Section: General Medicine

Original Research Article

EVALUATION OF LIPID PROFILE IN MALARIA – A CROSS-SECTIONAL STUDY

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Abstract

Background: Malaria caused by Plasmodium parasites disrupts lipid metabolism, leading to transient dyslipidaemia, which often resolves with effective malaria treatment. This dyslipidaemia can increase susceptibility to cardiovascular diseases if left unaddressed. This study evaluated the lipid profile changes in patients diagnosed with malaria. **Materials and Methods:** This prospective cross-sectional study included 100 smear-positive malaria patients aged 13-40 years over nine months. Blood samples were collected in EDTA tubes, and smears were prepared for Leishman staining. Patients were categorised based on their malaria type as P. falciparum, P. vivax, or mixed infections. A 5 ml blood sample was taken for lipid profile estimation, measuring total cholesterol, triglycerides, HDL, and LDL levels. **Result:** Among 100 patients, Plasmodium falciparum was found in 11 patients, vivax in 34, and mixed infections in 55. Total cholesterol levels were desirable (<200 mg/dl) in all falciparum (100%) and vivax patients (100%), whereas 98.2% of the mixed group patients were within this range (p=1). Triglyceride levels were significantly different among the groups (p=0.043), with no desirable levels in the falciparum and vivax groups, while 5.5% of the mixed group had desirable levels. The HDL levels also showed significant differences (p=0.002), with the mixed group having the highest percentage of desirable levels (63.6%). LDL levels were below 60 mg/dl for most patients, particularly in the falciparum group (90.9%), with significant differences noted across the groups (p=0.029). **Conclusion:** The study indicates that malaria significantly affects lipid profiles, particularly by increasing triglycerides and lowering HDL and LDL levels. These changes are transient and likely resolved with effective malaria treatment rather than lipid-lowering interventions.

INTRODUCTION

Malaria is an infectious disease caused by the protozoan parasite of the genus Plasmodium, which infects humans through the bites of infected female Anopheles mosquitoes. There are five species of Plasmodium known to cause malaria in humans: Plasmodium falciparum, Plasmodium ovale, Plasmodium vivax, Plasmodium malariae, and Plasmodium knowlesi. The female Anopheles mosquito acts as the vector for malaria transmission.[1]

The life cycle of malaria begins when an infected female Anopheles mosquito bites a human, introducing sporozoites from the mosquito's salivary glands into the human bloodstream. Sporozoites travel from the site of inoculation to the liver, where they undergo a multiplication process called pre-erythrocytic schizogony. Within liver cells, sporozoites multiply to form merozoites, which are eventually released from hepatocytes when the cells burst. Once released, the merozoites enter erythrocytes (red blood cells) and undergo the erythrocytic cycle, a process known as erythrocytic schizogony.[2]

Inside red blood cells, merozoites develop into trophozoites, which then multiply and mature into schizonts. The schizonts divide further to produce more merozoites within a single red blood cell. When the red blood cell bursts, these merozoites are released, and they have two possible pathways: they either invade new red blood cells, continuing the cycle or develop into gametocytes, which are capable of transmitting the infection.[3]

Some sporozoites in the liver may become dormant forms called hypnozoites, which can cause relapse in infections caused by P. vivax and P. ovale. When another mosquito feeds on a person carrying gametocytes, male and female gametocytes fuse within the mosquito to form a zygote. The zygote

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develops into an ookinete that penetrates the mosquito gut wall and encysts to form an oocyst. The oocyst divides to produce sporozoites that migrate to the mosquito's salivary glands, ready to be injected into another human during the mosquito's next bite, thus completing the cycle.[4]

Patients with a desirable lipid profile were considered normal. Patients with borderline and high-risk ranges are considered dyslipidaemia and more prone to coronary artery disease and peripheral artery disease. Dyslipidemia may have primary(genetic) or secondary causes. Malarial infection is a secondary cause of dyslipidaemia. Most secondary dyslipidaemias should resolve following adequate treatment of the underlying disease rather than treating dyslipidemia.[5] The disturbance of lipid metabolism caused by malarial infection is transient and malarial infection should be treated efficiently rather than using lipid-lowering drugs.6 Since there are only a few studies implicating malaria causing dyslipidaemia and because of the high incidence of malaria in our state, the present study was selected to determine if there is any association between the changes in serum lipid profile in malaria patients. Hence, we conducted this study among patients presenting with fever who were diagnosed with smear-positive malaria at the Stanley Medical College.

