FREQUENCY OF TOXOPLASmosis IN CHILDREN WITH GLUcose-6-phosphate DEHYDROGENASE (G6PD) DEFICIENCY

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ABSTRACT
A prospective study was carried out to evaluate the frequency of toxoplasmosis in children with glucose-6-phosphate dehydrogenase deficiency at Basrah General Hospital and Basrah Maternity and Children Hospital for 1 year (from October 2004 - October 2005). One hundred ten children (6 months - 60 months) of age were screened for both toxoplasmosis antibodies and glucose-6-phosphate dehydrogenase deficiency; fifty (45.5%) children had glucose-6-phosphate dehydrogenase deficiency, 48% of them had toxoplasmosis. The reminder sixty children (54.5%) had normal glucose-6-phosphate dehydrogenase activity; among them fifteen (25%) had toxoplasmosis. From this study it was concluded that acquired toxoplasmosis was significantly higher among children with G6PD deficiency compared to those of normal G6PD activity.

INTRODUCTION
Glucose-6-phosphate dehydrogenase deficiency (G6PD), the most common enzymatic deficiency worldwide, causes a spectrum of diseases including neonatal hyperbilirubinemia, acute hemolysis, and chronic hemolysis, this x-linked inherited disorder affects approximately 400 million people worldwide[1]. It is distributed in the Mediterranean region, across Middle East including Iraq[2], India, Indonesia, South China as well as Middle Africa, with an incidence over 1%[3]. The over all incidence of G6PD deficiency varies from 8%-50% in the Arab Word[4]. The frequency of G6PD deficiency 18% in Eastern Province of Saudi Arabia& 8.4% for the western province[5,6]. In Iraq (6.1%) were G6PD deficient[2]. In Basrah; southern Iraq, G6PD deficiency was detected in (12.5%) of people[7]. Patients with G6PD deficiency are predisposed to bacterial infections of variable severity. The underlying immune deficiency is due to phagocytic killing defect. An unusual propensity for infection with catalase positive organisms has been reported in patients with severe deficiency or complete absence of G6PD[4,8]. In normal person there are two host defense mechanisms, non-specific and specific types, the non-specific ones include physical and chemical barriers as well as complement system and phagocytic cells, the specific ones are the cell mediated immune responses, both mechanisms are involved in host defense against toxoplasma infection[9,10]. The parasite Toxoplasma gondii infects between 30-60% of people across the world. Most human infections are benign; in adults and children past the neonatal period, the disease is usually asymptomatic. Nevertheless, a generalized infection probably occurs. When symptoms are seen, they are most frequently mild and the disease picture simulates infectious mononucleosis. Diagnosis of toxoplasma infection is seldom made by recovery of the parasite; usually it is done by serological tests[11]. The prevalence of toxoplasmosis was (41.1%–52.1%) in a community–based sero-epidemiological study done in 3 areas (rural, urban and suburban semi rural) in Basrah governorate, southern Iraq[12]. In some variants of G6PD deficiency with absent leucocyte G6PD, there may be abnormal leucocyte function and such subjects present with infection similar to chronic granulomatous disease[9]. Literature reports have shown increase frequency of G6PD deficiency in the red cells of patients with typhoid fever as compared to general population[13,14] and increased susceptibility to catalase-positive bacterial infection in red cells among G6PD deficient newborn infants[15]. In addition, an increased increase incidence of specific infections such as viral hepatitis is also found in G6PD deficiency[4,6,16]. This study was carried out to assess the frequency of toxoplasma antibodies among children who had G6PD deficiency in comparison to children with normal G6PD activity.

PATIENTS AND METHODS
One hundred ten children (6-60) months of age were included in the study, all were presented with mild acute illnesses as upper respiratory
tract infections or diarrhea, (children with chronic illnesses were excluded), those children were attending outpatient clinic in Basrah General Hospital and Basrah Maternity and Children Hospital for 1 year (from October 2004-October 2005). All children were normal on examination showing no abnormal physical signs. Malnourished children, children with chronic illnesses or those who had positive physical signs were not included in the study.

**Toxoplasma antibodies estimation:**
Four milliliters of blood was aspirated from each child, 2 ml were put in separate test tube, centrifuged and the serum was screened for toxoplasma serum titer by indirect fluorescence antibody test technique, the children with positive titer were considered to have acquired toxoplasma infection because all of them were more than six months of age.

