



Lipid Profile and Electrolyte Level in Malaria Patients Attending Muhammadu Abdullahi Wase Specialist Hospital, Kano State, Nigeria

**A. I. Kiru¹, R. K. Bala^{2*}, A. M. Abdulazeez², S. Y. Bello², A. L. Adam¹,
S. M. Suleiman¹, A. Shamsu¹ and M. L. Abdulkadir¹**

¹Department of Biochemistry, Faculty of Basic Medical Sciences, Bayero University, Kano State, Nigeria.

²Center for Biotechnology Research, Bayero University, Kano State, Nigeria.

Authors' contributions

This work was carried out in collaboration between all authors. Authors AMA and AIK designed the study. Authors ALA, SMS, AS and MLA carried out laboratory analyses and performed the statistical analysis. Author RKB wrote the protocol and the first draft of the manuscript. Author SYB managed the literature searches. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/JOCAMR/2018/41215

Editor(s):

(1) Sachin Kumar Jain, College of Pharmacy, IPS Academy, India.

Reviewers:

(1) Suparna Roy, Calcutta National Medical College, India.

(2) Emmanuel Ifeanyi Obeagu, Michael Okpara University of Agriculture, Nigeria.

Complete Peer review History: <http://www.sciencedomain.org/review-history/24600>

Original Research Article

Received 23rd February 2018

Accepted 2nd May 2018

Published 12th May 2018

ABSTRACT

Aim: This study was carried out to determine the serum lipid profile and electrolyte level and their association with severity of malaria among patients attending Muhammadu Abdullahi Wase Specialist Hospital, Kano State, Nigeria.

Methodology: Blood samples were obtained from four hundred (400) subjects: Two hundred and forty (240) pathological samples collected from malaria-infected patients and one hundred and sixty (160) from apparently healthy persons of the same age range with no evidence of malaria infection. Blood samples were screened for *P. falciparum* infection using both thin and thick film method, and severity of malaria was classified as described by World Health Organization. Also, the electrolyte (Na⁺, K⁺ and Cl⁻) level and lipid profile (TC, HDL-C, LDL, TAG and VLDL) were determined. Results

*Corresponding author: E-mail: rkbala.cbr@buk.edu.ng;

were presented as Mean \pm SD, evaluated by one-way ANOVA and the differences between the means assessed using Duncan's test. Statistical significance was considered at $P < 0.05$.

Results: From the results, there was significantly ($P < 0.05$) higher levels of TC, LDL, TAG and VLDL and lower levels of HDL in malaria-infected patients compared to control group. The lipid fragments (TC, LDL, TAG and VLDL) increased significantly ($P < 0.05$) with an increase in severity, while HDL decreased significantly ($P < 0.05$). Also, there was a significant ($P < 0.05$) decrease in the levels of Na^+ and K^+ while Cl⁻ was not significantly ($P > 0.05$) different in malaria-infected and control patients.

Conclusion: The study demonstrated that characteristic serum lipid profile and electrolyte changes occur during malaria, hence the need to correct these derangements is of great significance in the management of malaria infection.

Keywords: Electrolytes; lipid profile; malaria; patients.

1. INTRODUCTION

Malaria is a life-threatening disease and remains a public health concern throughout the world. According to the World Health Organization [1], approximately 350-500 million cases of malaria infections occur in Sub-Saharan Africa, with 110 million cases of illness and 2 million deaths, of which 25% are childhood deaths [2,3]. It is a disease that can be cured in just forty-eight (48) hours, yet it can cause fatal complications if the diagnosis and treatment are delayed [4]. In the human body, the parasites multiply in the liver and then infect red blood cells [5].

