American Journal of Health, Medicine and Nursing Practice (AJHMN)



Effect of Malaria Parasite on Hepatic Parameters in People Living in Aba Metropolis

Ikpeazu V.O, Offiah S.A.U, Chikezie J, Chigbu L.N, Ekenjoku, A. J, Ugwa V.O. and Igboh N.M





Effect of Malaria Parasite on Hepatic Parameters in People Living in Aba Metropolis

Ikpeazu V.O¹, Offiah S.A.U¹, Chikezie J¹, Chigbu L.N¹, Ekenjoku, A. J¹, Ugwa V.O¹, and Igboh N.M^{1*}

¹College of Medicine Abia State University Uturu, Nigeria

*Corresponding Author's Email: <u>drngomi@yahoo.co.uk</u>

Abstract

Purpose: In Nigeria, malaria continues to be a huge public health problem. Incidentally, malaria is caused by either *Plasmodium falciparum* or *Plasmodium vivax*. Malaria associated deaths in Metropolis area is mainly due to *Plasmodium falciparum*. Plasmodium parasite is primarily transmitted by the bite of an infected female anopheles mosquito during blood meal though this can also occur through exposure to infected blood products (blood transfusion) and by congenital transmission. This study therefore aimed at investigating the effect of malaria parasite on hepatocytes by monitoring enzyme activities such as the transaminases and the excretory function of the liver (bilirubin).

Methodology: This study was a cross-sectional study where hundred adults participated. A total of 50 patients' samples (adults) both male and female were collected from the same geographic location and fifty were selected as control and another fifty for the test. Examination of a thick blood film stained with Giemsa was done to confirm the presence of plasmodium and its absence as control. The activities of Aspartate transaminase, Alanine transaminase and bilirubin level was determined using Redox enzymatic kit Standard methods. Data obtained were statistically analyzed using students t-test where P<0.05 was considered significant.

Findings: There was a positive relationship between the enzyme activities and the level of parasitemia (P<90.05). Derangement in AST (13.30 \pm 4.48U/L), ALT (13.9 \pm 4.52U/L), TB (3.92 \pm 1.30U/L), CB (0.56 \pm 0.28U/L) levels for the infected subjects were higher when compared with controls .This study has demonstrated that the invasion of the hepatocytes by the malaria parasite causes the liberation of the transaminase into the blood serum and equally, the excretory function through elevation of bilirubin of the liver was affected.

Recommendation: This study recommend transaminases and bilirubin to be assessed to assist in disease detection, prompt diagnosis and intervention especially in endemic malaria area.

Keywords: Plasmodium parasite, bilirubin, transaminases, sporozoites and malaria parasite.



INTRODUCTION

In Nigeria, malaria continues to be a huge public health problem. Majority of malaria infections are caused by either *Plasmodium falciparum* or *Plasmodium vivax*, and most malaria associated deaths are due to Plasmodium falciparum (Ezeji *et al.*, 2010). Plasmodium parasite is primarily transmitted by the bite of an infected female anopheles mosquito during blood meal, though, this can also occur through exposure to infected blood products (blood transfusion) and by congenital transmission. In industrialized countries, most cases of malaria occur among travelers, immigrants, or military personnel returning from areas of endemic for malaria.

The liver is an important organ involved during the hepatic stage of malaria parasites life cycle, where malaria sporozoites develop into merozoites. Liver dysfunction is a common complication due to the invasion of liver cells by sporozoites. The change caused in hepatic cells by sporozoites can lead to leakage of parenchyma and membranous enzymes of the liver into the circulatory system, which can be responsible for the increase in the liver enzymes.

Millions of deaths due to malaria occur annually. Malaria kills one child every 30 seconds and about 300 children daily. Of all these deaths, the overwhelming majority is among children aged 5 years and below. Malaria account for 90% of the deaths each year in rural Sub-Saharan Africa. At least 20% of all deaths in children under five in Africa is due to malaria (Mishra *et al.*, 2010). The classic malaria paroxysms, in which fever spike, chills, rigors occur at regular intervals, are relatively unusual and suggest infection with plasmodium vivax or plasmodium ovale (Dennis *et al.*, 2005).

