. In conclusion, the prevalence of G6PD deficiency in this Nigerian sub-population showed no association between the vital parameters considered and G6PD deficiency.

Keywords: Glucose-6-Phosphate Dehydrogenase, Liver Function Test, Blood Protein, Pregnancy

1. Introduction

Glucose-6-phosphate dehydrogenase (G6PD; EC 1.1.149) is an enzyme found in erythrocytes that catalyze the initial step in the hexose monophosphate shunt oxidizing glucose-6-phosphate to phosphogluconate and reduces Nicotinamideadenine dinucleotide phosphate (NADP) to NADPH [3, 21].

G6PD deficiency is the most common human enzyme deficiency in the world. It affects an estimated 400 million people and displays the characteristics of X-linked inheritance [12, 14]. The World Health Organisation (WHO) reports reveals that 7.5% of world population are carriers of G6PD deficiency while 2.9% are G6PD deficient [25], and G6PD deficiency causes increased susceptibility of erythrocytes to H_2O_2 and other reactive oxygen species that can lead to haemolytic anemia, favism (especially in G6PD Mediterranea), chronic non-spherocytic hemolysis, spontaneous abortions [19], and neonatal hyperbilirubinemia resulting in neonatal kernicterus [8]. G6PD deficiency is however mainly found in areas where malaria is or has been endemic. In these areas, malaria is treated with drugs that can cause (severe) hemolysis in G6PD-deficient individuals [19]. G6PD deficiency is transmitted X-chromosomally, and the deficiency can be detected reliably in homozygous women and hemizygous males with a number of tests [22, 26]. During pregnancy many physiological changes occur due to several hormonal fluctuations and metabolic changes a woman's body undergoes to sustain the growing embryo. Also, due to stress of pregnancy, several disorders that have previously been subclinical may become symptomatic, for example: primary biliary cirrhosis of the liver [13]. Since

15% of normal enzyme activity and its lower values were categorized G6PD deficiency based on the classification of WHO [24] hence this severe form of G6PD deficiency should be screened in populations where the incidence is one percentage and higher [8], especially in women at the fragile state of pregnancy.

In this study, the spectrophotometric test was used to determine the prevalence of G6PD deficiency in the population of Nigerian women resident in Akure. The estimated population of Akure is over one million. The State hospital which is now an arm of the University of Medical Science, Ondo is the major hospital in Akure and does not routinely screen pregnant women for G6PD deficiency. In the course of this study, we also investigated whether simple measures such as analysis of liver function parameters had correlation with the deficiency and could help identify healthy pregnant women which are G6PD deficiency carriers.

2. Materials and Method

2.1. Subjects

One hundred (100) consecutive healthy female blood donors (pregnant and non- pregnant) at the Ondo State Specialist Hospital who came for Antenatal and other laboratory test and who were resident in Akure (aged between 20 and 49 years) gave their consent to undergo this study.

2.2. Sample Collection

Two millilitres of blood were collected using aseptic procedure from the most prominent peripheral vein in the antecubital fossa of each subject. The blood was emptied into Ethylenediamine tetra acetic acid (EDTA) bottle and gentlymixed. The blood sample was then stored at 4°C ina refrigerator till required for use. All bloodsamples collected were analysed within 48 hours ofcollection.

2.3. Methodology

2.3.1. G6PD Assay

All samples were screened for G6PD activity spectrophotometrically [24] and was determined by quantitative assay of the enzyme activity in erythrocytes based on the RANDOX procedure for quantitative determination of G6PD using RANDOX diagnostic kit manufactured by RANDOX Laboratories Limited (Ardmore Diamond Road, Crumlin, Country Antrim, United Kingdom), with decreased activity or G6PD deficiency taken as any level of enzyme activity less than 6.97 I.U/g haemoglobin (reference range 6.97 – 20.5 U/g Hb [37°C] IU/g haemoglobin).

2.3.2. Serum Proteins

Serum total proteins was assayed by biuret method [15], the measurement of serum albumin was based on its quantitative binding to the indicator bromocresol green, (BCG). The albumin-BCG-complex absorbs maximally at 570nm, the absorbance being directly proportional to the concentration of albumin in the sample.