The clinical manifestations of *Plasmodium falciparum* malaria infection are variable and encompass a wide range of pathophysiological derangements that affects multiple organ systems including the liver and kidneys [6,7]. Lipoproteins such as chylomicrons, very low-density lipoproteins (VLDL), low-density lipoproteins (LDL), high-density lipoproteins (HDL) and free fatty acids (FFA) are major lipid components in plasma. Most plasma apolipoproteins, endogenous lipids and lipoproteins have their origin from the liver [8,9, 10], which depends on cellular integrity and functionality of the hepatocytes. Under normal physiological conditions, the liver ensures homeostasis of lipid and lipoprotein metabolism [9], thus, hepatocellular damage often associated with severe and acute *P. falciparum* infections impairs these processes, leading to alterations in plasma lipid and lipoprotein patterns [11,12]. Malaria severity can be caused by the presence of life-threatening features in patients with *P. falciparum* peripheral parasitemia. Studies have shown that malaria infection induces changes in lipid profile, the magnitude of which is related to the severity of malaria [13].

It is well established that the balance of electrolytes in the body is essential for normal function of cells and organs. Electrolyte disturbances are known to be common in malaria. Also, severe cases of renal problems are usually associated with malaria, and without treatment, can lead to renal failure [14]. In Nigeria, there is an increased incidence of kidney problems, and because Nigeria is a malaria-endemic country, assessment of renal function in malaria cases is important to ensure effective management of patients. Severe anaemia, hypovolaemia and perturbations of electrolyte levels in African children with severe malaria have been reported [15,16]. Acidosis is also a common complication of severe malaria and has been identified as the single most important prognostic feature of the disease [17]. Hence, correction of electrolyte imbalance and lipid profile forms a major component of the treatment of severely infected malaria patients. This study, therefore, aims to determine the lipid profile and electrolyte levels of malaria-infected patients attending Muhammadu Abdullahi Wase Specialist Hospital, Kano State, Nigeria.

2. METHODS

2.1 Study Subjects

A total of four hundred (400) samples were allocated for the research work. Two hundred and forty (240) pathological blood samples were collected from malaria patients attending Muhammadu Abdullahi Wase Specialist Hospital, Kano State, Nigeria. One hundred and sixty (160) apparently healthy persons of the same age range with no evidence of malaria infection served as normal control. All participants were within the age of 20-70 years. Patients with malaria included in this study were those

diagnosed using the WHO criteria (1999), with parasites per microlitre of blood in a thick smear assuming a leukocyte count of 8,000/ μ litre. Those who consented were included in the study, individuals below the age of 20 and those who refused to give their consent were excluded. Ethical approval was obtained from the ethics committee of the Hospital Management Board, Kano state, with an ethical clearance number HMB/GEN/488/VOL.I.

2.2 Sample Collection and Preparation

Venous blood (5 cm³) was aseptically collected from the subjects and parasitemia level determined as described by Cheesebrough (1998). Patients were considered having severe *P. falciparum* malaria if they met the predefined, modified WHO criteria for severe malaria (hyperparasitaemia >10% parasitaemia).

The malaria parasite density was graded as follows:

- 1 parasite/field: low density (+)
- 2-9 parasites/field: medium density (++)
- More than 20 parasites/field: high density (+++).

2.3 Biochemical Analysis

Sera was obtained from all samples and stored at 4°C until required for analysis, which was done within 24 h. The serum concentrations of total cholesterol (TC), low-density lipoprotein

(LDL), high-density lipoprotein (HDL) and triacylglycerol (TAG) were determined using RANDOX reagent kits according to the manufacturer's instructions. Serum Na⁺, Cl⁻ and K⁺ were determined using flame emission spectrophotometric method [18]

2.4 Statistical Analysis

All results are presented as Mean \pm SD. The data were evaluated by one-way ANOVA, and the differences between the means were assessed using Duncan's test. Statistical significance was considered at $P < 0.05$.

3. RESULTS

Table 1 shows the distribution of male and female malaria-infected and non-infected patients in the study population. Results show that 64.17% (154) of the male subjects and 35.83% (86) females were infected with malaria while out of the 160 control patients, 70% (112) were male and 30% (48) females.

All the malaria and non malaria-infected patients that participated in this study were between the ages of 40-70 years. Results from Table 2 shows that 70 (29.3%) of the malaria patients were between 40-44 years, 66 (27.5%) were between 45-49 years, 50 (20.8%) were 50-54 years, 20 (8.3%) were 55-59 years and 14 (5.8%) were 65 years and above.

