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Research Article

**A DESCRIPTIVE RESEARCH ON ASSOCIATION OF DISEASE
INTERVAL & DURATION IN FALCIPARUM MALARIA
CASES: LIVER FUNCTION TEST (LFT) Vs HAEMOGLOBIN**¹Zunaira Zulfiqar, ²Soma Butt, ²Tayyaba Ibrahim¹THQ Civil Hospital Daska²Children Hospital Lahore**Abstract:**

Objective: Aim of this particular research was to observe association of LFT (Liver Function Test) Versus hemoglobin test in falciparum malaria patients.

Patients And Methods: Our research was descriptive in nature which was held on 81 cases in the age bracket of (3 – 56) years at Biochemistry Dept. Mayo Hospital, Lahore (July, 2016 to August, 2017). Acute malaria cases were included from both the genders in our research after confirmation through consecutive sampling and blood smear. We did not include all the cases of HBV and HCV (09 cases). Remaining 72 cases had 48 cases (70%) Plasmodium falciparum malaria and 24 cases (30%) infected by Plasmodium vivax. Two equal groups were made of falciparum infected cases on the interval of their illness. Fever complainant patients were included in Group “I” having no rigors in the range of (1 – 7) days. Twenty-four cases were included in Group “II” with disease duration of (8 – 20) days.

Results: Both the groups showed significant positive association in terms of hematocrit percentage with hemoglobin (P -value < 0.01). The SGPT, bilirubin and SGOT in second group had a negative and weak association with hemoglobin; whereas, it was statistically significant (P -value < 0.05). Non-significant association was found in both groups about all other parameters.

Conclusion: As Fulminant hepatitis can be manifested by Falciparum; so, LFT is to be carried out along with Plasmodium falciparum malaria's early diagnosis for the possible prevention of complications to eradicate the chances of mortality.

Key Words: AST, ALT, Malaria, Falciparum and LFTs.

Corresponding author:

Zunaira Zulfiqar,
THQ Civil Hospital,
Daska

QR code



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INTRODUCTION:

Tropical areas face malaria as a deadly infection which causes the death of one to three million every year and infected cases are about 300 – 500 million all over the world [1]. Severe malaria caused most of the deaths with multiple complications of infection of *Falciparum* [2]. Infection of the malaria is caused by the liver's sporozites infection [3]. Sporozoites of the malaria are injected through female *Anopheles* mosquitoes' bite which attaches to hepatocytes with the help of properdin and thrombospondin receptor [4]. Liver stage is initiated by invasion of Hepatocyte. It ensues the excessive multiplication and differentiation, and every stage of the liver shows yielding of the thousands of merozoites [5]. Severe malaria's clinical manifestations are linked with the strong pro-inflammatory type – I induction of the immune responses [6]. Jaundice and liver involvement is common such as elevated bilirubin serum, liver enzymes (alanine and aspartate transaminases) and hepatomegaly [7].

Severe anemia pathogenesis in malaria can be attributed to such factors (malnutrition, bone marrow dysfunction, iron deficiency and parasitaemia level) [8]. AST and ALT elevation with conjugated hyperbilirubinemia in the prolonged illness brings adverse outcomes such as consciousness impairment which is also linked with mortality and associated with cerebral malaria or renal failure [9 – 11].

Therefore, Aspartate aminotransferase is available in liver and other diverse tissues such as muscle, kidney, heart and brain. In case of injury of any tissue it increases; therefore, it cannot be taken as liver damage marker [12]. Elevated alkaline phosphatase serum activity in patients shows *falciparum* malarial infection liver stage which is accompanied by host hepatocytes perturbation drainage pathways and hepatocytes membrane damage which leads to enzymes leakage from cells of liver [13]. Aim of this particular research was to observe association of LFT (Liver Function Test) Versus hemoglobin test in *falciparum* malaria patients.

PATIENTS AND METHODS

Our research was descriptive in nature which was held on 81 cases in the age bracket of (3 – 56) years at Biochemistry Dept. Mayo Hospital, Lahore (July, 2016 to August, 2017). Acute malaria cases were included from both the genders in our research after confirmation through consecutive sampling and blood smear. We did not include all the cases of HBV and HCV (09 cases). Remaining 72 cases had 48 cases (70%) *Plasmodium falciparum* malaria and 24 cases (30%) infected by *Plasmodium vivax*. Two

equal groups were made of *falciparum* infected cases on the interval of their illness. Fever complainant patients were included in Group "I" having no rigors in the range of (1 – 7) days. Twenty-four cases were included in Group "II" with disease duration of (8 – 20) days.

