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Research Article

STUDY OF SERUM LIPIDS IN SEVERE FALCIPARUM MALARIA

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A R T I C L E I N F O

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A B S T R A C T

Background and Objectives: Malaria is a potentially life threatening disease, which is transmitted by the infectious bite of the female Anopheles mosquito. In India, malaria is a major public health problem. Various studies have found that hypocholesterolemia and hypertriglyceridemia correlate with the severity of falciparum malaria.
This study intends to find a relationship between serum cholesterol and serum triglycerides done before initiating treatment and the severity of falciparum malaria.
Material and Methods: A total of 50 patients who had one or more of the features of severe malaria were selected and random serum cholesterol and serum triglycerides were sent prior to initiation of treatment .different parameters were compared with other similar studies. **Results:** All patients with severe falciparum malaria had low serum cholesterol levels. Seventy percent of patients had elevated serum triglyceride levels.
Elevated triglycerides were also associated with elevated levels of serum creatinine in these patients. A low random blood sugar correlated with a low cholesterol level in study population.
Conclusions: This study illustrates the significant impact of severe falciparum malaria on common lipid values in adults.

INTRODUCTION

Malaria is a potentially life threatening disease, which is transmitted by the infectious bite of the female Anopheles mosquito. In India, malaria has been a major public health problem since ages. Often 0.5% to 2% of P. falciparum cases (malignant variety of malaria) may develop severe malaria with complications. In such cases death rates may be 30% or more, if timely treatment is not commenced.[1]

Life cycle. Infection in humans begins when a female anopheline mosquito inoculates plasmodial sporozoites from its salivary gland during a blood meal. Within an hour, the sporozoites enter liver cells to initiate the exoerythrocytic phase. By this amplification process, a single sporozoite eventually may produce from 10,000 to >30,000 daughter merozoites. The swollen infected liver cell eventually bursts, discharging motile merozoites into the bloodstream. These then invade the red blood cells and multiply every 48 – 72 h. After entry into the bloodstream, merozoites rapidly invade erythrocytes and become trophozoites. As the trophozoites enlarge, pigment becomes visible, and the parasite assumes an irregular or ameboid shape. By the end of the 48-h intraerythrocytic life cycle, the parasite has consumed nearly all the hemoglobin and grown to occupy most of the RBC.

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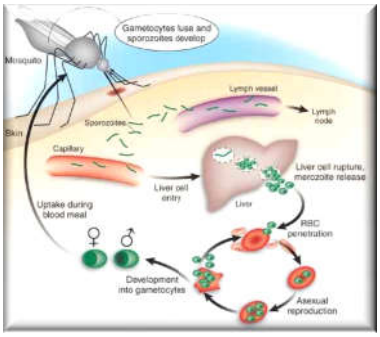
It is now called a schizont. Multiple nuclear divisions have taken place (schizogony or merogony), and the RBC then ruptures to release 6–30 daughter merozoites, each potentially capable of invading a new RBC and repeating the cycle. The disease in human beings is caused by the direct effects of RBC invasion and destruction by the asexual parasite and the host's reaction. After a series of asexual cycles, some of the parasites develop into morphologically distinct, longer-lived sexual forms (gametocytes) that can transmit malaria. This zygote matures into an ookinete, penetrates and encysts in the mosquito's gut wall. The resulting oocyst expands by asexual division until it bursts releasing many motile sporozoites, which then migrate in the hemolymph to the salivary gland of the mosquito to await inoculation into another human at the next feeding.[2,3] lifecycle of plasmodium [2,3]



Objective

To assess serum serum cholesterol and serum triglycerides levels in severe falciparum malaria.

Background

RBCs are highly differentiated and have a limited metabolic repertoire and no protein synthesis or protein trafficking capability. The virulence of P. falciparum is due in large part to the export of proteins to the host cell compartment. These exported proteins modify the properties of the host cell membrane thereby facilitating access to nutrients and providing a mechanism for evading the immune system. Of particular importance is a parasite-encoded family of proteins known as P. Falciparum erythrocyte membrane protein 1 (PfEMP1). Members of this large variant family mediate adhesion of infected RBCs to a number of host molecules, including ICAM-1, CD36, complement receptor-1 and chondroitin sulphate A (CSA).