The liver is an important organ involved during the hepatic stage of malaria parasites life cycle, where malaria sporozoites develop into merozoites. Liver dysfunction is a common complication due to the invasion of liver cells by sporozoites during malaria parasites life cycle. The change caused in hepatic cells by sporozoites can lead to leakage of parenchyma and membranous enzymes of the liver into the circulatory system, which can be responsible for the increase in the liver enzymes (Burris *et al.*, 2001; Lopez-Rodnguez *et al.*, 2007). Malaria has been implicated as one of the factors responsible for human renal and hepatic dysfunction in malaria endemic countries. World Health% Organization (WHO, 2012) estimates that in 2010 there were 219 million cases of malaria resulting in 660,000 deaths. In sub-Sahara Africa, maternal malaria infections figure up to 200,000 estimated infant death yearly.

Liver involvement in malaria is common in patients with severe malaria and may manifest as jaundice, hepatomegaly and elevated liver enzymes like Aspartate Amino Transferase (AST) and Alanine Transferases (ALT), hyperbilirubinemia. It is attributed to hemolysis of both parasitized erythrocytes and partly due to liver damage. Therefore, well informed changes in blood and biochemical parameters in malaria infections and hepatic function enable the clinician to establish reliable and therapeutic intervention (Azubuike & Nkanginieme, 2016). Equally, liver involvement in malaria is a known entity with specific histopathological changes, which may altered liver function test, incidentally, fulminant hepatic failure and hepatic encephalopathy have been reported (Mishra et al., 2010).

Many studies have been done on serum hepatic biochemical parameters and malaria especially in adults within and outside Nigeria (Mishra et al., 2010). The limitation of most of these studies was the failure of these studies to correlate the changes in serum level of hepatic biochemical parameters with the degree parasitemia. This study is a cross-sectional study



cried out to determine the effects of degree of malaria parasite on serum liver enzymes. This is with a view to correlate degree of malaria parasite with serenity of hepatic injury.

MATERIALS AND METHOD

Hundred adults were used for the study. Fifty patients' samples (adults) both male and female were recruited from the same geographic location and fifty were selected as control and the other fifty for the test. Subjects with chronic diseases such as diabetes mellitus heart disease and cardiovascular diseases were excluded from the study while those free from the afore mentioned diseases met the inclusion criteria. Blood samples (5ml) were collected by venipuncture into EDTA and plain containers. A drop of whole blood was used for microscopic examination of malaria parasite by thick blood film and was stained with 10% Giemsa solution. The sample on the plain container was spun to obtain serum for the determination of serum activities of Transaminases (ALT and AST) as well as bilirubin concentration. Redox enzymatic kit was used for the determination of enzyme activities. Total and conjugated bilirubin concentration were determined using Diazo method. Data obtained were statistically analyzed using Students t-test where P-value is less than or equal to 0.05 (P=<0.05) which was considered as being statistically significant. Results were expressed as Mean \pm SD (standard deviation).

RESULTS AND DISCUSSION

Table 1 shows the comparison of Transaminases (AST and ALT) and bilirubin (TB and CB) levels among malaria positive patients and control. All the controls had normal levels of AST, ALT and bilirubin (TB and CB) while those with malaria parasites (positive patients) showed remarkable increase in the Transaminases activities and bilirubin level. (P<0.05).

Parameters	(TEST) 50 Mean ± SD	(Control) 50 Mean ± SD	P-value
AST	13.30±4.48	5.73±3.42	0.05
ALT	13.94±4.52	7.49 ± 4.50	0.05
TB	3.94 ± 1.30	2.01±0.60	0.05
СВ	0.56 ± 0.28	0.64±0.30	0.05

Table 1: Variations in serum level of transaminases and bilirubin of malaria patients and control patients.

The levels of the transaminases (AST and ALT) and total bilirubin were elevated in subjects with malaria parasite.

Key: SD: Standard deviation, P Value <0.95 is significant, P Value > 0.05 is not significant

The results showed a significant difference (p<0.05) between the levels of AST, ALT and bilirubin (Total and Conjugated) in malaria infected patients when compared with non-malaria infected group (control) which was higher in malaria infected patients. This could be attributed to the destruction of erythrocytes during the induced intravascular hemolysis of parasitized red cells during erythrocytic stage of the plasmodium parasite. Merozoites caused the destruction of the infected red blood cells prior to their differentiations into male and female gametocytes leading significant alterations in cell physiology morphology (Anand *et al.*, 2006).

