

Original Research Article

Assessment of Serum Lipid Profile among Sudanese Patients with MalariaIbrahim Yousif Abdalla Hamid¹, Amina Osman. M. Elzein², Abdelgaidr Eltom³¹M.Sc.–clinical chemistry- Al-Zaiem Al-Azhari University, Sudan²Department of clinical chemistry, faculty of Medical laboratory Sciences, Al zaiem Al Azhari University-Sudan³Medical Laboratories department, college of allied health sciences, Gulf Medical University, Ajman, UAE***Corresponding author**

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Abstract: Changes in lipid profile are seen in many patients infected with malaria parasite. The malaria parasite causes hepatocellular damage and disturbs lipid handling by the liver. Inside hepatocytes and erythrocytes the parasite replicates rapidly scavenging cholesterol and lipids required for its growth and metabolism from the host. It also requires host lipids for detoxification of free heme to form the malarial pigment, haemozoin. Aim to Assess Serum lipid profile in patients with malaria in Khartoum state. Material and methods the present study was analytical cross-sectional study carried out during Sep 2016 to Dec 2016, at Khartoum State at assagai rural hospital. Total of one hundred individuals were enrolled in this study, and classified into two groups 50 patients with malaria as case group and 50 healthy individuals as control group. Result as compared there is difference in HDL, LDL, cholesterol and triglycerides between malaria patients and control subjects ($p < 0.05$). Patients with malaria showed low HDL (20.00 ± 3.76 mg/dL versus 44.52 ± 4.85 mg/dL), low LdL (59.00 ± 16.15 mg/dL versus 107.76 ± 9.76 mg/dL), low cholesterol (114.78 ± 16.75 mg/dL versus 175.34 ± 16.83 mg/dL) and elevated triglycerides (172.4 ± 25.63 mg/dL versus 135.7 ± 11.66 mg/dL). The observations show a statistically significant. Conclusions malaria infection, both *P. vivax* and *P. falciparum* infections, causes derangements in lipid profile that are characterized by low serum, low HDL, low LDL and high triglyceride levels but differ in total cholesterol level. These derangements may be of diagnostic, prognostic or therapeutic value.

Keywords: lipid profile, malaria.

INTRODUCTION:

Malaria is a mosquito borne infectious disease caused by a protozoan parasite belonging to the genus *Plasmodium*. It is widely endemic in tropical and subtropical parts of the world where it continues to cause significant morbidity and mortality [1]. Malaria is recognized as a serious public health problem worldwide, affecting nearly 50% of the population in more than 109 countries. Its estimated 300 million new cases and 1 million deaths per year, mostly in children under 5 years and pregnant women [2]. The vast majority of morbidity and mortality from malaria is caused by infection with *P. falciparum*, although *P. vivax*, *ovale*, and *P. malaria* also are responsible for human infection [3]. Patients with malarial infection show a wide range of metabolic derangements including changes in serum lipid profile [1]. The exact mechanisms resulting in these derangements in serum lipid profile in patients infected with the malaria parasite is still poorly understood. The several probable biological mechanisms that can explain these changes involve a complex interplay between the parasite and the host [4].

Malaria parasite-induced hepatocellular damage leads to abnormal lipid handling by the liver and hence, unable to maintain homeostasis of lipid and lipoprotein metabolism. This causes derangements in plasma lipid profile [1].

Patients with malaria often exhibit laboratory abnormalities due to an acute phase response, but little is known about serum lipid profile changes in malaria. In 1978, Lambrecht *et al.*; [5] reported transient lipid profile changes in six returning travellers with malaria caused by *Plasmodium vivax* and suggested for the first time that changes in high-density lipoprotein (HDL) and very low-density lipoprotein (VLDL) in human serum are related to the lipid metabolism of the parasite. It was hypothesized that the malaria parasite uses cholesterol and phospholipids from its host, resulting in a decrease of serum HDL. Prior to this report, Angus *et al.*; [6, 7] utilized lipoprotein electrophoresis in rhesus monkeys infected with *Plasmodium knowlesi* to study serum lipids in malaria. Their results were not conclusive because lipoprotein bands could barely be

detected in the serum of controls. Subsequently, several clinical studies showed lipid profile changes in the setting of both uncomplicated and complicated malaria [7- 13]. Although the magnitude of changes seems to be related to the severity of malaria in several studies [14, 15], others found no correlation between the severity of malaria attacks and the extent of lipid profile changes [16, 17]. These transient lipid profile changes in the parasitaemic phase have been suggested by some researchers as a potential adjuvant diagnostic tool for malaria [18-20].