2.3.3. Bilirubin Assay

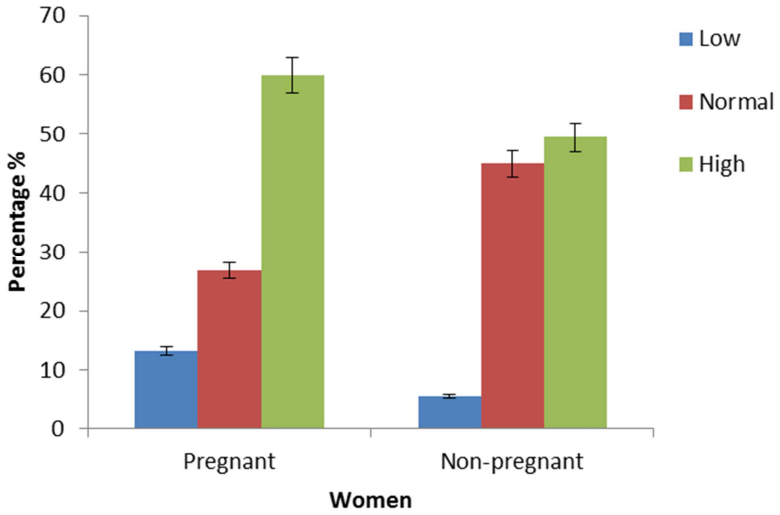
Bilirubin test was also assayed by method of Kawamoto *et al*., [10].

2.3.4. Liver Function Test

Liver function parameters; aspartate transaminase (AST), alanine transaminase (ALT) and alkaline phosphatase (ALP) levels were determined in both pregnant and non-pregnant women by spectrophotometric method, using RANDOX diagnostic kit.

3. Results and Discussion

From the result in figure 1, the percentage of G6PD deficient persons was higher in pregnant women (13.3%) than that of non-pregnant women (5.5%) with an observable low G6PD activity.

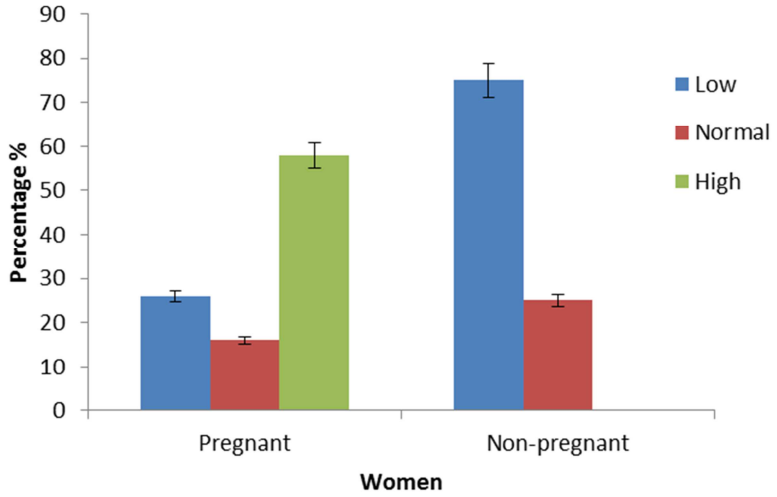


Low range: < 6.97 U/gHb; Normal (Standard) range: 6.97 – 20.5 U/gHb; High range: > 20.5 U/gHb

Figure 1. *Prevalence of G6PD deficiency in Pregnant and Non-pregnant Women.*

In the work of Owusu *et al*. [18], the overall prevalence of G6PD deficiency was 19.3% in pregnant women; 2.3% G6PD full defect and 17.0% partial defect. This partial defect can be due to reported cases of women in whom haemolysis occurred during pregnancy [20] and may also account for the higher prevalence of G6PD deficiency in this study. The factors that may lead to G6PD deficiency in pregnant women include drugs such as sulfonamides an antimalarial which can make them become susceptible to anaemic conditions characterized by low haemoglobin level and high total bilirubin level. Also, the 60% of pregnant women in the study which showed high G6PD activity, may be due to the rich iron containing foods and supplements intake at that stage which women are advised to take.