The adhesion process sequesters infected RBCs away from the peripheral circulation and prevents phagocytic clearance in the spleen. Accumulation of infected RBCs within the microvasculature in vital organs such as the brain and the placenta leads to complications such as cerebral malaria, which is often fatal, and placental malaria, which predisposes to foetal and maternal. Studies show that lipids and lipoproteins are required for parasite growth and effective adhesion molecule expression.[4]

Parola *et al*[5] studied a total of 278 febrile patients returning from the tropics who were hospitalized .Patients were 15–75 years old with a mean age of 35.7 years A total of 222 malaria cases were diagnosed including 198 (89%) due to P. falciparum, 14 (6%) due to P. vivax,8 due to P. ovale (4%), and 2 due to P. malariae (2%).Mean value of plasma triglycerides was significantly higher in patients with malaria .Mean values of triglycerides were similar in mild falciparum and non-falciparum malaria groups, but clearly higher in patients with severe malaria than in those with mild malaria. In 1983, lipid concentrations were determined during and after falciparum malaria in 18 young male adults (average age 21 years) in Nigeria[6]. Triglycerides levels were higher during both mild (n=9) and severe (n=6) malaria than after convalescence. Although the comparison was not provided in this last study, mean triglyceride level during severe malaria was higher than during mild malaria.

Alterations in the levels of plasma and erythrocyte membrane lipids in fresh and repeated P. vivax malarial patients were studied by Sumithra *et al*[7]. A significant fall in plasma cholesterol and phospholipids was observed in repeated malaria. The decrease was highly significant when the number of attacks were more than five ($p < 0.0001$). A significant increase in plasma triglycerides and non-esterified fatty acids were observed when the number of attacks was between 4-5 ($p < 0.0001$). Erythrocyte membrane cholesterol and phospholipids were increased in repeated malaria.

Nilsson-EI[8] showed that plasma triglyceride concentrations were moderately increased and plasma low density lipoprotein (LDL) cholesterol concentrations decreased during the course of infection.

There have been studies that have shown a fall in triglycerides and rise in cholesterol in malaria contrary to the above mentioned studies.

MATERIAL AND METHODS

Source of data: Patients admitted to SAIMS Hospital with severe falciparum malaria. Method of Collection of Data Sample size: Data was collected in 2 years (01/01/2018 to 31/12/2019) with a sample size of 50 patients.

Methods: The following clinical parameters were assessed at the time of admission Higher mental functions using Glasgow coma scale, Blood pressure, Urine output Blood was drawn under aseptic precautions from cases of malaria and sent for peripheral smear to detect malarial parasite.

The biochemical and haematological markers studied at the time of admission were: Platelet counts, Hb, TC, Random serum cholesterol and serum triglycerides done at admission just before starting treatment, Total bilirubin, Random blood glucose, Prothrombin time.

Selection Criteria-Inclusion Criteria: **1.** A case is defined as any patient hospitalized in the inpatient ward with fever who has microscopically proven diagnosis of Falciparum malaria. Patient should have one or more of the features of severe malaria mentioned below[1.] Cerebral malaria- unexplainable unarguable coma lasting more than 30 minutes after a seizure. Severe anaemia –haematocrit <15% or haemoglobin<5gms/dl. Renal failure-urine output <400ml/24 hr inspite of adequate hydration; serum creatinine>3mg/dl. Disseminated intravascular coagulation-spontaneous bleeding from gums, nose and gastrointestinal tract; prolonged prothrombin time, prolonged activated prothrombin time; platelet count <1,50,000/mm3 Hypoglycemia- plasma glucose level <40 mg/dl. Acidosis–arterial ph<7.25 or plasma bicarbonate levels <15mmol/l. Haemoglobinuria 2. Age group >12 years.