Table 1. Distribution of malaria positive and negative patients based on gender

Gender	Malaria-positive		Control	
	Frequency	Percentage	Frequency	Percentage
Male	154	64.17%	112	70%
Female	86	35.83%	48	30%
Total	240	100%	160	100%

Table 2. Age group distribution of malaria and non-malaria patients

Age(years)	Malaria-infected		Control	
	Frequency	Percentage	Frequency	Percentage
40-44	70	29.3%	40	25.0%
45-49	66	27.5%	44	27.5%
50-54	50	20.8%	20	12.5%
55-59	20	8.3%	30	18.7%
60-64	20	8.3%	14	8.8%
65-70	14	5.8%	12	7.5%
Total	240	100.00%	160	100.00%

Result of the serum lipid profile of the malaria and non-malaria-infected patients attending Muhammadu Abdullahi Wase Specialist Hospital, Kano State, Nigeria is presented on Table 3. From the results, the TC levels in males and females with malaria were significantly ($P<0.05$) higher than their control counterparts. The HDL-C levels decreased significantly ($P<0.05$) by 50 and 58% in males and females, respectively, after malaria infection compared to non-malaria control. As for TAG, the mean value was elevated significantly ($P<0.05$) by 52% and 35% in male and female malaria patients, respectively, relative to control. The LDL-C level in males with malaria, though higher than control, was not significantly ($P>0.05$) different. However, the difference was significant when serum LDL-C levels in females with malaria were compared to those without malaria. The VLDL-C level increased by 51% in male patients, and 48% in female malaria patients compared to control.

Based on severity of malaria infection, the TC, LDL-C, TAG and VLDL-C levels of patients with severe malaria were significantly ($P<0.05$) higher

than those with non-severe malaria, while HDL-C decreased significantly (Table 4).

Results of the electrolyte levels shows that the serum Na^+ level of females with malaria was significantly ($P<0.001$) lower than non-malaria patients. There were no significant ($P>0.05$; $P>0.001$) differences in levels of Na^+ and Cl^- in males with malaria and those in control group. However, the levels of serum K^+ decreased significantly ($P<0.05$; $P<0.001$) in males and females with malaria than their control counterparts. Also, the K^+ levels in females with malaria were significantly ($P<0.05$) higher than their male counterparts (Table 5).

Table 6 shows serum levels of Na^+ , K^+ and Cl^- based on severity of malaria in the patients. Of the 240 patients tested, 190 had mild malaria, and 50 had severe malaria. More than 50% of the patients with severe malaria had lower Na^+ (26 patients) and K^+ (30 patients). Although only 18% (34 patients) of those with mild malaria had lower Na^+ levels, they were all within normal range. A higher number of patients within all three groups had Cl^- levels between 98-106 mEq/L.

Table 3. Serum lipid profile in malaria positive and negative patients

Parameters (mmol/L)	Malaria patients		Non-malaria patients	
	Male	Female	Male	Female
TC	6.38 ± 0.10 ^a	7.97 ± 1.60 ^b	5.75 ± 0.30 ^d	5.59 ± 0.30 ^c
HDL-C	0.73 ± 0.20 ^d	0.62 ± 0.30 ^d	1.45 ± 0.40 ^f	1.06 ± 0.20 ^e
TAG	2.80 ± 0.50 ^g	2.87 ± 0.70 ^g	1.84 ± 0.40 ⁱ	2.13 ± 0.50 ^j
LDL-C	4.23 ± 1.40 ^k	5.49 ± 1.40 ^m	3.80 ± 0.70 ^k	3.55 ± 0.50 ^l
VLDL-C	1.27 ± 0.20 ⁿ	1.44 ± 0.50 ^r	0.84 ± 0.20 ^p	0.97 ± 0.20 ^p

Values are Mean ± SD

Values with different superscript letters within the same row are significantly ($P < 0.05$) different
 TC = Total Cholesterol; HDL-C = high density lipoprotein cholesterol; LDL-C = Low density lipoprotein cholesterol; TAG = Triacylglycerol; VLDL-C = Very low-density lipoprotein cholesterol