Changes in serum lipid profile and lipid metabolism are due to a whole range of at least partially disease-specific mechanisms [21]. The extent of serum lipid profile changes during malaria infection and their underlying biological mechanisms remain unclear. Mechanisms may be partly host related (i.e., related to an acute phase reaction [22]), parasite-related [23- 25], or a combination of these two. If a link between human host serum lipid alterations and the pathogenesis of malaria can be demonstrated, further studies to elucidate the precise pathways can be conducted. Moreover, novel treatment approaches could be explored with lipid metabolism-regulating drugs. Therefore, it is hypothesized that the lipid profile of malaria exhibits characteristic changes. In addition, it is understood that these changes are specific for the malaria pathogen-host interplay.

MATERIAL AND METHODS:

Study Design:

Cross-sectional study.

Study area:

Khartoum state

Study population:

Patients with clinical malaria and healthy control

Sample size:

50 study group (case) – 50 healthy control group

Selection criteria:

A. Inclusion criteria:

Patient with clinical malaria

b. Exclusion criteria:

Hypertensive, diabetics, suffering from renal diseases, liver disease, obstructive jaundice, cancer, alcoholism and persons living with human immunodeficiency virus (HIV) infection

Study Duration:

This study was carried out during period from Sep to December 2016

Study area: Khartoum state in alssagai rural hospital.

Data collection

Data were collected using self-administrated preceded questionnaire which will specifically designed to obtained information that help in either including or excluding certain individuals from the study respectively.

Collection and Processing of Blood Specimen:

Five milliliters (5 ml) of venous blood was obtained from the anterior cubital vein by sterile venipuncture procedure using 5ml disposable, sterile syringe. The sample was obtained after a 12 hour fasting period for all controls. For all malaria cases, sample was taken on day 2 after a 12 hour fasting period. The blood was collected in di-potassium ethylene-diamine-tetra acetic acid (K2EDTA) tubes and stored at 5oC until required for analyses.

Microscopic Detection of Malaria Parasite

Thick blood films were made from EDTA blood sample and stained with Giemsa's staining technique. The slides were observed under microscopy using $\times 100$ objectives lenses under oil immersion

Biochemical Analysis of Prepared Serum

Serum triglycerides, total cholesterol and HDL cholesterol are analyzed by standard enzymatic procedures biosystem kits using spectrophotometer and LDL cholesterol by Friedewald's equation.

Ethical consideration:

Permission to participate in this study will take from each participant orally. Permission of this study will obtain from the area of the study.

STATISTICAL ANALYSIS:

Descriptive analysis was done and for continuous data, t-Test was used to compare experimented and control groups. A p- value of < 0.05 was considered significant. The IBM SPSS 21 (Statistical Package for Social Sciences) was used for statistical analysis.

RESULTS

The study population included 100 (36 male and 64 female). The volunteers their ages ranged between (18- 67) years (mean \pm SD:27.8 \pm 9) years old. 50 of the population are infected with malaria which considered as cases and 50 apparently healthy individuals as control.

The statistical analysis showed that there was significant difference in most of the biochemical parameters of lipid profile between the studied cases

and control group including TG , HDL and LDL P value 0.000 , 0.018 , 0.002 respectively , while there is insignificant difference in TC p-value 0.60 as shown in table-1.

The patients infected with malaria are stratified into 2groups: infected with *P. vivax* (N = 15) and infected with *P. Falciparum* (N = 35). The study also revealed that there was no statistically difference among both group concerning the biochemical parameters of

the lipid profile P value (0.907, 0.098, 0.289 and 0.891). As shown in table-2.

In addition to that the patients grouped to severe (N= 19) and mild infected with malaria (N=31) ,surprisingly TC was significantly different between the two groups P –value 0.011 conversely there was no significant difference to TG , HDL and LDL (P value 0.723 , 0.747 and 0.134) respectively shown in table-3.

Table-1: Show compression between lipid profile among the studied malaria patients and control group:

Parameters	Control n = 50	Cases n = 50	P-value
Cholesterol mg/dl	175.34 ± 16.83	114.78 ± 16.75	0.60
Triglyceride mg/dl	135.7 ± 11.66	172.4 ± 25.63	0.000
HDL mg/dl	44.52 ± 4.85	20.00 ±3.76	0.018
LDL mg/dl	107.76 ± 9.76	59.00 ±16.15	0.002

Independent t test was used to calculate P value
P value less than 0.05 considered significant

Table-2: Show compression between lipid profile among the studied patients with *P. vivax* infection and *Falciparum*