A high level of total protein was observed in pregnant women (58%) compared to that of the non-pregnant women, while 75% of non-pregnant women have lower total protein level than those of pregnant women (26%) – figure 2. The low level of total protein concentration in pregnant women can be as a result of malnutrition and malabsorbtion, dilution of protein due to extra fluid held in the vascular system or malaria which reduces protein synthesis [5, 7].

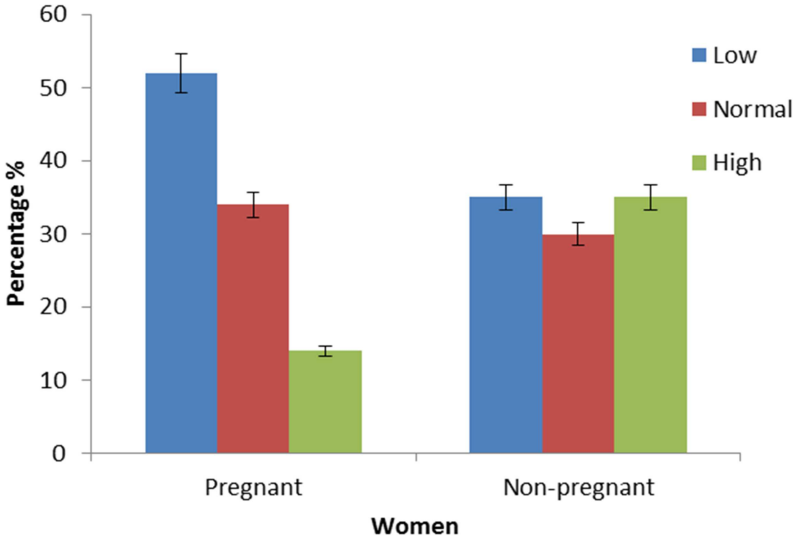


Low range: < 64 g/l; Normal (Standard) range: 64 – 83 g/l; High range: > 83 g/l

Figure 2. *Total protein level in Pregnant and Non-pregnant Women.*

The findings in this study agrees with that of Bygbjerb and Flash [5] who reported that chronic infections and autoimmune diseases may lead to reduced protein synthesis. Also, the percentage of pregnant women with high total protein level is manageable except for those with cases of pre-eclemsia.

Result of total albumin level in pregnant and non-pregnant women is shown in figure 3. Serum albumin level in pregnancy related hypertension is a significant determinant of disease severity, and may be considered as a useful marker for predicting time of delivery, severe proteinuria, and pregnancy outcomes [23].

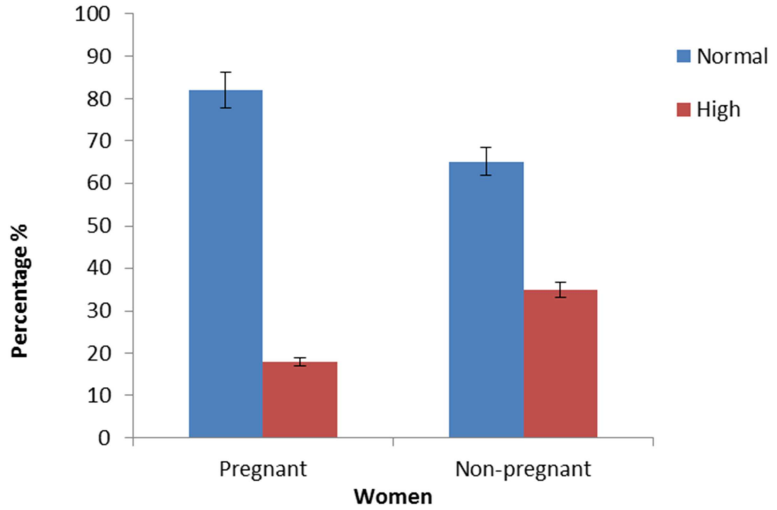


Low range: < 38 g/l; Normal (Standard) range: 38 – 44 g/l; High range: >44 g/l

Figure 3. *Total Albumin level in Pregnant and Non-pregnant Women.*

Low level of total albumin was observed in pregnant women compared to the non-pregnant ones, while higher level of albumin was observed in non-pregnant women. An increase in albumin level may be generally due to high protein diet, dehydration while a decrease can be as a result of liver dysfunction, kidney diseases, malnutrition, low protein intake etc. However, during pregnancy, serum albumin level usually decreases [2]. Serum albumin levels decrease during the first trimester, and this decrease becomes more accentuated as the pregnancy advances. This decrease in serum albumin concentration may be due to the hemodilution phenomenon [1, 17].

