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LIPID PROFILE IN PLASMODIUM FALCIPARUM INFECTED SUBJECTS IN EKPOMA, EDO STATE, NIGERIA

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Keywords:
Malaria, lipid profile, Plasmodium falciparum.

Abstract

Background: Malaria remains a major health problem worldwide with 35% of the human population being at risk of becoming infected.

Methods: In this study lipid profile (HDL, LDL, total cholesterol and triglycerides) were measured in 90 subjects with *P. falciparum* infection and 30 apparently healthy individuals as controls. Laboratory diagnosis of subjects was confirmed by Giemsa staining method for thick blood film and plasma lipid levels were assayed by colorimetric methods.

Results: Triglyceride levels were significantly lower in the test subjects (0.73±0.32mmol/l) with *P. falciparum* than in the control subjects (1.33±0.52mmol/l). In contrast, the levels of LDL were significantly higher in test subjects (2.01±0.89mmol/l) with *P. falciparum* infection when compared with the control subjects (1.73±1.09mmol/l). Total cholesterol and HDL-cholesterol showed no statistically significant difference when test subjects were compared with the control.

Conclusion: It was concluded from this study that *P. falciparum* infection induces significant changes in triglyceride and LDL-cholesterol level thereby supporting the hypothesis that *P. falciparum* infection affects lipid profile.

Introduction

Malaria remains a major health problem worldwide and it is caused through the bite of an infected female *Anopheles* mosquito [1]. Moreover, the vast majority of morbidity and mortality from malaria is caused by infection with *P. falciparum*, although *P. vivax*, *P. ovale*, and *P. malariae* also are responsible for human infections [2]. Interestingly, malaria causing parasites has been observed to have a tremendous requirement for lipids during replicative stage of their development. Vial *et al [3]*, reported that plasmodium malarial parasites showed proficiency at scavenging and modifying of lipids in mammalian host. Furthermore, Studies have also shown that the parasites could obtain free fatty acids (FFAs) directly from the serum or from sources such as high-density lipoprotein (HDL) [4, 5, 6]. Systematically, scavenged phospholipids within the parasite can be incorporated without modification [6, 7]. However, plasmodium parasites have the capacity to modify fatty acids as needed for elongation or desaturation and incorporate them into phospholipids, diacylglycerols (DAGs), and triacylglycerols (TAGs) [8, 9] with also a concomitant increase in the oxidative stress [10]. Since most scavenged and synthesized lipids are likely incorporated into the membranes of the rapidly growing parasite, monitoring of plasma and liver lipid concentrations during malarial infection could be of immense contribution in establishing the parasite source of lipids for its propagation [8]. Hence, this study is designed to evaluate the lipid profile of malaria infected subjects.

Materials and methods

Study area: This study was carried out in Ekpoma, Esan-West Local Government Area of Edo state, Nigeria. Ekpoma is fairly an urban area with a University situated in it. It is located at latitude 6.75^0N and longitude 6.13^0e with population of 61,870 [11]. The inhabitants are mainly students, civil servants and farmers.

Study population: The study population consisted of 120 subjects recruited from Eromosele Hospital, Somos medical laboratory and AAU College of Medicine. Test subjects comprised of 90 malaria positive subjects of ages 18 to 65 years and 30 apparently healthy subjects of ages 18-60 years serving as control.

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Inclusion criteria: In this study only confirmed malaria (positive) infected subjects were recruited.

Exclusion criteria: The exclusion criteria for this study includes: non confirmed malaria (negative) infected subjects, pregnancy, diabetes mellitus, hypertension, obesity, smoking, alcoholism, HIV infection and non-fasting subjects.

Sample collection: Five mililitres of fasting venous blood were obtained from the subjects using sterile disposable syringes and needle at the anti-cubital fossa vein by veni-puncture after sterilization with 70% alcohol with the use of tourniquet. The blood was dispensed into lithium heparin container and mixed gently. Thick blood films were made for malaria test and the blood samples were centrifuged at 4000rpm for 5minutes. The plasma obtained were placed into a clean dry plain container and stored frozen prior to analysis.

