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ORIGINAL ARTICLE

A STUDY OF CHANGES IN LIVER FUNCTION BIOMARKERS AMONG MALARIA INFECTED PATIENTS

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ABSTRACT: Malaria is one of the commonest parasitic diseases in the tropical countries. Hepatic compromise has been identified in malaria infection but information in urban Bangalore is very limited, hence this study was carried out. Serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP) activities, bilirubin (total & direct), total protein (TP) & albumin (ALB) levels were assayed in 36 patients presenting with malaria infection & 36 subjects without malaria infection. The malaria patients & control subjects were matched for age and sex. Patient selection was done by simple random sampling from those presenting at VIMS & RC, Bangalore, with a history of fever, chills & malaise & who were subsequently confirmed to be malaria positive by Geimsa stained peripheral blood film. Results of the study shows, increase in the values (except ALB and TP) for malaria infected patients were significant ($p<0.05$) when compared with the values for the non-infected subjects. Evidence from this data indicates a measure of liver dysfunction among the malaria infected patients. Post-treatment data need to be documented in order to advise health care providers.

KEYWORDS: Albumin, Enzyme, Liver, Malaria, Protein.

INTRODUCTION: Malaria has been and is still the cause of human morbidity & mortality. Karnataka is one among the most affected states and Bangalore is one of the 15 major cities including 4 metropolitan cities that account for nearly 80% of malaria cases covered under urban malaria control schemes. Malaria is one of the commonest parasitic diseases in tropical countries characterized by febrile paroxysm occur with definite intermittent periodicity repeating every third or fourth day depending upon the species of the parasite involved.[1] Malaria is a disease transmitted by the female Anopheles mosquito. The disease is caused by protozoan parasites of the genus Plasmodium. Four species of the Plasmodium parasite can infect humans.[2] Malarial transmission to the human host is established by sporozoites infection to the liver. The malarial sporozoites once injected in blood by the bite of female Anopheles mosquitoes are attached to hepatocytes through receptor for thrombospondin and properdin.[3] Liver dysfunction has been recognized in malaria infection[4] but information about geographical variation in urban Bangalore is scarce. This research attempts to report the changes in liver function biomarkers in malaria infected patients in urban Bangalore.

MATERIALS AND METHODS: Study centre and period: This duration based study was done from January 2012 to March 2013 in Department of Biochemistry, VIMS & RC, and Bangalore.

SUBJECT SELECTION: Patients selection was done by simple random sampling from those presenting at VIMS & RC, Bangalore, with a history of fever, chills and malaise & who were subsequently confirmed to be malaria positive by Geimsa stained peripheral blood film. Based on the

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following selection criteria 36 patients were selected. 36 subjects in apparent good health and malaria parasite negative were included as control individuals. Consent was sought and obtained from both cases and controls.

EXCLUSION CRITERIA: Patients on self-medication with any anti-malarial drugs prior to presentation and patients with any type of liver disease were excluded from the study.

SPECIMEN COLLECTION AND ASSAY: 5ml of venous blood sample was taken by aseptic precaution. Samples were centrifuged and serum AST, ALT, ALP, TP, ALB, TB & DB were measured by using auto analyzer Beckman Coulter Synchron DxC by using Beckman Coulter Kits (USA).

STATISTICAL ANALYSIS: The data obtained were analyzed using the student's t test and level of significance was set at $p<0.05$ and were expressed as Mean ± Standard Deviation (SD)

RESULTS: Table 1 shows the changes in liver function biomarkers of the malarial (case) and control subjects. When the malaria positive (test) patients were compared with the non - infected subjects, there was increase in the mean activity values of the various liver enzymes, serum total and direct bilirubin. However, serum total protein and albumin levels were reduced among the malaria infected patients.

Changes in liver enzymes (AST, ALT and ALP) activity and total & direct bilirubin values for the malaria patients were significantly ($p<0.05$) higher than those for the non - malaria infected subjects.

Variables	Malaria cases (N=36) Mean ± SD	Controls (N=36) Mean ± SD	P-value
Total Bilirubin(mg/dl)	1.96 ± 3.47	0.53 ± 0.20	0.023*
Direct Bilirubin(mg/dl)	0.84 + 1.85	0.09 + 0.05	0.026*
Total Protein(g/dl)	7.64 + 9.34	7.10 + 1.30	0.749
Albumin(g/dl)	2.80 + 0.34	4.37 + 0.46	6.551
AST(IU/L)	38.75 + 20.32	24.72 + 5.31	0.000*
ALT(IU/L)	26.92 + 8.73	19.88 + 6.76	0.000*
ALP(IU/L)	77.19 + 28.18	94.59 + 37.3	0.032*

