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ORIGINAL ARTICLE

HOSPITAL BASED STUDY OF LIPID PROFILE IN NEWLY DIAGNOSED CASES OF PLASMODIUM FALCIPARUM INFECTED MALARIA PATIENTS IN KOSI REGION OF BIHAR

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ABSTRACT: Malaria continues to be one of the major public health problem of India. Almost all deaths are caused by Plasmodium falciparum infected malaria.[1] Under normal physiological conditions, liver ensures homeostasis of lipid and lipoprotein metabolism.[2] Hepatocellular damage often associated with severe and acute P falciparum infections impairs these processes, leading to alteration in plasma lipid profile and lipoprotein patterns. An observation on the nature of dyslipidaemia in malaria patients with reference to correlation if any that exists between malaria and lipid profile in these patients was performed. Results revealed that the total cholesterol, HDL and LDL decreased while triglyceride and VLDL were found to increase in malaria patients. It can be concluded that alteration in lipid profile can be an index of malaria infection but specificity of malaria infection with lipid changes is questionable and need further studies.

KEYWORDS: Lipid profile, Malaria, Plasmodium falciparum.

INTRODUCTION: Malaria is a protozoan disease transmitted by the bite of infected Anopheles mosquitoes. The most important of the parasitic diseases of humans, it is transmitted in 108 countries containing 3 billion people and causes nearly 1 million deaths each year. Almost all deaths are caused by falciparum malaria.[3] India is a vast country with multiethnic society of 1.2 billion living in diverse geo-ecological paradigms and ecotype of malaria. Malaria continues to be one of the major public health problem of India with around 1.5-2 million confirmed cases/year, of which approximately 1000 reported malarial death per year.[4] As per World Health Organization report 2011-2012, South-east Asia region bear the second largest burden of malaria (13%), only being next to African region (81%). Among South-east Asia region, India shares two thirds of the burden (66%) followed by Myanmar (18%) and Indonesia (10%).[5] Contrary to the African scenario, where much of the malaria mortality burden is borne by the infants and children, in India, it is the middle productive ages in both genders that suffer the most.[6] Under normal physiological conditions, liver ensures homeostasis of lipid and lipoprotein metabolism.[7] Hepatocellular damage often associated with severe and acute P falciparum infections impairs these processes, leading to alteration in plasma lipid and lipoprotein patterns.[6–7] Parasites forage nutrients from their host as well as possess limited enzyme pathway for de novo synthesis of certain nutrients. So far, studies suggest that there may be some factors or enzymes, which allow the parasite to breakup and consume lipid/cholesterol from their host and utilize them for internalization of eukaryotic protozoa or for reproduction in case of helminthes.[8] Plasmodium is incapable of de novo synthesis of fatty acids and cholesterol; but it can fabricate its glycerides and phosphoglycerides with host-supplied nutrients, and can produce the glyceryl moiety during glycolysis.[9]

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In view of the incidence of malaria in state like Bihar Where large populations is migrant workers and indulge in construction activities in Cities, and farming, the present study was undertaken to find out any correlations between the changes in lipid profile of malaria patients.

MATERIAL AND METHODS: The study was carried out on clinically confirmed malaria positive patients attending the outpatient department of Katihar Medical College and Hospital, Katihar for a period of one year i.e. from January 2014 to December 2014 which was preapproved by the Ethical Committee of this institution review board. 41 participants were enrolled for the present study. 23 participants were malaria confirmed cases and 18 participants were without any clinical or laboratory evidence of malaria served as the control. The informed consent was obtained from the patients before enrolling them for study. Patients who are hypertensive, diabetics, suffering from nephrosis, liver disease or any condition suspected to affect serum lipids were excluded. Participants who presented with fasting less than 12 hours before collection of blood samples, also excluded from the study. The age range of participants was between 18 to 50 years.

Serum samples were collected and stored at 5^0C in the presence of EDTA anti-coagulant; and values were read at wavelength of 500nm. Thick blood film using anti-coagulated (EDTA) venous blood was employed in the detection of malarial parasite. Lipid profile of all participants was estimated by enzymatic methods[10,11] at day 0 (Pre-treatment). Other routine investigation was also done. Data obtained were statistically analyzed using Student t test, assuming $P<0.05$ as significant.

OBSERVATIONS: observations are presented in tabular form.