Table 4. Mean values of lipid fractions based on severity of malaria in patients

Severity	Parameters				
	TC	HDL-C	LDL-C	TAG	VLDL-C
Mild	5.3±0.7 ^b	1.2±0.3 ^d	3.1±0.7 ^f	2.3±0.2 ^h	1.1±0.1 ^l
Severe	8.5±1.4 ^a	0.6±0.1 ^c	6.5±0.4 ^e	3.5±0.5 ^g	1.7±0.4 ⁱ

Values are Mean ± SD

Values with different superscript letters within the same row are significantly ($P < 0.05$) different

Table 5. Serum electrolyte levels in malaria positive and negative patients

Parameters (mEq/L)	Malaria patients		Non- malaria patients	
	Males	Females	Males	Females
Sodium	105.78 ± 25.86	98.69 ± 7.28 ^a	102.12 ± 27.54	108.82 ± 23.89 ^a
Chloride	177.31 ± 63.42	158.35 ± 14.96	157.99 ± 21.09	163.85 ± 17.22
Potassium	4.33 ± 3.36 ^{a, b}	5.49 ± 2.74 ^{a, b, c, d}	5.95 ± 2.54 ^{a, b}	5.86 ± 1.88 ^{c, d}

Values are expressed as Mean ± S.D

Values with the same superscript within the same row are statistically significant at $P<0.05$ and $P<0.001$

Table 6. Serum Na⁺, K⁺ and Cl⁻ levels based on Parasitemia of malaria patients

Electrolyte level(mEq/L)	Mild	Severe	
Sodium	< 35	0	26
	35-100	34	6
	100- 155	114	4
	>155	42	14
Potassium	< 3.4	0	30
	3.4- 4.0	40	4
	4.0- 5.3	88	10
	> 5.3	62	6
Chloride	< 98.0	0	10
	98 – 100	98	8
	100- 106	58	18
	>106	34	14

4. DISCUSSION

This study presents a high number of the malaria cases (186) within the age group of 40-54 years constituting 77.60% of the total infected population. However, there was a sharp decrease in prevalence above 55 years. This is far more than 30.59% reported by Gobir and Tukur [19] from Kano metropolis. This may be due to climatic condition at the time of study, geographical location, seasonal variation, and socio-economic pattern of the population [20]. Male population was observed to constitute 64.17% (154) prevalence of the population under study while females were less affected with a prevalence of 35.83% (86), signifying an increase in malaria prevalence as against reports by WHO [21]. This is also not consistent with the findings by Gobir and Tukur [19] who reported a prevalence rate of 61% in females and 38% for males. However, the present study conforms to studies by Akanbi et al. [22] who reported that males had a higher prevalence of malaria than their female counterparts. This may be attributed to the dress norms and vulnerability of men, especially the youth, to malaria, as studies have shown that men are more likely than women to expose their bodies, thereby increasing their chances of being bitten by mosquitoes, while females are mostly indoors, reducing their exposure to the malaria vector (22). Also, men may be at occupational risk of contracting malaria, especially when their work occurs during peak biting times around dusk [23].

From the result, the TC, LDL, TAG and VLDL increased, while the HDL decreased in malaria-infected patients compared to non-malaria group

(control). Also, the level of HDL decreased and TC, LDL, TAG and VLDL increased with increase in severity of malaria. This result agrees with studies [13,24,17,25] demonstrating that malaria infection usually perturbs plasma lipoprotein metabolism causing alterations in lipid and lipoprotein profiles. Although it is established that acute infection and inflammation produce moderate changes in plasma lipoprotein resulting in increased serum TAG concentration and decreased HDL due to increased VLDL production and mobilization of free fatty acids from adipose tissue, thereby increasing de novo hepatic fatty acid synthesis and suppression of fatty acid oxidation [17], the mechanism involved in the changes during malaria infection is not well known, but believed to be partly host-related [26], as it was initially speculated that increase in serum lipid was due to the lipid content of the parasite [27]. More recently, alterations in lipid profile of malaria patients have been attributed to the level of hemolysis in malaria, which is proportional to severity of infection. This is because the erythrocyte membranes are predominantly lipid and liberation of membrane lipids following sustained hemolysis accounts for alterations seen in infected patients [28].