Aim

This study aimed to analyse the changes in lipid profiles of patients with different types of malarial fever.

MATERIALS AND METHODS

This prospective cross-sectional study was conducted on 100 inpatients who were smear-positive for malaria at the Government Stanley Medical College Hospital, Chennai, for nine months from May 2018 to January 2019. The study protocol was approved by the Institutional Ethics Committee. Consent forms were prepared in both English and Tamil, and the patients were verbally informed of their native language. Written consent was obtained and confidentiality was ensured throughout the study.

The sample size calculation was based on an expected proportion of 0.66, with a precision of 10% and a confidence level of 95%. Using Master software version 2.0, and assuming a 10% precision rate, the initial estimated sample size was 86 patients. The sample size was increased to 100 participants to account for 15% of the potential data loss.

Inclusion criteria

Male and female patients aged between 13 and 40 years admitted to Stanley Medical College Hospital during the study period who were positive for peripheral smear for malaria were included.

Exclusion criteria

Patients with febrile illness lasting more than seven days, and patients with comorbidities such as hypertension, diabetes, renal or hepatic disorders, malignancies, metabolic syndrome, HIV, pre-existing dyslipidaemia, or those on statin therapy were excluded.

Methods

Microscopic detection of malaria parasites

Blood samples were collected in EDTA tubes, and both thick and thin smears were prepared. Following Leishman staining, the slides were examined under a ×100 objective lens with oil immersion. Based on the smear results, patients were categorised into the following groups: Plasmodium falciparum infection, Plasmodium vivax infection, and mixed infection (Plasmodium falciparum + Plasmodium vivax).

Lipid profile estimation

Inclusion and exclusion criteria were applied to smear-positive patients, from whom a 5 ml blood sample was collected by sterile venipuncture. After centrifugation, serum levels of total cholesterol, triglycerides, HDL, and LDL were measured. Lipid levels were categorised as follows:

The desirable level for total cholesterol is < 200 mg/dl, whereas levels between 200-239 mg/dl are considered borderline, and levels ≥ 240 mg/dl fall into the high-risk category. For triglycerides, a desirable level is < 150 mg/dl; levels from 150-199 mg/dl are borderline, and levels ≥ 200 mg/dl are high risk. HDL cholesterol is considered desirable at levels ≥ 60 mg/dl, borderline between 35-45 mg/dl, and high risk if it falls < 35 mg/dl. LDL cholesterol has a desirable range between 60-130 mg/dl, borderline between 130-159 mg/dl, and is classified as high-risk at levels ≥ 160 mg/dl. Non-fasting samples were included based on evidence supporting the utility of non-fasting lipid profiles in cardiovascular risk assessment.

Statistical analysis

The data are presented as frequencies and percentages. Categorical variables were compared using Pearson's chi-squared test. Continuous variables were compared using an independent-sample t-test. Significance was defined as $p<0.05$, using a two-tailed test. Data analysis was performed using IBM-SPSS version 23.0 (IBM-SPSS Science Inc., Chicago, IL).

RESULTS

The total number of patients with malarial fever included in the study was 100. Among them, 53 were males and 47 were females. Of the 100 malaria cases, 11 patients were infected with P. falciparum, 34 patients had vivax, and 55 patients presented with mixed plasmodium. In all three types of malaria, the majority of the patients were in the age group of 31-40 years with a p-value of 0.597, which was not significant. The falciparum group had a higher proportion of females (72.7%) than the vivax (40%) or mixed (50%) groups. There were no significant gender differences between the groups (p=0.126).

Most patients had total cholesterol levels in the desirable range (<200 mg/dl), with 100% in both the falciparum and vivax groups and 98.2% in the mixed

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group. A single patient in the mixed plasmodium group was in the borderline category (200-239 mg/dl), with no significant difference (p=1). There were no patients with desirable triglyceride levels (<150 mg/dl) in both the falciparum and vivax groups, while 5.5% of the mixed plasmodium group had desirable triglyceride levels. The mixed plasmodium group also had the highest percentage of patients in the borderline (150-199 mg/dl) (21.8%). Triglyceride levels were significantly different between groups (p=0.043).

