Journal of Medicine and Medical Science Vol. 2(5) pp.876-878, May 2011
Available online @http://www.interesjournals.org/JMMS

Short communication

Changes in serum vitamin c concentration by *P. falciparum* malarial infection in man

Uzuegbu U. E.

Department of Medical Biochemistry, Delta State University, Abraka, Nigeria.

Email: efiyugos@yahoo.com; Tel: +2348037199279, +2348057924906, +2348024488278

Accepted 15 January, 2011

Falciparum **malarial infection is associated with significant destruction of erythrocytes. This leads to the release of toxic products, including oxidant compounds, yet information on the influence of malarial severity on vitamin c level has remained scarce in our society. The plasma concentration of the antioxidant, ascorbic acid, was measured in 80 patients with varying degrees of malarial parasitaemia and presenting with acute *falciparum* malarial infection using standard procedure. Twenty (20) healthy individuals matched in age and sex was included as control. Results show that overall *falciparum* malarial infection reduced plasma vitamin c concentration. However, severe *falciparum* malarial infection significantly reduced ($P < 0.05$) plasma vitamin c level when compared with either mild or moderate infection, irrespective of sex and age, but effects were more marked among the infected children. Supplementation should be given and biochemical effects assessed in order to modify the chemotherapeutic regimen for the treatment of *falciparum* malarial infection.**

Keywords: Serum, Vitamin C, Malaria, Parasitaemia.

INTRODUCTION

Malaria is a vector – borne infectious disease caused by protozoan especially *plasmodium falciparum*. It is wide spread on tropical and subtropical regions including parts of America (South America), Asia and Africa. Each year, there are approximately 350 – 500 million cases of malaria, killing between one and three million people, the majority of whom are young children in sub – Saharan Africa (Snow et al., 2005). Ninety percent of malaria – related deaths occurs in sub – Sahara Africa (ISL, 2008). Malaria is commonly associated with poverty, but is also a cause of poverty and a major hindrance to economic development.

Malaria is caused by protozoan parasite genus *plasmodium.* Five species of the plasmodium parasite can infect humans; the most serious forms of the disease are caused by *plasmodium falciparum.* Malaria caused by *plasmodium vivax, plasmodium ovale* and *plasmodium malariae* cause milder disease in humans that is not generally fatal. A fifth specie *plasmodium knowelsi,* cause malaria in macaques but can also infect humans. This group of human – pathogenic *plasmodium* species is usually referred to as malaria parasites.

People get infected with malaria parasites, by being bitten by an infective female *anopheles* mosquito. Only *anopheles* mosquito can transmit malaria, and they must have been infected through a previous blood meal taken from an infected person.

When a person is infected, some symptoms can be observed which include, anemia, as well as fever, and death. Transmission can be reduced by preventing mosquito bites with mosquito nets and insect repellants and removing/draining of stagnant water.

Work has been done on malaria vaccines with limited success and more exotic controls, such as genetic manipulation of mosquitoes to make them resistant to the parasite have also been considered (Voshida et al., 2007).

Falciparum malaria causes destruction of the erythrocytes. Antioxidants in the body help in the fight against free radicals which is increased when an infection including malaria infection abounds (Des et al., 1986). Ascorbic acid which is one of the known antioxidants is very essential. The main action of ascorbate is functioning as a reducing agent. It reverses oxidation in aqueous solution. When there are more free radicals in the body versus antioxidants, human is under the

Table. 1: Changes in serum ascorbate content by mild, moderate and severe parasitaemia

Age (yrs)	5 – 15		16 – 45	
Sex	M	F	M	F
Parasitaemia /µL	change in plasma vitamin level		(mg/dl)	
Mild				
[498.67 ± 103.40]	0.98 ± 0.20	0.59 ± 0.10	1.98 ± 0.21	1.21 ± 0.81
Moderate				
[3748.38 ± 1310.60]	0.54 ± 0.24	0.71 ± 0.34	1.01 ± 0.24	0.98 ± 0.16
Severe				
[9954.60 ± 4279.40]	0.28 ± 0.88	0.32 ± 0.22	0.38 ± 0.18	0.41 ± 0.33
Control				
[Nil]	1.41 ± 0.23	1.66 ± 0.22	1.34 ± 0.39	1.37 ± 0.51

condition – oxidative stress (*McGregor* and *Biesalski* 2006). Oxidative stress induces a lot of other diseases (*Kelly* 1998, *Mayne* 2003). The plasma ascorbate concentration in oxidative stressed patients (less than 0.5µL) is lower than that of healthy individuals (*Tak et al*., 2000).

