Incidence of Glucose-6-Phosphate Dehydrogenase Deficiency in South East of Nigeria

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Abstract

Between June 1976 and July 1994, 6,477 blood specimens from 3, 809 males and 2,668 females were sent to Chemical Pathology Laboratory for glucose-6-phosphate dehydrogenase (G6PD) status. A total of 2,159 made up of 1,356 (20.9 %) males and 805 (12.4 %) females were found to be G6PD deficient. 75 volunteers comprising 51 males and 24 females were screened for this enzyme disorder. Of this number 11 males and 3 females were found to be deficient. The result shows that the number of individuals who are deficient for the enzyme is higher in hospital patients than in volunteers. Secondly, the study confirms a higher incidence of the disorder in makes than females. The incidence among volunteers is in agreement with findings by other workers in normal population.

Keywords: Glucose-6-phosphate dehydrogenase, Deficiency Incidence, South East Nigeria

Introduction

Glucose-6- phosphate dehydrogenase (G6PD) is hydrogen transfer enzymes with mediate the reversible transfer of hydrogen from β-D-glucose phosphate to co-enzyme NADP (Burns and Werners, 1962). G6PD deficiency is a sex –linked enzymopathy which dates as far back as 5th century BC. It has been described as various anaemic conditions in the past like Baghdad anaemia and fauvism (Senazam and Thielman, 1991).

Currently an estimated 200-400 million people are affected by G6PD deficiency worldwide (Beutler, 1991; Melita, 1994). The geographical distribution of G6PD deficiency with high frequency in tropical and semi-tropical malaria endemic zones, suggests that the trait provide an evolutionary selection advantage against malaria. (Harris and Gilles, 1961). This theory is based on the understanding that the malaria parasite requires reduced pathway of carbohydrate metabolism for survival and optimum growth.

Internationally, the highest prevalence rates of anaemia and some other blood disorders are found in tropical Africa, the Middle East, tropical and subtropical Asia, some areas of the Mediterranean, Papua New Guinea and Southern Europe (Nutrition and Blood, 1996). Not much was understood about this disorder until early 1950's when a big US Army funded research was carried out to determine the cause of a drug related sensitivity in up to 15 % of the Negro soldier (Senazam and Thielman, 1991).

One of the researchers, Carson discovered that red blood cells from the drug-sensitive patients were deficient in the glucose-6-phosphate dehydrogenase enzyme. The most common clinical feature is that symptomatic patients present with neonatal jaundice which appear by the age of 1-4 days at the same time as or slightly earlier than the so called physiological jaundice. G6PD deficiency affects all races and severity varies significantly between racial groups because of different variants of the enzyme. Severe deficiency variant primarily occur in Mediterranean population. The enzymatic variant in the African population has more activity and produce a milder form of the disease. (Beutler et al., 1992). It is only of the genetically determined disorder of drug induced haemolysis.

In Nigeria, most of the work has been carried out among the Yoruba speaking people of Western Nigeria with little work done in Igbo speaking area of South-East. Present study was done to determine the incidence of G6PD deficiency and also to highlight inherent dangers associated with self medication in the presence of this disorder among the Igbos of South East.

Materials and Methods

The subjects were of two categories: (a) Those for retrospective study include all the patients both in patients and out-patients) whose blood samples were sent to Chemical Pathology Laboratory, UNTH for G6PD screening from June 1976 – July 1994, and (b) Healthy subjects from random sampling. This includes students within UNTH and beyond as well as hospital workers who were randomly selected. Their age ranged from 11 years to 60 years. All were Igbos made up of 51 males and 24 females.

Blood samples were collected into EDTA anticoagulant tubes in the ratio of 5 ml of blood to 0.155 ml of anticoagulant. Blood samples were screened immediately for G6PD using the methaemoglobin reduction method (Brewer *et al.*, 1960).

Interpretations: Clear red colour as the colour of blood initially before analysis (Normal sample); brown in colour similar to that of positive control (Deficient sample) and blood with intermediate result (Heterozygous sample).

Results

Table 1 shows the pattern of G6PD deficiency from retrospective study. 2,159 subjects were deficient

out of 6,477 subjects whose blood samples were sent to Chemical Pathology Department for screening. This translates to 33.3% of the number. 1,354 (20.9 %) were males and 805 (12.4%) were females, indicating a higher incidence of enzymes deficiency in males.