The serum Na⁺ and K⁺ levels of patients with malaria were significantly ($P < 0.001$) lower, while Cl⁻ levels were not significantly ($P > 0.001$, $P > 0.005$) different than the control group (Table 5). This result agrees with several reports demonstrating a progressive decrease in Na⁺ and K⁺ within 12 h of the parasite's invasion during infection [4]. In another study, Kakkilaya [29] reported mild hyponatraemia in malaria patients. These demonstrate the complicated and conflicting relationship between malaria infection and renal function. Based on the severity of malaria (Table 6), the result from this study shows that hyponatremia and hypokalemia are common in malaria patients and particularly associated with the severe condition. It has been reported that urinary potassium waste and hypokalemia, as well as potassium depletion are common complications of severe malaria and makes the correction of acidosis pertinent. The reduction in K⁺ levels has been attributed to the loss of about 75 to 80 % of the normal potassium content of host cells during the course of malaria infection. However, the pathophysiology of hyponatraemia in malaria remains unclear, but several studies have suggested increased secretion of vasopressin plays an important role. Other studies have

shown that hyponatraemia is not an exclusive reflection of malaria, but also reflects the severity of other diseases [30,31].

5. CONCLUSION

In conclusion, this study has demonstrated that mild and severe *P. falciparum* infection causes profound changes in lipid profile and disturbances in Na⁺ and K⁺ levels, with a hyponatremia and hypokalaemia evident in severe malaria. Thus, it is pertinent to estimate serum electrolytes in malaria patients in order to manage electrolyte derangements in the overall management of malaria infections.

CONSENT

As per international standard or university standard, patient's written consent was collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard, written approval of Ethics committee was collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. World Malaria Report. World Health Organization. Geneva. 2012;xxxiv.
2. Adekunle AS, Adekunle OC, Egbewale BE. Serum status of selected biochemical parameters in malaria: An animal model. Biomedical Research. 2007;18(2):109-113.
3. Onyesom I, Ekeanyanwu RC, Achuka N. Correlation between moderate *Plasmodium* and antioxidant vitamins in serum of infected children in South Eastern Nigeria. *Falciparum* Malarial Parasitaemia. African Journal of Biochemical Research. 2010;4(12):261-264.
4. Dworak JA, Miller LH, Whitehouse WC, Shiroshi T. Invasion of erythrocytes by malaria parasite. Science. 1975;187:748-750.
5. World Health Organization. Guidelines for the treatment of malaria, 3rd ed; 2015. Geneva. Available:<http://www.who.int/malaria/publications/atoz/9789241549127/en/>
6. Ehrich JHH, Horstmann RD. Origin of proteinuria in human malaria. Tropical Medical Parasitology. 1985;36:39-45
7. Maitland K, Marsh K. Pathophysiology of severe malaria. Acta Tropica. 2004; 90:131-140.
8. Tietge UJ, Boker KH, Bahr MJ, Weinberg S, Pichlmayr R, Schmidt HH, et al. Lipid parameters predicting liver function in patients with cirrhosis and after liver transplantation. Hepato-gastroenterology. 1998;45(24):2255-2260.
9. Jiang J, Nilsson-Ehle P, Xu N. Influence of liver cancer on lipid and lipoprotein metabolism. Lipids in Health and Disease. 2006;5:4-10.
10. Mayes PA, Botham KM. Lipid transport and storage. In: Murray R.K., Granner DK, Mayes PA, Rodwell VW. Editors. Harper's Illustrated Biochemistry. 26th ed. California: Lange Medical Books/McGraw-Hill; 2003.
11. Faucher JF, Ngou-Milama E, Missinou, MA, Ngomo R, Kombila M, Kreamsner PG. The Impact of Malaria on Common Lipid parameters. Parasitology. 2002;88:1040-1043.
12. Sibmoo N, Yamanont P, Krudsood S, Leowattana W, Brittenham G, Looareesuwan S. et al. Increased fluidity and oxidation of malarial lipoproteins: Relation with severity and induction of endothelial expression of adhesion molecules. Lipid Health Distribution. 2004; 3:15-10.
13. Visser BJ, Rosanne WW, Ingeborg MN, Martin PG. Serum lipids and lipoproteins in malaria -a systematic review and meta-analysis. Malaria Journal. 2013;12:442
14. Uzuegbu UE, Emeka CB. 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.
15. Dekker E, Hellerstein MK, Romijn JA, Neese RA, Peshu N, Endert E, et al. Glucose homeostasis in children with falciparum malaria: Precursor supply limits gluconeogenesis and glucose. Journal of Clinical Endocrinology and Metabolism. 1997;82:2514-2521.
16. Maitland K, Pamba A, Fegan G, Njuguna P, Nadel S, Lowe B, et al. Perturbations in severe malaria infection. Paediatric Care Medicine. 2005;5:226-235.
17. Krishna AP, Chandrika SK., Manasa A. Shrikant LP. Variation in common lipid