Inspite of the association between malaria infection and oxidative stress, the effect of malaria parasitaemia on the levels of serum vitamin c is yet to be fully documented in our society. This paper reports the investigation into the influence of *P. falciparum* count on the changes in vitamin c concentration in Mushin area, of Lagos State, Nigeria.

MATERIALS AND METHODS

Study Period and Centre

This study was carried out between May and September, 2009 in the Tropical Disease Unit, Department of Medical Biochemistry, Delta State University, Abraka, Nigeria. Patients were selected from the General Hospital, Mushin area of Lagos State, Nigeria.

The study population/group consisted of a hundred patients, who included children and adults between the ages of 5 and 45 years. The subjects were symptomatic (presenting with symptoms of malaria) as at the time of obtaining the blood samples.

Permission and ethical approval were given by the Hospital Management Board.

Malarial parasite count

P. falciparum parasitaemia was determined in various blood smears stained by Giemsa stain. The parasitaemia was graded as low (1 – 999/µL) , moderate (1000 - 9999/µL) and severe (>10,000/µL).

Serum vitamin c analysis

The plasma ascorbate level was determined using 2, 4 – dinitrophenyl hydrazine method (*Teiz* 1986)

Statistics

Data were expressed as mean ±SD and analysed by ANOVA followed by Dunnet's post hoc test using SPSS version 10.0. Level of significance was set at $p<0.05$.

RESULTS

The results obtained are shown on Table 1. Table 1 indicates the changes in serum ascorbate content by mild, moderate and severe parastaemia.

From table 1, the mean plasma parasitaemia in infected children and adults was found to be 498.67 ± 103.40 mg/dL, 3524.20 ± 1151.90 mg/dL, in severe cases it was found to be 9954.60 ± 4279.40mg/dL, the mean plasma ascorbate concentration in mild cases was found to be 0.98 ± 0.20mg/dL in male children and in female children 0.59 ± 0.10mg//dL. In adult male it was found to be 0.98 ± 0.21, in female it was found to be 0.21 ± 0.81 mg/dL. In moderate cases the mean plasma ascorbate in male children was found to be 0.54 ± 0.24 mg/dL, in female 0.71 ± 0.34 mg/dL, in adult males, it was 1.01 ± 0.24 mg/dL and in female 0.98 ± 0.16 mg/dL. In severe cases in male children plasma ascorbate was found to be 0.28 ± 0.88mg/dL and in female 0.41 ± 0.33mg/dL. Among the control subjects, plasma ascorbate level for the male children was found to be 1.41 ± 0.23mg/dL and in females it was 1.66 ± 0.22mg/dL in male adults it was found to be 1.34 ± 0.39mg/dL and in females, it was found to be 1.37 ± 0.51mg/dL.

From the presentation above, it was found that the mean ascorbate level for adults was found to be higher than that of children. In comparison to their control, it was found that the ascorbate level of adult control was higher than that of the children.

From these results, a clear reversal trend was observed and the plasma ascorbate level was considerably higher in control children than in control adults.

DISCUSSION

The plasma ascorbate concentration was higher in healthy children (5 – 15yrs) than the adults (16 – 45yrs, table 1). This is due to the fact that children require vitamins for both growth and maintenance (*Kallner et al*., 1979). Evidence from this study indicates that vitamin c could play important roles on the pathogenesis of *falciparum* malaria infection. Considering earlier reports from various workers (*Thomas* and *Holt* 1978, *Kallner* 1971, *Greenwood* and *Mutabingwa* 2002, *Kremsner, et al*., 2000) on the role of vitamins on the immune system, higher levels of the plasma concentration of vitamin c seen in adult *falciparum* malaria patients compared with the children's level may be part of the machinery aimed at boosting host immunity. Reason for this is leucocytes are known to participate as components of cell – mediated immunity in the early response of the host to *falciparum* malarial infection (*Bruce – chiwatt* 1985). also, it has been observed that while serum (or plasma) levels follow the circadian rhythm or short term dietary changes, those within the tissues themselves are more stable and give a better view of the availability of ascorbate within the organism (*Emadi – konjin et al.,* 2005, *Yamada et al.,* 2004). From all that have been observed, it can be seen that since leucocytes are the best store for ascorbate and they are also mediated when the *falciparum* malaria infection occurs, it can be a very good reason why the level of leucocytes is increased in infected patient, than their uninfected counterpart. The release of the ascorbic acid from leucocytes is due to response to parasite induced stress. Also, the release toxic substance/product, including oxidant compounds, as a consequence of erythrocytic merogany, and the homolysis of red blood cells, may impose a demand on the patient for increased mobilization of antioxidants (vitamin c). However, considering the complex nature of the pathogenic processes associated with *falciparum* malaria infection (Miller et al., 2002) either factors may be responsible for the increased concentration of ascorbate in infected patients. The level of serum ascorbate in infected patients was above the range 0.5 – 2.0mg/dL (*Kapan* and *Pesce* 1989). Proves evidence that *falciparum* infection in adult patients mobilize the tissue stores of the antioxidant as apart of early response to the infection. *Falciparum* malarial is known to be associated with depressed immune function on children 5 and a little above 5 years of age, this may also be responsible for the reduced concentration of plasma ascorbate and immune function, as evidence shown by early reports (*Goets et at*., 2000).