Parameters	<i>P. vivax</i> n = 15	<i>P.falciparum</i> n = 35	P-value
Cholesterol mg/dl	118.13 ± 16.66	113.34 ± 16.34	0.907
Triglyceride mg/dl	163.33 ± 33.41	176.28 ± 20.87	0.098
HDL mg/dl	20.53 ± 4.08	19.82 ± 3.65	0.289
LDL mg/dl	64.53 ± 16.25	56.45 ±15.65	0.891

Independent t test was used to calculate P value
P value less than 0.05 considered significant

Table-3: Show compression between lipid profile among the studied severe and mild group:

Parameters	Severe n = 19	Mild n = 31	P-value
Cholesterol mg/dl	101.36 ± 9.21	123.00 ± 14.35	0.011
Triglyceride mg/dl	166.57 ± 26.61	175.96 ± 24.77	0.723
HDL mg/dl	19.10 ± 3.26	20.61 ± 3.98	0.747
LDL mg/dl	49.05 ± 10.70	65.09 ±16.00	0.134

Independent t test was used to calculate P value
P value less than 0.05 considered significant

DISCUSSION

The results of this study are in agreement with the reports of several other workers who studied the impact of malaria infection on serum lipid profile .Data show characteristic pattern of derangements that include lower levels of HDL, LDL, and higher levels of triglycerides in malaria patients as compared with those for healthy subjects (controls) these changes were all statistically different P value less than 0.05. one of these studies done by Warjri *et al.*; in 2016 [1] but at the same time we disagree with them concerning the Total cholesterol level which show no statistically difference P value more than 0.05 . Moreover our study revealed that there were no statistically difference between the

patients affected with *p.vivax* or *p. Falciparum* and this also agree with Warjri *et al.*;

Since only controlled studies were included in the quantitative synthesis, other non-infectious diseases and genetic factors are probably comparable among groups. This supports lipid profile changes as malaria-characteristic features; however, it does not exclude confounding. To investigate the main confounder, namely other infectious diseases, studies that compare lipid profile changes during malaria with control patients that present with other infectious diseases are pivotal. The meta- that included comparisons between malaria patients and symptomatic controls suggests that the observed lipid profile changes are indeed specific

for malaria. TC, HDL and LDL concentrations were lower in malaria and other febrile diseases compared to healthy controls, however, the decline was more pronounced and statistically significant during malaria. If these lipid profile changes are characteristic for malaria, one could expect more pronounced lipid alterations in severe malaria compared to uncomplicated malaria; this is confirmed by three studies. Biological mechanisms of lipid profile changes may be partly host-related, i.e., related to an acute phase reaction [26] or parasite-related [27] or a combination of these two.

The results of this present study strengthen the argument that the pattern of derangement of lipid profile seen in malaria is characteristic and specific for the disease. In this study showed that there is no difference between patient with mild and severe malaria, surprisingly TC was the only parameter significantly different between the two groups conversely to others. In which disagree with Mohanty *et al.*; Since only controlled studies were included in the quantitative synthesis, other non-infectious diseases and genetic factors are probably comparable among groups. This supports lipid profile changes as malaria-characteristic features; however, it does not exclude confounding. To investigate the main confounder, namely other infectious diseases, studies that compare lipid profile changes during malaria with control patients that present with other infectious diseases are pivotal. The meta- that included comparisons between malaria patients and symptomatic controls suggests that the observed lipid profile changes are indeed specific for malaria. TC, HDL and LDL concentrations were lower in malaria and other febrile diseases compared to healthy controls, however, the decline was more pronounced and statistically significant during malaria. If these lipid profile changes are characteristic for malaria, one could expect more pronounced lipid alterations in severe malaria compared to uncomplicated malaria; this is confirmed by three studies [28, 29].

The implications of these findings are manifold; the characteristic pattern of derangement in lipid profile in malaria may possibly serve as an aid to the clinical diagnosis of malaria especially in the absence of a positive blood film. In this regard, it is essential to understand that lipid derangements also occur in other infectious diseases. Hence, studies that compare lipid profile derangements between malaria and other infectious diseases are crucial to understanding the diagnostic potential of these lipid derangements in malaria. Labaied M *et al.*; [29] in their meta-analysis and review of studies that included comparisons between malaria patients and control subjects suffering from other febrile diseases suggested

that the observed changes in total cholesterol, HDL and LDL concentrations were more pronounced and statistically significant during malaria than in other febrile diseases. The study concluded that these changes were characteristic and specific for malaria.

CONCLUSION:

In conclusion, our study further strengthens the findings in other similar studies detailing the characteristic pattern of derangements in lipid profile in malaria patients. Malaria infection, both *P. vivax* and *P. falciparum* infections, causes derangements in lipid profile that are characterized by low serum, low HDL, low LDL and high triglyceride levels but differ in total cholesterol level. And also find that the severe case of malaria can affect cholesterol more than mild case

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