Iranian J Parasitol: Vol.2, No.1, 2007, pp.43-45

Case report

A Patient with G6PD Deficiency and Falciparum Malaria

** M Nateghpour [1], A Miahipour [1], M Yousefi [1], A Mohammadiha [1], Y Fagani [2]*

[1]Dept. of Medical Parasitology and Mycology, School of Public Health & Institute of Public Health Research, Tehran University of Medical Sciences, Iran
[2]Infections Diseases Ward, Bou-Ali Hospital, Tehran, Iran

(Received 3 Nov 2006; accepted 3Mar 2007)

Abstract

A 20 year old male patient from Afghanistan with a history of G6PD deficiency and clinical manifestations of malaria referred to Bou-Ali Hospital in Tehran, capital of Iran. Giemsa stained thick blood films revealed an infection of *Plasmodium falciparum* with 33700 parasite/µL of blood. The patient was successfully treated according to malaria treatment guideline.

Key words: *G6PD deficiency, Falciparum, Malaria, Iran*

Introduction

Glucose-6-phosphate-dehydrogenase (G6PD) is a cytoplasmic enzyme that is essential for a cells capacity to withstand oxidant stress. The geographic correlation of G6PD deficiency distribution with the endemicity of malaria suggests that such enzymopathy has risen in frequency through natural selection by falciparum malaria.

Motulsky and Allison (1, 2) suggested that individuals with G6PD deficiency trait might be more than normally resistant to falciparum malaria. These authors and later some others found a notable correlation between the prevalence of such enzymopathy of human and the endemicity of *Plasmodium falciparum* malaria in some areas of the world. The suggestion of such inherent relative resistance to falciparum malaria in the enzyme deficient subjects has been supported by low parasitaemia and low mortality in G6PD deficient patients (3-5). Nevertheless, this hypothesis does not seem to constitute the whole story. Some evidences have been put forward by the some researchers (6-8).

Case report

In this report, we introduce a twenty year old Afghani worker resident in Tehran, capital of Iran with a history of traveling to some villages located on the southeast borderland of Iran, three weeks before to feel clinical signs. The patient with typical malaria symptoms (chilliness, fever, and sweating), headache, and hemoglobinuria referred to Bou-Ali Hospital in Tehran. The laboratory analysis of blood revealed as: G6PD partial deficiency (using colorimetry and fluorescence activity methods), WBC 3100/ micro/L, eosinophils 3%, RBC 3.47 million/µl, SGOT 173 IU/L,

***Correspondent author:** Tel: +98 21 88989130, E-mail: nateghpourm@sina.tums.ac.ir

SGPT 147 IU/L, hemoglobin 10.2 g/dl and hematocrit 29.6%. Giemsa stained thick and thin blood films indicated *P. falciparum* malaria infection with high parasitaemia of 33700/ µl. The patient was successfully treated according to malaria treatment guideline.

Discussion

Malaria-protection hypothesis of G6PD deficiency seems to be a conflicting negotiation between relevant investigators. While Powell and Brewer (9) showed no significant difference between corresponding mean levels of parasitaemia in G6PD deficient people compared to G6PD normal individuals, Gilles and co-workers found that the incidence of G6PD deficiency in children with sever malaria is significantly lower than that of control children (5). In another investigation, 700 children of both sexes from a rural area of holoendemic malaria *(P. falciparum*) were studied (10). The investigators found that there was no evidence that enzyme-deficient subjects had any greater resistance against malaria. In three cross-sectional studies using IFA and the blue dye, decolorization G6PD tests by Edrissian *et al.* in Hormozgan Province, a malarious area in southern Iran; different results were found out. In the first study, the G6PD deficient individuals had significantly lower seropositive rate and considerably lower total geometric mean of reciprocal titers with *P. falciparum* antigen as compared to the G6PD normal subjects, but not in *P. vivax* antigen. However, in the second and third studies no such distinct serological differences between G6PD deficient patients with falciparum malaria and G6PD normal subjects were observed (11). Our reported case, parasitologically, was alike to those were found out by Edrissian *et al.* in the second and third studies.

Acknowledgements

Authors would like to thank Mrs A Motavali Haghi and staffs of medical lab of Bou-Ali Hospital for their technical assistance.

References

1. Motulsky AG. Metabolic polymorphism and the role of infection disease in human evolution. Hum Biol. 1960; 32, 28.
2. Allison AC. Glucose-6-Phoshate dehydrogenase deficiency in Red blood cells of East Africans Nature. 1960; 186, 531.
3. Gilles HM, Taylor BG. The existence of the glucose-6- phosphate dehydrogenase deficiency tract in Nigeria and its clinical implications. Ann Trop Med Parasit. 1961; 55: 64.
4. Harris R, Gilles HM. Glucose-6-phosphate deyhdrogenase deficiency in the people of the Niger Delta. Ann. Hum. Genet. 25:199. Quoted from: Jranian J Publ Health (1983). 1962; 12(1-4): 9-25.
5. Gills HM, Fletecher KA, Hendrickse RG, Lindne R, Reddy S, Allan N. Glucose-6-phosphate-dehyrogenase deficiency, Sickling, and Malaria in African children in South-Western Nigeria. The lancet. 1967; 139-40.
6. Krautrachue M, Charoenlarp P, Chongsuphajaisiddhi T, Harinaluta G. Erythrocyte glucose-6-phosphate dehydrogenase and malaria in Thailand. Lancet. 1962; 11: 1343.

7. Bienzle U, Guggenmoss-Holzmann I, Luzzaltto L. Malaria and erythrocyte glucose-6- Phospate dehydrogenase variants in West Africa. Am J Trop Med Hyg. 1979; 28: 619.
8. Martine SK, Miller LH, Alling D, Okoye VV, Esan GJF, Osunkoya BO, Deane M. Sever malaria and glucose-6-phosphate dehydrogenase deficiency: A reappraisal of the malaria G-6- PD hypothesis. Lancet. 1979; 524.
9. Powell RD, Brewer GJ. Gloucose-6-phosphate dehyolrogenase deficiency and faciparam malaria. Am J Trop Med.1965; 14: 3.
10. Bienzle V, Ayeni O, Lucas AO, Luzzato L. Glucose-6-phosphate dehydrogenase and Malaria. Lancet. 1972; 107-110.
11. Edrissian GhH, Montazemi K, Nasseri AR, Afshar A. Malaria antibodies and glucose-6-phosphate deydrogenase (G-6-PD) deficiency. Iranian J Publ Health. 1983; 12(1-4): 9.