



Journal of Medical Sciences

ISSN 1682-4474

science
alert

ANSI*net*
an open access publisher
<http://ansinet.com>

Research Paper

J. Med. Sci., 3 (4): 294-297

July-August, 2003

JMS (ISSN 1682-4474) is an International, peer-reviewed scientific journal that publish original article in experimental & clinical medicine and related disciplines such as molecular biology, biochemistry, genetics, biophysics, bio-and medical technology. JMS is issued six times per year on paper and in electronic format.

For further information about this article or if you need reprints, please contact:

Dr. Arsala Mansoor
Biochemistry Department
Bolan Medical College
Quetta, Pakistan

Blood Biochemical Analysis of Afghan Malaria Patients

Arsala Mansoor, ¹Shah Mohammad Mari and Sadia Nasir

The hazardous biochemical effects of the malarial parasite *Plasmodium vivax* and *Plasmodium falciparum* was compared in the blood of Afghan patients. The role of glucose-6-phosphate dehydrogenase (G-6-PD) and immunoglobulins (Igs) was also detected in the increased incidence of malaria in Afghans. Blood biochemical analysis for bilirubin, serum glutamate pyruvate transaminase (SGPT), G-6-PD and Igs was conducted in hundred Afghan malaria patients coming in Malaria Control Centre, Quetta. Patients were grouped according to the form of species present. An increase of 1.0 - 2.0 mgs% of Bilirubin was observed in 14% patients, while rise in SGPT was associated to 50% patients. None of the patients were found to be deficient in G-6-PD activity. The most significant finding was a low rise of Igs, limited to less than 50% patients only. Thus, *P. falciparum* appears to produce more hazardous effects.

Key words: Malaria, Afghan patient, biochemical analysis

ANSI*net*
Asian Network for Scientific Information

Biochemistry Department, ¹Pathology Department,
Bolan Medical College, Quetta, Pakistan

Introduction

About 400 million people living in the whole of tropical Africa, much of Central and South America and most of the countries of Southern Asia and South-West Pacific are still exposed to malaria, resulting in over 1.5 million deaths a year (Bruce, 1980). It is still recognized as a growing hazard for South Asian countries. Malaria in Balochistan is considered to be seasonal and stable. Until seventies it was not considered to be a public hazard in this part of the world. In the past few years after the influx of the Afghan refugees there are confirmed statistics of gradual increase of malaria cases around their residential areas, such as Quetta. The resurgence of Malaria is accompanied with rise in *Plasmodium falciparum* cases. The carriers of the new infection may be asymptomatic, but their reservoir of gametocytes might have been highly infective to the local Anopheles (Mansoor, 2003).

Malaria epidemic takes place amongst a population under severe malnutrition or due to a decline in human resistance to infection. In order to overcome an infection of such a tricky parasite as plasmodium, the host uses a complicated immune system employing various alternatives for eliminating the parasite (Ruwanda *et al.*, 1995). Plasma immunoglobulins play a major role in body's defense mechanism. In most humoral responses antibodies with identical specificity but of different class are generated in a specific chronological order.

Since the increase in cases of malaria has been reported after the migration of Afghans; therefore a comparative study of sick and healthy Afghans was conducted to find the concentration of Igs, G-6-PD and other important constituents of blood like bilirubin and SGPT that might be affected by malaria.

Materials and Methods

This study was carried out in Malaria Control Centre, Quetta. For the investigation of the type of malarial parasite present, the patient's blood was examined with the help of thick and thin slides prepared by the routine method and stained by Giemsa staining. 4 ml of blood was collected from confirmed hundred male Afghan malarial patients, for the detection of bilirubin, SGPT, G-6-PD and Igs. Only those patients were selected who were not very weak and were between the ages of 15-60 years. ½ ml blood of each patient was separated in small vials containing ethylene diamine tetra acetic acid (EDTA) reagent. Rest of the blood was kept in plain vials. Whole blood was used to estimate the concentration of the enzyme G6-PD. After careful examination of the parasite by thick and thin slides, the patients were grouped according to the type of parasite present.

Blood of 10 healthy male Afghans was also analyzed to compare the results. Bilirubin and SGPT analysis were carried out by the standardized kit methods of Merck. Concentrations of Igs and G-6-PD were determined by kit methods provided by Spinreact and Trizma respectively.

Results

Microscopic examination

The largest group of 38 patients were enlisted under *P. vivax trophozoite* gametocyte as shown in Table 1. *Vivax trophozoite* was positive for 21 patients, *falciparum* gametocyte for 10

Table 1: Mean values of blood biochemical analysis±SEM of Malaria patients and the Controls

Type of Parasite	No of patients	S. Bilirubin mg dl ⁻¹ ±SEM	SGPT u L ⁻¹ ±SEM	IgG mg dl ⁻¹ ±SEM	IgA mg dl ⁻¹ ±SEM	1 gM mg dl ⁻¹ ±SEM
P.f.r.	31	0.99±0.33	39±13	1346±348	243±95	115±52
P.f.g.	10	1.1±0.28	46±8	1295±200	200±53	106±16
P.vt.	20	0.78±0.22	30±10	1700±156	312±28	100±44
P.vt.	43	0.95±0.35	43±5	1644±185	292±36	88±14
Healthy Controls	10	0.38±0.24	15±6	842±102	163±44	76±20