Table 1: Liver function biomarkers of cases and controls

*Significant

DISCUSSION: Malaria is endemic in India with an estimated 70-100 million cases each year. Of these 45-50 % are plasmodium falciparum.[6]

Malaria is a mosquito-borne tropical disease caused by the Plasmodium species of protozoa. It affects mainly the hepatocytes and red blood cells (RBCs) and manifests clinically as fever and splenomegaly.[7]

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LIVER DISFUNCTION: Mild hemolytic jaundice is common in malaria, severe jaundice is associated with P. falciparum infections, is more common among adults than among children and results from hemolysis, hepatocytic injury and cholestasis. When accompanied by other vital organ dysfunction (often renal impairment) liver dysfunction carries a poor prognosis. Hepatic dysfunction contributes to hypoglycemia, lactic acidosis and impaired drug metabolism. The hemolysis results from the invasion of the erythrocytes by malarial parasites. P. malariae is known to invade mature RBCs, whereas P. vivax and P. ovale are known to invade young RBCs. P. falciparum parasites are capable of invading RBCs of any age and hence can lead to very high levels of parasitemia; while parasitemia is limited in all other types of Plasmodium infections.[8]

In this study, it was observed that the values for liver function profiles among patients with malaria were elevated when compared with those without infection. The observed increase ($p<0.005$) in serum liver enzymes (AST, ALT and ALP) could be due to leakage from hepatic cells that were killed or injured by the auto - immune progress and /or by abnormal cell activation induced by the parasites. This finding supports previous reports.[2]

Total protein and albumin levels were reduced in malaria patients may be due to the fact that liver, which is the primary organ for protein synthesis, is the site for parasite multiplication in case of malaria. It can also be a part of the acute phase response. The reduced TP & ALB finding supports previous reports.[3]

According to WHO, jaundice is one of the important manifestations of severe malaria. There is evidence of focal hepatocyte necrosis, cholestasis, bile stasis, granulomatous lesion or malarial nodules.[4] Anand et al conducted a study on 39 patients with falciparum malaria and jaundice, out of whom 13 had serum bilirubin around the mean value of 16.3%, and most had predominantly conjugated hyperbilirubinemia[5] Especially noted is the elevation of alanine transaminase to more than three times the upper limit of normal value.[9] The elevation of serum transaminases in falciparum malaria patients is more than 5 times the upper limit of reference range.[10]

CONCLUSION: Hepatic involvement is a common accompaniment of acute malaria infection, and hepatic dysfunction ranges from a mild elevation of liver enzymes to the range of acute hepatitis. The presence of hepatitis in malaria patients indicates a more severe illness with a higher incidence of complications, multiorgan failure and a bad prognosis.

REFERENCES:

1. Epidemiology of Communicable Diseases. In: K. Park K. Park's text book of Preventive and Social Medicine, 20th edition. Jabalpur (India): Banarsidas Bhanot; 2009. 222-232.
2. U E Uzuegbu, C B Emeka. Changes in liver function biomarkers among malaria infected patients in Ikeja Lagos state, Nigeria. Current Research Journal of Biological Sciences, 2011; 3 (3): 172-174.
3. Muhammad Waseem Kausar, Khalida Moeed, Naghmi Asif, Farwa Rizwi, Sajid Raza. Correlation of bilirubin with liver enzymes in patients of falciparum malaria. International Journal of Pathology, 2010; 8 (2): 63-67.
4. Singh R, Kaur M, Arora D. A prospective study of hepatic involvement in plasmodium falciparum malaria. Journal of Clinical and Diagnostic Research, 2010 April; (4): 2190-2197.

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5. Anand AC, Ramji C, Narula AS, Singh W. Malarial hepatitis; a heterogenous syndrome? Nat Med J India 1992; 5: 59-62.
6. Preetam N Wasnik, TP Manohar, NR Humaney, HR Salkar. Study of Clinical Profile of Falciparum Malaria in a Tertiary Referral Centre in Central India. JAPI 2012, October; Vol. 60: 33-36.
7. Dilip Karnad. Malaria. In: Gurumukh S Sainani. A. P. I Text Book of Medicine, 6th Edition, Association of Physicians of India Mumbai, 1999; p.104-108.
8. Nicholes J, White, Joel G. Breman. Malaria. In: T R Harrison, E Braunwald, D L Kasper, S C Hauser, D L Longo, J L Jameson, Seph Loscalzo. Principals of Internal Medicine, 17th Edition, New York Mc Graw Hill; 2005; p. 1285.
9. Bhalla A, Suri V, Singh V. Malarial hepatopathy. J Postgrad Med 2006; 52: 315-320.
10. Srivastava A, Khanduri A, Lakhtakia S, Pandey R, Choudhuri G. Falciparum malaria with acute liver failure. Trop Gastroenterol 1996; 17: 172-174.

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