- parameters in malaria infected patients. Indian Journal of Physiology and Pharmacology. 2009;53(3):271-274.
18. Tietz N, Pruden LE, Andersen S. Electrolytes. In: Tietz Fundamentals of clinical chemistry. 2nd ed. WB. Saunders Co., Philadelphia, USA. 1996;721-738. ISBN: 0-7216-3763-9.
 19. Gobir Z, Tukur Z. Prevalence of malaria parasitemia using rapid diagnostic test among apparently healthy children in Kano, Nigeria. Journal of Medicine in the Tropics. 2014;16:1-4.
 20. World malaria report. World Health Organization; 2009.
 21. The African malaria report. World Health Organization. Geneva: WHO/ UNICEF; 2014.
 22. Akanbi OM, Badaki JA, Adeniran OY, Olotu OO. Effect of blood group and demographic characteristics on malaria infection, oxidative stress and haemoglobin levels in south western Nigeria. African Journal of Microbiology Research. 2010;4: 877-880.
 23. Gender, health and malaria. World Health Organization. Geneva; 2007.
Available:https://www.k4health.org/sites/default/files/gm_guide-en%5B1%5D.pdf
 24. Nwobodo N, Okonkwo PO, Nwobodo E, Igwe SA. Evaluation of the effects of malaria infection on serum lipid profile of patients attending two district hospitals in Enugu, Nigeria. Oriental Journal of Chemistry. 2008;24:415-418.
 25. Akanbi OM. Effect of malaria infection on oxidative stress and lipid profile in pregnant women. Journal of Medicine and Medical Sciences. 2013;4(3):128-133.
 26. Resonson RS. Myocardial Injury. The acute phase response and the lipoprotein metabolism. Journal of American College of Cardiology. 1993;22:9333-940.
 27. Beach DH, Sherman IW, Holz GG. Lipids of *P. lophurae*, and of erythrocytes and plasmas of normal and *P. lophurae* infected Pekin ducklings. Journal of Parasitology. 1977;63(1):62-75.
 28. Chikezie PC, Okpara RT. Serum lipid profile and hepatic dysfunction in moderate *P. falciparum* infection. Journal of Public Health Epidemiology. 2013;5(9):379-384.
 29. Kakkilaya BS. Rapid diagnosis of malaria. Journal of Laboratory Medicine. 2003;34: 602-608.
 30. Beukhof CM, Hoorn EJ, Lindemans J, Zietse R. Novel risk factors for hospital-acquired hyponatraemia: A matched case-control study. Clinical Endocrinology (Oxf). 2007;66:367-372.
 31. Waikar SS, Mount DB, Curhan GC. Mortality after hospitalization with mild, moderate, and severe hyponatremia. American Journal of Medicine. 2009; 122:857-865.

© 2018 Kiru et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:

The peer review history for this paper can be accessed here:
<http://www.sciencedomain.org/review-history/24600>