The mixed plasmodium group showed the highest percentage of patients with desirable HDL levels (63.6%), whereas the falciparum group had the lowest (9.1%). A significant difference in HDL levels was observed between the groups (p=0.002). Most patients across all groups had LDL levels below 60 mg/dl, particularly in the falciparum group (90.9%), followed by the vivax (76.5%) and mixed Plasmodium groups (69.1%). Only a few patients in each group had LDL levels within the desired range (60-130 mg/dl), with no patients across all groups in the borderline or high-risk LDL categories. A significant difference was noted in the LDL levels between the groups (p=0.029) [Table 1].

Both female (100%) and male (98.1%) were within the desirable range for total cholesterol. Patients with desirable triglyceride levels were low, with only 4.3% of females and 1.9% of males. The desirable HDL levels were relatively similar, with 44.7% of females and 49.1% of males. LDL levels showed that 76.6% of females and 71.7% of males were within the desirable range. There were no significant differences in TC, TGL, HDL, and LDL levels between the gender (p=0.343, p=0.42, p=0.908, and p=0.651, respectively) [Table 2].

For total cholesterol, males show a slightly higher mean level (114.21 ± 38.17 mg/dl) compared to females (108.89 ± 28.99 mg/dl). Triglyceride levels are also slightly higher in males (269.91 ± 71.24 mg/dl) than in females (260.13 ± 74.06 mg/dl). HDL levels are nearly the same between males (47.3 ± 10.24 mg/dl) and females (47 ± 10.24 mg/dl). LDL levels are slightly higher in males (55.43 ± 12.81 mg/dl) compared to females (52.21 ± 12.17 mg/dl), but the P-value of 0.202 indicates no significant difference. Both genders showed mean LDL levels below the desirable range, suggesting generally well-controlled LDL levels in this population. There were no significant differences in TC, TGL, HDL, and LDL levels between the gender (p=0.382, p=0.503, p=0.884, and p=0.202, respectively) [Table 3].

For TC < 200, no significant association was found across the groups (vivax, falciparum, and mixed plasmodium) (p=0.47, p=0.72, p=0.36). For TGL > 200, no significant associations were observed, with p=0.80 for vivax, p=0.27 for falciparum, and p=0.35 for mixed plasmodium. For HDL < 35, a significant association was found for both falciparum and mixed plasmodium, (p=0.05). However, no significant association was observed in the vivax group (p=0.39).

For LDL < 60, the falciparum group showed a significant association (p=0.01), whereas the vivax and mixed plasmodium groups did not show a significant association (p=0.93, p=0.33). For TC < 200 and TGL > 200, a significant association was found in the falciparum group (p=0.01), whereas vivax and mixed plasmodium showed no significant association (p=0.74 and p=0.51).

For TC < 200 and LDL < 60, no significant associations were observed across all groups, with p=0.86 for vivax, p=0.21 for falciparum, and p=0.54 for mixed plasmodium. For HDL < 35 and LDL < 60, a significant association was observed only in the vivax group (p=0.01), whereas falciparum (p=0.72) and mixed plasmodium (p=0.26) were not significant.

For TC < 200, TGL > 200 and LDL < 60, no significant associations were found in any of the groups, with p=0.65 for vivax, p=0.23 for falciparum, and p=0.74 for mixed plasmodium. For all four positive tests, a significant association was found in both the falciparum and mixed plasmodium groups (p=0.05), whereas the vivax group showed no significant association (p=0.39).

Table 1: Comparison of demographic details and lipid profile by malaria diagnosis.

		Diagnosis			P-value
		Falciparum (n=11)	Vivax (n=34)	Mixed plasmodium (n=55)	
Age (years)	Up to 20	2 (18.2%)	4 (11.8%)	13 (23.6%)	0.597
	21-30	3 (27.3%)	13 (38.2%)	14 (25.5%)	
	31-40	6 (54.5%)	17 (50%)	28 (50.9%)	
Gender	Female	8 (72.7%)	22 (40%)	17 (50%)	0.126
	Male	3 (27.3%)	33 (60%)	17 (50%)	
Total cholesterol	Desirable (< 200 mg/dl)	11 (100%)	34 (100%)	54 (98.2%)	1
	Borderline (200-239 mg/dl)	0	0	1 (1.8%)	
	High risk (<240 mg/dl)	0	0	0	
Triglycerides	Desirable (< 150mg/dl)	0	0	3 (5.5%)	0.043
	Borderline (150-199 mg/dl)	1 (9.1%)	6 (17.6%)	12 (21.8%)	
	High risk (<200-499 mg/dl)	10 (90.9%)	28 (82.4%)	40 (72.7%)	
HDL	Desirable (60 mg/dl)	1 (9.1%)	11 (32.4%)	35 (63.6%)	0.002
	Borderline (35-45mg/dl)	8 (72.7%)	20 (58.8%)	19 (34.5%)	
	High risk (<35 mg/dl)	2 (18.2%)	3 (8.8%)	1 (1.8%)	
LDL	Desirable (60-130 mg/dl)	1 (9.1%)	8 (23.5%)	17 (30.9%)	0.029
	Borderline (130-159 mg/dl)	0	0	0	