Total bilirubin level was estimated in pregnant and non-pregnant women to confirm hyperbilirubinaemia. From the results in figure 4, there was no significant difference between the percentage total bilirubin level in both pregnant and non-pregnant women. This observation was also confirmed in the work done in Sudan by Devarbhavi *et al*., [6], where slight increase in total bilirubin level between the pregnant and non-pregnant women was also observed. Conditions such as malaria infection contribute greatly to the rise in bilirubin level especially in pregnant women with G6PD deficiency due to acute hemolysis [9].



Normal (Standard) range: up to 17 µmol/L; High range: >17 µmol/L

Figure 4. *Total Bilirubin level in Pregnant and Non-pregnant Women.*

Liver function enzymes assay in study subjects is shown in figure 5. Alanine transaminase (ALT) level in both women were normal, which indicate that there was no destruction of the liver hepatocyte in both women while slight increase in Aspartate transaminase (AST) level in both pregnant and non-pregnant women was observed. This might be due to other causes like stress, gestational age, and malaria but not liver related. Similar findings was reported by Nour *et al*., [16], who observed that there was significant difference in the level of AST between the pregnant women and non-pregnant women.

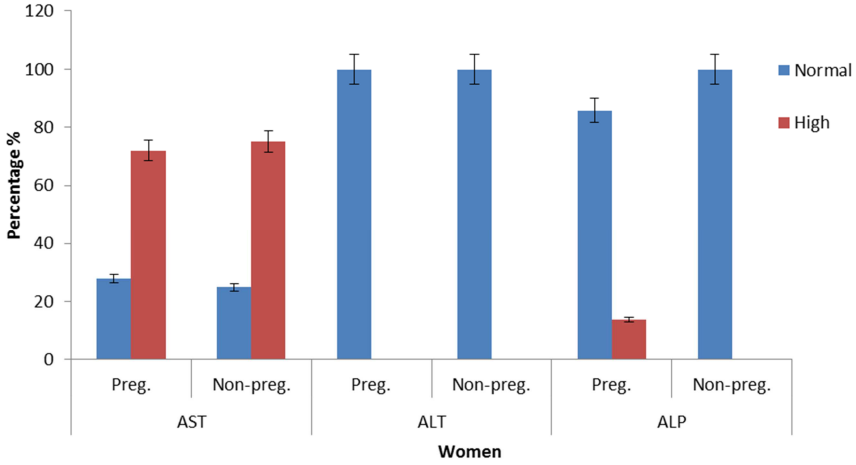


Figure 5. *Liver function parameters level in Pregnant and Non-pregnant Women.*

AST: Normal (Standard) range: up to 18 U/L; High range: > 18 U/L
ALT: Normal (Standard) range: up to 18 U/L; High range: > 18 U/L
ALP: Normal (Standard) range: 9 – 35 U/L; High range: > 35 U/L

For alkaline phosphatase (ALP) level, only 14% pregnant women had slightly elevated level of ALP. The slight increase in ALP level in the pregnant women might be related to the trimester stages as the placenta formation during pregnancy causes slight increase in ALP level during pregnancy [11]. Since other clinical findings of liver function parameters show that causes of liver function parameter were within range, increase in ALP level is observed in either the intrahepatic or extrahepatic cases of cholestatis, hepatitis, cirrhosis etc.

Result obtained from regression analysis showed no significant difference in levels of all parameters considered in this study in relation to G6PD deficiency in pregnant women except for total protein which had a positive effect, while for non-pregnant women, only AST, ALT and total bilirubin had a positive effect on G6PD deficiency.

4. Conclusion

This study therefore reveals that the prevalence of G6PD deficiency in this Nigerian sub-population showed no association between the vital parameters considered. Also, G6PD deficiency was also linked as a potential cause of haemolytic episode in neonates in the study area. Hence it becomes imperative and necessary for health care givers to include G6PD deficiency testing during ante-natal care.

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