Exclusion Criteria: Hypertensive and diabetic patients. Patients with prior history of hyperlipidemia. Patients who have nephrotic syndrome and chronic alcoholics. Patients who have already been started on antimalarial treatment prior to admission.

RESULTS

In this study population, out of 50 patients, 38 patients (76%) were males and 12 patients (24%) were females. The average age of patients in the study group was 34.8 years.

Table 1 Range and mean age

	Numbers	Minimum	Maximum	mean
Age	50	15.00	82.00	34.82

Table 2 distributions of symptoms in study population.

Symptom	Positive	Negative	Freq of positive symptom (%)
Fever	47	3	84
Headache	20	30	40
Vomiting	27	23	54
Cough	1	49	4
Jaundice	3	47	6
Reduced urine output	1	49	2

Table 3 CNS manifestations in study population

CNS manifestations	Frequency	Percent of total
Altered sensorium	3	6
Seizures	2	4
Cerebellar signs	1	2

The most common presenting symptoms for patients in this study were fever and vomiting.

Table 4 Mean values of laboratory investigations.

	Number	Minimum	Maximum	Mean
Urine output(cc/day)	50	150	1800	885
Haemoglobin(g/dl)	50	6.3	16.2	10.9
Platelets(per mm3)	50	23000	1,40,000	62640
Creat(mg/dl)	50	0.5	5.3	1.49
Urea (mg/dl)	50	36	180	47.29
Rbs (mg/dl)	50	70	175	111.36
t bil (mg/dl)	50	0.6	18.5	3.38
Cholesterol(mg/dl)	50	32	122	78

All patients in the study had low cholesterol levels. Elevated triglycerides were found in 35 patients (70%).

Table 5 Correlations between serum lipids and other variables. Correlationsa

	Uo	Hb	platelet	creat	urea	rbs	t bil
cholesterol r	-.018	-.096	-.033	.038	.038	.342	-.188
P value	.904	.507	.818	.792	.793	0.01	.192
N	50	50	50	50	50	50	50
triglycerides	-.222	.059	-.009	.255	.059	.117	-.017
p value	.121	.683	.951	.074	.682	.420	.908
N	50	50	50	50	50	50	50

Elevated triglycerides were also associated with elevated levels of serum creatinine in these patients. Among the other parameters tested a low random blood sugar correlated with a low cholesterol level in study population. The most common combination of complications found in this study group was that of thrombocytopenia and jaundice.

DISCUSSION

The number of reported cases of malaria in India in 2005 by the WHO was 1.8 million cases. There were 963 deaths reported due to malaria. Forty four percent of cases were falciparum malaria. Mortality due to falciparum malaria is a major concern.[1]

In this study the most common symptoms patients presented with were fever and vomiting and the most common signs were pallor and icterus. The most common combination of complications found in our study was thrombocytopenia and icterus. In a study done by Tripathy *et al* the most common manifestations of severe malaria in adults were complications like acute renal failure, respiratory distress and cerebral malaria.[9,10]

Hypocholestrolemia was present in all the patients of our study and this correlated with a low plasma sugar level at admission. Previous studies done by Kittl *et al*[11], Djoumessi [12] and Nilsson *et al*[8] have shown the same relation between severe malaria and serum cholesterol.

The mechanisms involved in lipid changes related to malaria remain uncertain .They may be partly host-related, i.e. related to an acute phase reaction, which is known to increase triglyceride values and to decrease both HDL- and LDL-cholesterol values. [13,14].However, a selective uptake of HDL particles by P. falciparum has been speculated and further shown by in vitro experiments.[4]

CONCLUSION

This study illustrates the significant impact of severe falciparum malaria on common lipid values in adults. Mechanisms underlying these changes though not completely understood, could be useful in the future to prognosticate patients with severe malaria. Hence more studies are required to study this relation.

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