Table 1: Pattern of G6PD deficiency from retrospective study

Subjects	Males	Females	Total
Deficient Number (n)	1,354	805	2,159
Percentage (%)	20.9	12.4	33.3
Non – deficient Number (n)	2064	2254	4318
Percentage (%)	12.4	52.2	66.7
Number screened	3418	3159	6477

Table 2 shows the pattern of G6PD deficiency among volunteers. A total of 75 subjects were screened for enzyme deficiency. 14 (18.7 %) were deficient. 11 patients (14.7 %) were males while 3 (4 %) were females yielding once again a higher incidence of enzymes deficiency in males than females.

Table 2: Shows the pattern of G6PD deficiency among volunteers

Subjects	Males	Females	Total
Deficient Number (n)	11	3	14
Percentage (%)	14.7	4	18.7
Non – deficient Number (n)	40	21	16
Percentage (%)	53.3	28	81.3

Discussion

A total of 6552 subjects were used for this study. This includes those for retrospective study and random sampling analysis. Out of this number, 6477 represent the number of subjects used for retrospective study. The pattern of G6PD deficiency in this group was 33.3 %. However, the pattern of enzyme deficiency for the random population sampling was 18.7%.

Harris and Gilles recorded an incidence of 20 % among South Western Nigeria (Harris and Gilles, 1961). Allison and co-workers found an incidence of 20.6 % among Northern Nigeria (Allison et al., 1961). Both group worked on subjects that were randomly selected. The value obtained from our retrospective study does not agree with that of either Harris and Gilles or Allison and co workers. It however agreed with the result obtained by Worblinks using hospital patients at ABUTH Zaria Nigeria (Worblinks, 1981). Azubuike reported an incidence of 26.7 % among children with neonatal jaundice UNTH Enugu (Azubuike, 1979). While Ransom Kuti has a value of 71 % among outpatient with neonatal jaundice (Ransom Kuti, 1972). It can be seen that there is generally a higher incidence of G6PD deficiency when hospital patient are screened than when subjects are randomly selected from the general population. The value obtained in the present study from random sampling analysis agrees more with that of Harris and Gilles. It can be seen that though fewer subjects were used, the data represents more of the G6PD deficient pattern than from the retrospective study. This study shows that incidence of G6PD deficiency among Igbo's is higher in males than females. This agrees with the work of Harries and Gilles, Camp *et al* who found an incidence of 20.6 % (males) and 21 % (females) respectively (Capps and Gilles, 1963).

In drug-induce hemolytic anaemia, the primary defect is a deficiency in G6PD. Production of NADPH is diminished. The major role of NADPH in red cells is to reduce the disulphite from of glutathione. Reduced glutathione plays an important role in detoxification by reacting with H_2O_2 and organic peroxides during electron transport. Glutathione is essential for maintaining normal red cell structure and for keeping haemoglobin in the ferrous states. Drugs such primaquine may disrupt the surface of red cells in the absence of reduced glutathione which will make them more liable to destruction and removal by the spleen. These drugs also increase the rate of formation of toxic peroxides capable of damaging various biomolecules.

Favism is a limited aspect of G6PD deficiency and is confined to relatively small geographical areas such as the Mediterranean regions, the Far East and Southern Asia, although sporadic cases have also been described elsewhere (Sansone *et al.*, 1958; Sarton, 1957). The highest incidence about 15 % of all G6PD deficient subjects has been described (Sartori, 1957). The G6PD enzyme of subjects sensitive to fava beans has been characterized as the Mediterranean variant (Kirkman *et al.*, 1965).

Fresh fava beans are the major cause of haemolytic anaemia although dried beans and sometimes, the pollen of plants have also been incriminated (Schiliro *et al.*, 1979). This is a very forward attempt to look at one of the most wide-spread inherited enzyme deficiency with human beings. The understanding of the principle behind the mechanism of haemolysis in Glucose-6-phosphate dehydrogenase deficient patients when subjected to Oxidative stress is by far the most important discovery. In the more severe variants, haemolysis may sometimes occur without any evident precipitating factor but more significantly in the increased sensitivity to drug and chemical agent. Give this potential haemolytic danger, health care professional should always bear in mind the adverse consequences when administrating drugs to patients with glucose-6-phosphate dehydrogenase deficiency.

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