A general inference could be made and advice can be to a reasonable point given that, medical personnel should try and include vitamins especially vitamin c supplements in their dietary formulation which is the case in developed countries, whereby vitamin supplements are compulsorily added to food products

REFERENCES

Bruce – chwatt LJ, (1985). Essential Matariology, oxford university press, Oxford.

Das B S, Thurnham DT, Das DB (1996). Plasma alpha – tocopherol, retinol and carotenoids in children with *falciparum* malaria. *Am. J. Clin. Nutr.* 64: 94 – 100.

Emadi – konjin P, Vorjee Z, Levin A, Adeli K (2005). Measurement of intracellular vitamin c levels in human lymphocytes by reverse phase high performance liquid chromatography (HPLC). *Clin. Biochem.* 38 (5):450 – 456.

Galloway P, Mcmillan C, Scatter N (2000). Effect of the inflammatory response in trace element and vitamin status. *Ann Clin Biochem.* 37 : 289 – 297.

Goets E J, Wasserman SI, Austen K F (1994). Enlargement of random migration and chemotatic response of human leucocytes by ascorbic acid. *J. Clin Invest.* 53 : 813 – 818.

Greenwood B, Mutabingwa T (2002). Malaria in 2002. *Nature*. 415:670 – 672.

Kallner A, Hartman D, Horing D (1979). Steady state turnover and the body pool of ascorbic acid in man. *Amer. Clin. Nutri.* 32 : 530 – 539.

Kaplan LA, Pesce AJ (1989). *Clinical chemistry*: theory, analysis and correlation. 2nd (ed). The C. V. Mosby company, S. Louis, Missouri.

Kelly FJ (1998). Use of antioxidant in the prevention and treatment of disease. *J. Int. Fed. Clin. Chem.* 10 : 21 – 23.

Kremser PG, Greve B, Lell B, Lackner D, Schmid D (2002). Malaria anemia in Africa children associated with high oxygen radical production. *Lancet*. 355 : 40 – 41.

Mayne ST (2003). Antioxidant nutrients and chronic disease; use of biomarkers of exposure and oxidative stress status in epidemiologic research. *J. Nutr.* 133 (suppl.3) : 9335 – 9405.

McGregor GP, Biesalski HK (2006). Rationale and impact of vitamin c in clinical nutrition. *Curr. Opin. Clin. Nutr. Matab. Care.* 9 : 697 – 703

Miller LH, Baruch DI, Marsh K, Dounbo OK (2002). The pathogenic basis of malaria. *Nature.* 415 ; 673 – 679.

Siegel BV (1975). Enhancement of interferons production by poly (Ri), poly (Rc) in mouse cell cultures by ascorbic acid. *Nature*. 254 : 531 – 536.

Snow RW, Guerra GA, Noor AM, Myint HY, Hay SI (2005). The global distribution of clinical episodes of plasmodium *falciparum* malaria. *Lancet. Infect. Dis*. 4(6) : 327 – 336.

Tak PP, Zraifer NJ, Green DR, Firestain GS (2000). Rheumatoid arthristis and p53: how oxidative stress might alter the course of inflammatory disease. *Immunol. Today*. 20 : 78 - 82

Teic NW (1986). Methods of determination of ascorbic acid. *Textbook of clinical chemistry*. Teic, N. W. (ed.), W. B. Saunders company, Philadelphia pp. 960 – 962.

Thomas WR, Holt PG, (1978). Vitamin c and immunity, an assessment of the evidence. *Clin. Exp. Immuno.* 32 : 370 – 376.

Wenner RF (1971). Observations on the dose and administration of ascorbic acid when employed beyond the range of a vitamin in human pathology. *J. Appl. Nutr.* 23 : 1 – 31.

Yamada H, Yamada K, Waki M, Umegaki K (2004). Lymphocyte and plasma vitamin c levels in type 2 diabetic patients with and without diabetes complications. *Diabetes Care* 27 : 2491 - 2492

Yoshida S, Shimada Y, Kondoh D (2007). Hemolytic c – type lectin cell – III from sea cucumber expressed in transgenic mosquitoes impairs malarial parasite developmenet. *Plus pathology* 3(12) : 271 – 274.