Lipid types	Control (n=18)	Malaria patients(n=23)	p-value
Total cholesterol ±SD	136.9444 ±14.87	116.9565 ±11.5423	<0.0001**
Total triglycerides ±SD	155.3333 ±21.5461	178.9565 ±38.6458	0.0255*
Total VLDL ±SD	31.1111 ±4.3506	36.2609 ±7.7648	0.0160*
Total LDL ±SD	68.9444 ±16.1335	55.913 ±12.9752	0.0066*
Total HDL ±SD	36.8889 ±4.8615	24.7826 ±6.7214	<0.0001**

Table 1: Mean value of serum lipid profile in malaria patients and control.

Results are presented as mean ± standard deviation. SD= Standard deviation, *=Significant value, ** =extremely significant value.

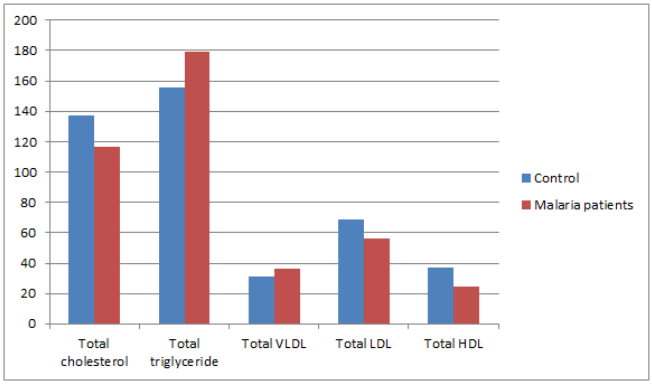

Fig. 1: Shows the effect of malarial parasites on the lipid profile in (Mean mg/dl).

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Table 1. & Figure 1. Shows that mean total cholesterol decreased from 136.9444±14.87mg/dl to 116.9565±11.5423 in malaria patients from controls (p=<0.0001), similarly LDL and HDL also decreased in malarial patients from 68.9444 & 36.8889 to 55.913 and 24.7826 respectively (p<0.05). But, there were significant increase of total triglyceride and VLDL noted in comparison to control in malaria infected patients, i.e. from 155.3333 & 31.1111 to 178.9565 & 36.2609 respectively, (p<0.05). The 'p' value shows difference in significance in different lipid parameter; the unequal significance may be due to small sample size.

DISCUSSION: This study included data on 23 confirmed malaria subjects and 18 controls. The study observed alteration in lipid profile of malaria positive subjects compare to the controls. There were increased triglyceride and VLDL in malaria confirmed subjects but LDL, HDL & Total cholesterol decreases.

It is shown in Table 1, that there is a statistically significant difference (p<0.0001) between malaria patients and control in the total cholesterol. Mohanty et al.[12] and others,[13,14] had earlier observe that serum level of total cholesterol is reduced in malarial infection, which support this finding.

Marked decline in HDL has been seen in malaria patients, which is similar to another study[6] of Faucher et al. as they reported that malaria infection produces moderate changes in plasma lipid profile in man, with typical decline in HDL concentration. It is worthwhile to note that Ogbodo et al[13] posited that oxidative modification of HDL and reduced serum levels of this class of lipoprotein was associated with the pathophysiology of malaria.

The observed mean value of triglyceride is higher in malaria patients which differ to Mohanty et al,[12] but it is similar to other studies with low level malaria infection[12] and malarial infection in children[13] parasitic protozoa infection[15] and in animal model.[16]

Sibmoh et al[7] noted that oxidized LDL from malarial patients increased the endothelial expression of adhesion molecules. In contrast to the present findings, in moderate malaria infection, serum levels of LDL and HDL were lower than in control subjects (Table-1). These observations suggest the critical role of oxidized lipoproteins, especially LDL on the pathogenesis of malaria. In addition, moderate malaria infection was associated with reduced serum level of VLDL and HDL (Table-1) that was in conformity with previous report of Mohanty et al.[12] From another report, the finding of the present study (Table-1) coincided with that of Faucher et al.[6]

Dyslipoprotinemia does occur in patients having active infections with most of the parasites e.g. Leishmaniasis, Toxoplasmosis, helminths. Membrane proteins are probably involved in such reactions.[8] Changes in lipid levels may not be unique to malaria infection. Another study, found striking elevation of total serum lipids in gram-negative bacilli infection while patients with severe gram positive cocci infection had normal serum lipids.[17] A possibility of a correlation between plasma cholesterol levels and the acute phase response during sepsis, burns, critical illness, etc has also been established.[18,19] The changes in the level of plasma lipoprotein may be attributed as a part of an acute phase reaction.[20–21] Hence a question raises regarding the specificity of lipid changes with malaria infection.

CONCLUSION: We have observed that a relation can be established between lipid profile and malaria infection, however, specificity regarding lipid changes with malaria infection is questionable. Further specificity should be assessed by studies including large sample size and/or case control method.

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