SEM: Standard Error of Mean

P.f.r: *Plasmodium falciparum* ring

P.f.g: *Plasmodium falciparum* gametocyte

P.v.t: *Plasmodium vivax* trophozoite

P.v.t.g: *Plasmodium vivax* trophozoite gametocyte

patients and *falciparum* ring for 31 patients. Analysis of liver function test included bilirubin and SGPT only, alkaline phosphatase was not determined. Amongst the total patients studied, a 14% increase of bilirubin was observed, which was from 1.0 to 2.0 mgs%. Most of the values of serum bilirubin were towards the higher limits of the normal range. 15% of the total cases were on the highest limit of the normal range i.e. 0.90 to 0.99 mgs%. The highest concentration of 2.0 mgs% was positive for falciparum gametocyte, while the maximum numbers of higher cases for bilirubin were present in falciparum ring infection. This lack of immunity might be the most probable reason for high malaria incidence in Afghans.

A correspondingly similar increase was observed in SGPT and reached as high as 60 u l⁻¹. Slight increase from the normal values of SGPT was more prevalent in most of the patients than the values of bilirubin. Almost 56% of the total cases were more than 30 u l⁻¹. Analysis of the values for G-6-PD indicates that none of the patient was found to be deficient in G-6-PD.

Almost 50% of the total cases were more than 30 U l⁻¹. Analysis of the values for G-6-PD indicate that none of the patient was found to be deficient in G-6-PD. Three types of immunoglobulins IgG, IgA and 1 gM having specific activity of malarial antibodies were detected in this study. The concentration of Igs in the patients presented an increased value due to malaria infection. This increase is calculated in terms of percent increase of total patients. IgA being present in the highest concentration was 48%, slightly less IgG 45% and the least amount of 34% was present in 1 gM. The rise in Igs was associated to less than 50% patients.

Discussion

The raised values of bilirubin and especially SGPT were observed in malaria caused by both the parasites. This shows that liver function is impaired during the disease, causing an increase in bilirubin and SGPT. Higher values were present in patients having *P. falciparum* parasite. This indicates the dangerous effects of falciparum malaria. Jaundice of the hepato cellular type is common in falciparum malaria but is usually mild, occasionally severe.

The results obtained for G-6-PD were quite shocking. Some studies have found Afghans and also 2.6% Pakistanis to be deficient in this enzyme (Ruwanda *et al.*, 1995; Insiripong, 1993). This deficiency was equally distributed amongst all the ethnic groups at that time i.e. Bengalis, Punjabis and Pathans. However conclusions in this regard had been conflicting. Attempts have been made by a number of Scientists to confirm that G-6-PD deficiency is protective against

malaria as shown by Ruwanda *et al.* (1995) but still a larger number are of the view that there is no protective effect against malaria in G-6-PD deficiency (Thakar *et al.*, 1997); rather suffer to a greater extent. It has been suggested that there are clinical manifestations of G-6-PD deficiency that are related to other tissues, but the existence of these is not well documented.

Plasmodial infection constitutes an intensive form of antigenic stimulation which leads to accelerated rates of immunoglobulin synthesis, associated with greatly increased levels of serum immunoglobulin (Bruce, 1971).

It has been observed by Collins (Carter, 1979). that primary infection in volunteers of malaria caused an increase in IgG, IgA and 1 gM within the first week. Clinical as well as experimental studies have shown that malarial infection leads to markedly increased Igs level, and to the production of antimalarial antibodies, providing a control of malarial infections. Although the antibodies act mainly against the five developmental stages of the parasite namely: sporozoites, trophozoites, schizonts, merozoites and gametocytes; but a significant increase in IgG, 1 gM and I gA has been observed with the onset.

References

- Bruce, C.L.J., 1971. Malaria epidemiology. *Brit. Med. J.*, 2: 91-93.
- Bruce, CL.J., 1980. Essential Malariology. William Heinemann Medical Books Ltd. London.
- Carter, R.W., 1979. Naturally acquired immunity and antimalarial antibodies in relation to infectivity to mosquitoes in endemic *P. falciparum* malaria. Transactions of the third meeting of the scientific group on the immunology of malaria. Panama, pp: 137-154.
- Insiripong, S., P. Tulayelak and C. Amatachaya, 1993. Prevalence of Thalassemia Haemoglobinopathies and G-6-PD deficiency in malaria patients. *J. Med. Assoc. Thai.*, 76: 554-8.
- Mansoor, A. 2003. Resurgence of Malaria in Quetta (in press). *Pak. J. Med. Sci.*
- Ruwanda, C., S.C. Khoo, R.W. Snow and S.M. Yates, 1995. Natural selection of hemi and heterozygotes for G-6-PD deficiency by resistance to severe Malaria. *Nature*, 37: 246-49.
- Thakar, Y.S., C. Chande, A.G. Dhanvijay, S. Pande and A.M. Saoje, 1997. Analysis of Immunodeficiency cases: A five year study. *Indian J. Pathol. Microbiol.*, 40: 309-313.