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	High risk (160-180 mg/dl)	0	0	0	
	LDL < 60 mg/dl (160-180 mg/dl)	10 (90.9%)	26 (76.5%)	38 (69.1%)	

Table 2: Lipid Profile Distribution by Gender

		Gender		P-value
		Female	Male	
TC	Desirable	47 (100%)	52 (98.1%)	0.343
	Borderline	0	1 (1.9%)	
TGL	Desirable	2 (4.3%)	1 (1.9%)	0.420
	Borderline	11 (23.4%)	8 (15.1%)	
	High risk	34 (72.3%)	44 (83%)	
HDL	Desirable	21 (44.7%)	26 (49.1%)	0.908
	Borderline	23 (48.9%)	24 (45.3%)	
	High risk	3 (45.3%)	3 (5.7%)	
LDL	LDL < 60	11 (23.4%)	15 (28.3%)	0.651
	Desirable	36 (76.6%)	38 (71.7%)	

Table 3: Mean Lipid Profile Levels by Gender

	Gender		P-value
	Male	Female	
TC	114.21 ± 38.17	108.89 ± 28.99	0.382
TGL	269.91 ± 71.241	260.13 ± 74.06	0.503
HDL	47.3 ± 10.239	47 ± 10.239	0.884
LDL	55.43 ± 12.809	52.21 ± 12.167	0.202

Table 4: Statistical analysis of lipid profile parameters by Plasmodium species

Tests studied	Vivax		Falciparum		Mixed plasmodium	
	chi-square value	P-value	chi-square value	P-value	chi-square value	P-value
TC<200	0.52	0.47	0.12	0.72	0.82	0.36
TGL>200	0.05	0.8	1.2	0.27	0.85	0.35
HDL<35	0.72	0.39	3.25	0.05	3.7	0.05
LDL<60	0.007	0.93	2	0.01	0.94	0.33
TC<200 & TGL>200	0.1	0.74	2.3	0.01	0.43	0.51
TC<200 & LDL<60	0.03	0.86	1.5	0.21	0.37	0.54
HDL<35 &LDL<60	1.9	0.01	0.24	0.72	1.2	0.26
TC<200, TGL>200 & LDL<60	0.19	0.65	1.4	0.23	0.1	0.74
All 4 positive	0.72	0.39	3.2	0.05	3.7	0.05

DISCUSSION

The findings of this study revealed significant alterations in the lipid profiles of patients with malaria, indicating a clear association between the disease and dyslipidaemia. Specifically, all patients exhibited hypertriglyceridemia, with 90.9% of those with Plasmodium falciparum and 82.4% with Plasmodium vivax classified in the high-risk triglyceride range. Additionally, HDL levels were notably low across all malaria types, particularly in falciparum infections, where 90.9% of the patients fell below the desirable range. LDL levels also demonstrated a significant decrease, with 90.9% of falciparum patients showing LDL < 60 mg/dl, suggesting a disturbance in lipid metabolism due to malaria. There was a significant decrease in total cholesterol, HDL, and LDL, with an increase in triglyceride levels.