This study conforms to the study of Olusegun (2015) who reported an increase in AST and ALT level in severe malaria patients when compared with control group which was attributed



to intravascular hemolysis of parasitized red caused by Plasmodium. Anurag et al. (2013) stressed that plasmodium infection may be responsible for the increase in the Transaminases (AST and ALT). Also, noted was a significant increase in the level of total and conjugated bilirubin among malaria infected patients compared to control. This was attributed to intravascular hemolysis caused by the destruction of the infected red blood cell at the erythrocytic stage (Sharma *et al.*, 2012). However, the study by Anand and Puri (2015) reported that hepatic dysfunction in malaria parasite infection, does not appear to be due to direct inflammation of hepatocytes but rather due to increased destruction of red blood cells when the parasite density is high and this destruction eventually occurs in the spleen. The resulting hemolytic anemia from this destruction also leads to increased plasma level of bilirubin.

The findings of this study revealed that the derangement in the levels of serum AST, ALT and Bilirubin (Total and Conjugated) observed in malaria positive patients does not conclusively imply liver diseases.

CONCLUSION AND RECOMMENDATION

It is also important that Transaminases and bilirubin be assessed to assist in disease detection, prompt diagnosis and intervention, especially in endemic malaria area is very necessary, where possible, people should sleep in mosquito treated nets.

Acknowledgment

Authors are grateful to the staff of Department of Chemical Laboratory, Abia State University for their technical assistance.

REFERENCES

- Anand A. C., and Puri P. (2015). Jaundice in Malaria. *Journal of Gastroenterology and Hepatology*, **20**: 1322-1332.
- Anurag, C., Asaranti, K., Dipannweeta, R. and Vidyut, P. D. (2013). Assessment of Abdominal Liver chemistry in malaria and dengue infection. *International Journal of Science and Research*, 4(4): 2412-2414.
- Azubuike, J.C. and, Nkanginieme.E.O (2016) Paediatrics and child health in atropical region .(3 rd Ed). *Educational printing and publishing. Lagos, Nigeria* 536-543
- Burris, C., Ashwood, E. and Border, B. (2001). Liver functions, In; Tiet,z, L. (5th Ed). *Fundamental of Clinical Chemistry*. Saunders Company, Philadelphia.748-770.
- Dennis L. K. Anthony S. F., Dan L. L. Eugene B., Stephen L. H., and Jameson J. L. (2005). *Harrison's Principles of Internal Medicine. Infectious Diseases;* **16**: 1218-1232.
- Ezeji G. C., Ezeadnachi E. N., Okay E. A., Female E. I., Ikpatta N. W. and Alaribe A. A. (2010). Malaria and its treatment in rural villages of Aboh Mbaise, Imo State, Nigeria. *Acta Tropica*, **48**: 17-24.
- Lopez-Rodnguez C., Torres D., Gone, R. M. and Portola J. (2007). Systematic febrile illness in patients coming from the tropics: analysis of the cases attended in the University General Hospital of Alicante, Spain. *Tropical Medicine and International Health*, 1(60): 25-78.
- Mishra S. K., Mohanty S, and Das B. S. (2010). Hepatic changes in P. Falciparum malaria. *Indian Journal of Malaria*; **29**: 167-171.



- Olusegun, M.A., (2015). Influence of Malaria infection on kidney and liver Function in Akoko Area of Ondo State, Nigeria. *Journal of Parasitology and Vector Biology* **7**(8): 163-68.
- Sharma,N.N.,Nand,H.K and Lata,S. (2012). Evaluation of liver function in Falciparum Malaria. *Journal% of international medical Sciences Academy*. **25**(4): 229-30.
- Warell D. A., Molyneux M. E., and Beales P. F. (2015). Severe and complicated malaria. *Transaction of Royal Society of Tropical Medicine Hygiene;* **84**(2): 1-65.
- WHO (2012). World Malaria Report (PDF). Retrieved 6 March 2016.
- WHO (2014). Malaria Fact sheet N⁰94. *Retrieved 28 April* 2016.