Sample analysis: Malaria parasite determination was carried out by examining a thick blood film stained by Giemsa method as descriobed by Cheesbrough [12]. Total cholesterol, Triglycerides, High Density Lipoprotein – Cholesterol and low density lipoprotein-cholesterol were estimated colorimetrically as described by Richmond [13], Trinder [14], Lopes – Virella *et al*., [15] and Friedewald *et al*., [16] respectively.

Statistical analysis: The obtained data were then analyzed using the Statistical Package for Social Sciences (SPSS,version 20) and results were described using mean and standard deviation. Where applicable, the student "t – test" and ANOVA were used to compare the differences of total cholesterol, triglyceride, high density lipoprotein and low density lipoprotein between groups at significant level of p-value of <0.05 with a confidence level of 95%.

Results

Table 1: Shows the total cholesterol and triglyceride levels of control and malaria positive subjects. The result showed that total cholesterol levels of the test group was not statistically significant different (P>0.05) when compared with the control but a statistically significant increase (P<0.05) was observed when triglyceride levels of the test group was compared with the control.

Table 1: Shows the total cholesterol and triglyceride levels of control and malaria positive subjects.

Parameters (mmol/l)	Control group (Mean±SD) n=30	MP positive (Mean±SD) n=90	T-value	P-value
Total cholesterol	3.90±1.10	3.91±1.09	0 .110	P>0.05
Triglyceride	1.33±0.52	0.73±0.32	-17.943	P<0.05

Significant at *P<0.05.

Table 2: Shows the HDL- cholesterol and LDL-cholesterol levels of control and malaria positive subjects. The result showed that HDL- cholesterol levels of the test group was not significantly different (P>0.05) when compared with the control but statistically significant increase (P<0.05) was also observed when LDL-cholesterol levels of the test group was compared with the control.

Table 2: Shows the HDL- cholesterol and LDL- cholesterol level of control and malaria positive subjects.

Parameters (mmol/l)	Control group (Mean±SD) n=30	MP positive (Mean±SD) n=90	T-value	P-value
HDL-cholesterol	1.66±0.74	1.60±0.56	-0.977	P>0.05
LDL-cholesterol	1.73±1.09	2.01±0.89	3.043	P<0.05

Significant at *P<0.05.

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Discussion

Malaria parasite has been observed to have a tremendous requirement for lipids during the replicative stages that take place in the mammalian host and biochemical studies on blood-stage Plasmodium malarial parasites have demonstrated the parasite's proficiency at scavenging and modifying lipids obtained from the host [3]. Interestingly, triglyceride showed significant decrease when malaria infected test subjects were compared with the control and this agrees with the work of Mohanty *et al.* [17], who noted that serum levels of triglyceride (TG) were lower in patients than in the control group. This could be due to lipid oxidation [10], and they are consistent with the fact that host response to acute infection increases lipoprotein oxidation *in vivo [18]*. In contrast, Faucher *et al* [19], reported that in low-level malarial infection, the levels of total cholesterol, LDL and HDL are reduced while triglyceride levels are increased. As for total cholesterol, the result of this study showed no significant different which are in contrast with the report of Faucher *et al*[19] and Chikezie and Okpara [20].

Lipoproteins are major lipid component in plasma, and certainly targets for oxidative stress [10]. During malaria infection, the levels of LDL and HDL are decreased according to Nilsson-Ehle and Nilsson-Ehle [21], and Mohanty *et al* [17], but this study showed significant increase in the levels of LDL and no significant difference for HDL. It is important to remember that vascular endothelial cells play a pivotal role in pathogenesis of malaria infection, especially cytoadherence of the infected erythrocytes on endothelial cells. The expression of adhesion molecules on endothelial cells is partly regulated by reactive oxygen species and oxidized LDL [22] which could affect the serum LDL level.

Although, previous studies revealed decreased level of LDL which is contrary to the findings of this study, it showed sparing effect of acute phase oxidative effect of malarial infection on LDL and this could lead to LDL plague due to its involvement in endothelial cells adhesion during malarial infection. Conclusively, this study shows that lipid profiles of malarial patients are altered due to lipid oxidation and also supports the idea that this alteration can be explored for novel malaria therapy.

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