**G6PD estimation**
Other 2 ml of blood were put in test tube with 0.4 ml of anticoagulant (sodium citrate and citric acid) then 0.1 ml of sodium nitrate and 0.1 ml of methylene blue were added to it, then was put in the incubator for 3 hours, after that change of the color was assessed as screening for G6PD deficiency, red color was regarded as normal G6PD activity, brown color was regarded as intermediate deficiency deep brown was regarded as full or severe deficiency[17]. Children were divided into two groups, those with normal G6PD activity and those with G6PD full deficiency. (Intermediate deficiency was not considered in our study). Toxoplasma serum titers were assessed in both groups and the frequency of positive toxoplasmosis was compared in G6PD deficient group with group of normal G6PD activity. For sake of statistical analysis Chi-square (x²) test was used, the significance level was set as P<0.05

**RESULTS**
One hundred ten children were included in the study; 50(45.5%) had severe G6PD deficiency while 60(54.5%) had G6PD normal activity. In G6PD deficient group, 24 (48%) had positive titer for toxoplasmosis, the remaining 26(52%) had negative toxoplasma serum titer. (Table-1) Among group of normal G6PD, 15(25%) had positive toxoplasma titer while 45(75%) were negative for toxoplasma titer. The infection rate was 48% in G6PD deficiency group while it was 25% in G6PD normal group. X²=6.3, P-value < 0.025, so the results were statistically significant.

<table>
<thead>
<tr>
<th>Toxoplasma titer</th>
<th>G6PD deficiency</th>
<th>G6PD normal</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>24 48</td>
<td>15 25</td>
<td>39 35.4</td>
</tr>
<tr>
<td>Negative</td>
<td>26 52</td>
<td>45 75</td>
<td>71 64.5</td>
</tr>
<tr>
<td>Total</td>
<td>50 45.5</td>
<td>60 54.5</td>
<td>110 100</td>
</tr>
</tbody>
</table>

Chi square=6.3, P-value <0.05, the results were statistically significant

**DISCUSSION**
*Toxoplasma gondii* is ubiquitous intracellular protozoal parasite; the infective form can stimulate an inflammatory response and has capacity to invade the cells of reticuloendothelial system and neural cells, in immunologically normal children acute acquired infection may be asymptomatic, or causes lymphadenopathy[10]. While the effect of G6PD deficiency on red blood cells (RBC) is well known, there is less information available regarding the effect and impact of G6PD deficiency in polymorph nuclear neutrophils (PMNs) functions and its clinical impact[4]. In this study the rate of toxoplasma infection was compared in children with G6PD deficiency versus children of normal G6PD activity. It was found that toxoplasma infection was higher among G6PD deficient children, this result was similar to study done at Saudi Arabia at 1995[6]. The likely explanation is due to both direct destruction of the reticuloendothelial system by toxoplasma organisms and decreased killing of the phagocytic cells, this is clear from the fact that when polymorph neutrophils (PMNs) are stimulated, the enzymatic
negative produce hydrogen monoxide and were catalase deficient leukocyte can kill organisms which monophosphate shunt and however G6PD deficient leucocytes result in defective pentose system manifestations to concentrate, so could cause central nervous and muscle tissue could reduce people’s ability serious and highly underestimated public health acquired toxoplasmosis might in fact represent a even if they are asymptomatic as asymptomatic only in whole blood, and to assess toxoplasma measure G6PD level in PMNs suspension not do further study to assess bactericidal activity of factor for toxoplasmosis so it is recommend to It was concluded that G6PD is regarded as risk activity in 25% of them as demonstrated in Table-1.

It was concluded that G6PD is regarded as risk factor for toxoplasmosis so it is recommend to do further study to assess bactericidal activity of PMNs in children with G6PD deficiency and to measure G6PD level in PMNs suspension not only in whole blood, and to assess toxoplasma antibody titer in children with G6PD deficiency even if they are asymptomatic as asymptomatic acquired toxoplasmosis might in fact represent a serious and highly underestimated public health problem as cysts which are formed in the nerves and muscle tissue could reduce people’s ability to concentrate, so could cause central nervous system manifestations.

REFERENCES

3. TK chan EYang KD. G6PD deficiency, a review htm 2003; (1-13).