Visser et al. states that serum lipid profiles are altered during malaria. There was a transient decrease in total cholesterol, HDL, and LDL, and an increase in triglyceride levels. They also stated that the changes in serum lipid profile were temporary and recovered with malaria treatment. They concluded that serum lipid profile changes seen in malaria are hypocholesterolemia, decrease in HDL and LDL and hypertriglyceridemia.[6]

Babaliche et al. conducted a study on variations in common serum lipid parameters in patients with malaria. Serum lipid profile estimation in these patients revealed that 60% of them had low total cholesterol levels, 56% had low-LDL levels, and 58% had low-HDL levels. However, 92% of the patients had hypertriglyceridemia.[7]

Dungdung et al. studied the serum lipid profile of Plasmodium falciparum malaria. They stated that the total cholesterol, HDL and LDL were significantly decreased, while triglyceride was not significantly changed in P. falciparum malaria patients.[8] In our study though total cholesterol, HDL, LDL were decreased as of their study, but the level of triglyceride is not changed in their study but in our study, there is an elevation of triglyceride. Lambrecht et al. concluded that vivax patients had alterations in lipid profile, and major changes with alterations in HDL and LDL were noted.[9] But in our study, there was an alteration in lipid profile, in all four parameters such as total cholesterol, triglyceride, HDL, and LDL.

Kiru et al. conducted a study to determine the serum lipid profile and electrolyte level and their association with the severity of malaria. There were significantly

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higher levels of TC, LDL, TAG, and VLDL, and lower levels of HDL in malaria-infected patients than in the control group. However, the HDL levels decreased significantly. Also, there was a significant decrease in the levels of Na+ and K+, while Cl- was not significantly different between malaria-infected and control patients, demonstrating that characteristic serum lipid profiles and electrolyte changes occur during malaria. Triglycerides were elevated in our study as in their study, but total cholesterol and LDL were decreased in our study but increased in the study by Kiru et al. HDL is decreased in both studies.[10]

Solomon Sirak et al. conducted a study regarding changes in lipid profile. A large number of malaria patients were infected with Plasmodium falciparum (66%) in their study.[11] But in our study, majority were in the mixed plasmodium group. There were significant mean differences in the lipid profiles of patients with malaria in their study, as in our study.

Warjri et al. analysed the correlation between malaria infection and derangements in lipid profiles. The observations showed a significant difference in HDL, LDL, cholesterol, and triglyceride levels between malaria patients and control subjects. These results show a characteristic pattern of derangements of lipid profile in malaria.[12] Hence this study also supports our study in the changes that occur in lipid profile during malaria.

Musali and Reddy conducted a study on the lipid profile in malaria, which states that transitory changes in the plasma lipid levels have been observed and are related to the severity of malaria. Total cholesterol and HDL are decreased, while LDL shows no significant change and triglyceride levels decreased.[13] The results of this study do not align with our study.

Jean-Francois Faucher concluded that there is an alteration in lipid profile in malaria with an increase in triglyceride and a decrease in HDL, LDL and total cholesterol.[14] These findings in this study supported the results of our study.

Kumar et al. conducted a study on lipid profiles in newly diagnosed cases of Plasmodium falciparum-infected malaria patients. A study was done and results showed that THERE WAS an elevation in triglyceride level with a decrease in HDL, LDL and total cholesterol which supports the findings in our study.[15]

Mesquita et al. studied the transient change in lipid profile between the acute and convalescent stages of malaria. There was a raised level of total cholesterol, HDL and LDL with a decrease in triglyceride level.[16] This pattern of de arrangement is not seen in our study.

Limitations: A limited number of patients were evaluated in this cross-sectional study and controls were not included. The number of patients in the mixed Plasmodium group was higher than those in the P. falciparum and P. vivax groups, which may be attributed to the small sample size. Further studies with larger sample sizes are required to obtain stronger findings. Studies with larger patient groups across all age groups should be conducted. Prospective studies are encouraged to accurately determine the incidence of dyslipidaemia in patients with malaria.

CONCLUSION

Malaria infection is associated with significant changes in the serum lipid profiles. Elevated serum triglyceride levels indicate hypertriglyceridemia, whereas reductions in serum total cholesterol, HDL, and LDL are commonly observed in malaria cases. When all four lipid criteria are met, that is, total cholesterol below 200 mg/dL, triglycerides above 200 mg/dL, HDL below 35 mg/dL, and LDL below 60 mg/dL, the chances of diagnosing P. falciparum or mixed Plasmodium infections increase significantly. Specifically, an HDL level < 35 mg/dL has a strong diagnostic value for identifying mixed infections and P. falciparum, whereas HDL and LDL levels serve as predictors for P. vivax in regions where this infection is prevalent. This lipid profile-based approach could be a valuable diagnostic aid for differentiating malaria types, especially in resource-limited settings. Further research with a larger sample size is necessary to confirm these findings.

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