Detailed physical assessment and history was documented and a Performa was filled by the patients after informed consent about name, address, age and illness duration, fever type, jaundice history, drug history, diabetes history and other related diseases. Clinically hematocrit and hemoglobin estimation was also carried out. Random blood glucose level was also checked through a strip of glucometer. Nine cases were positive for HBV and HCV so they were not included in the research which reduced the final research sample. All the cases of fever but no rigors, intake of hepatotoxic drugs, mixed cases of malarial infections, expectant mothers and positive cases of hepatitis were not included in the research.

Statistical analysis was made on the SPSS software. We also measured Mean and SD values with significant P-values including correlation coefficient.

RESULTS:

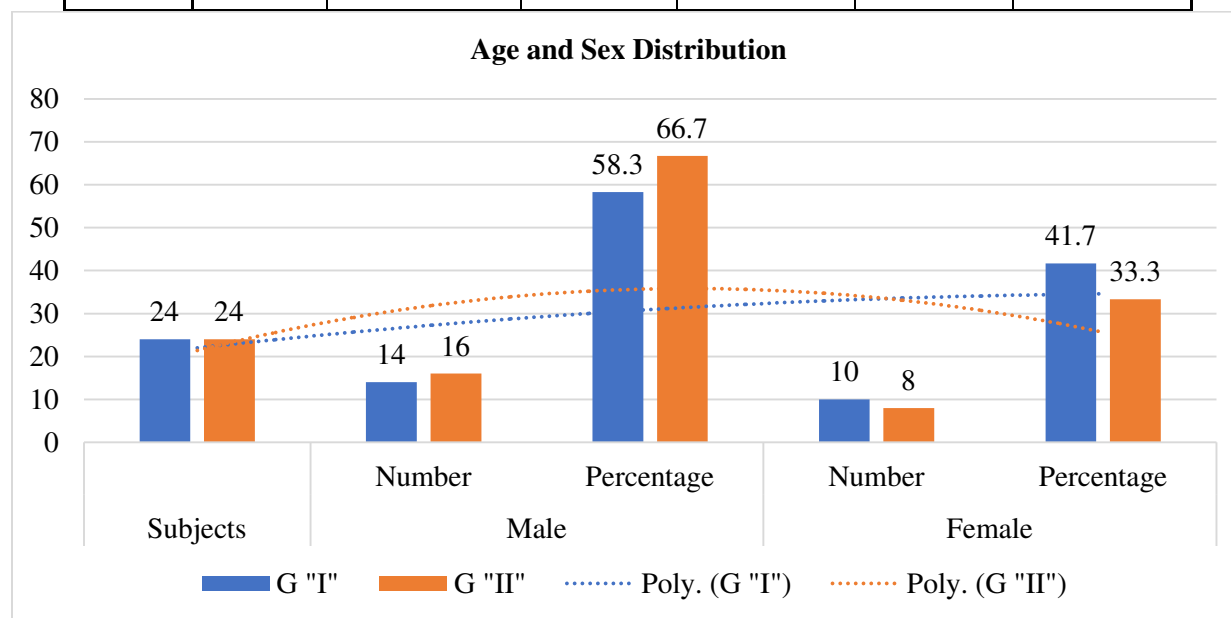
Both the groups showed significant positive association in terms of hematocrit percentage with hemoglobin (P-value < 0.01). The SGPT, bilirubin and SGOT in second group had a negative and weak association with hemoglobin; whereas, it was statistically significant (P-value < 0.05). Non-significant association was found in both groups about all other parameters.

The range of the temperature in fever was from (100 – 104) degree F, which had an association with body ache, nausea and headache in ninety-five percent of the cases and vomiting was noticed in five percent cases. Illness duration was (1 – 20) days and in the past four month no blood transfusion history. Jaundice and Pallor was present respectively in 52.7 % and 63.8 % cases. No case of ascites, bleeding or edema was observed. Enlargement of liver was observed in 28 cases (38.9%) in the range of (0.5 – 4.0) cm; whereas, 27 cases had palpable spleen (37.5%) in the range of (0.5 – 3.0) cm. Above two centimeters liver enlargement cases underwent Ultrasonography and normal architecture without any drainage obstruction or dilation was observed. No complications were noticed in all the cases without any unconsciousness.

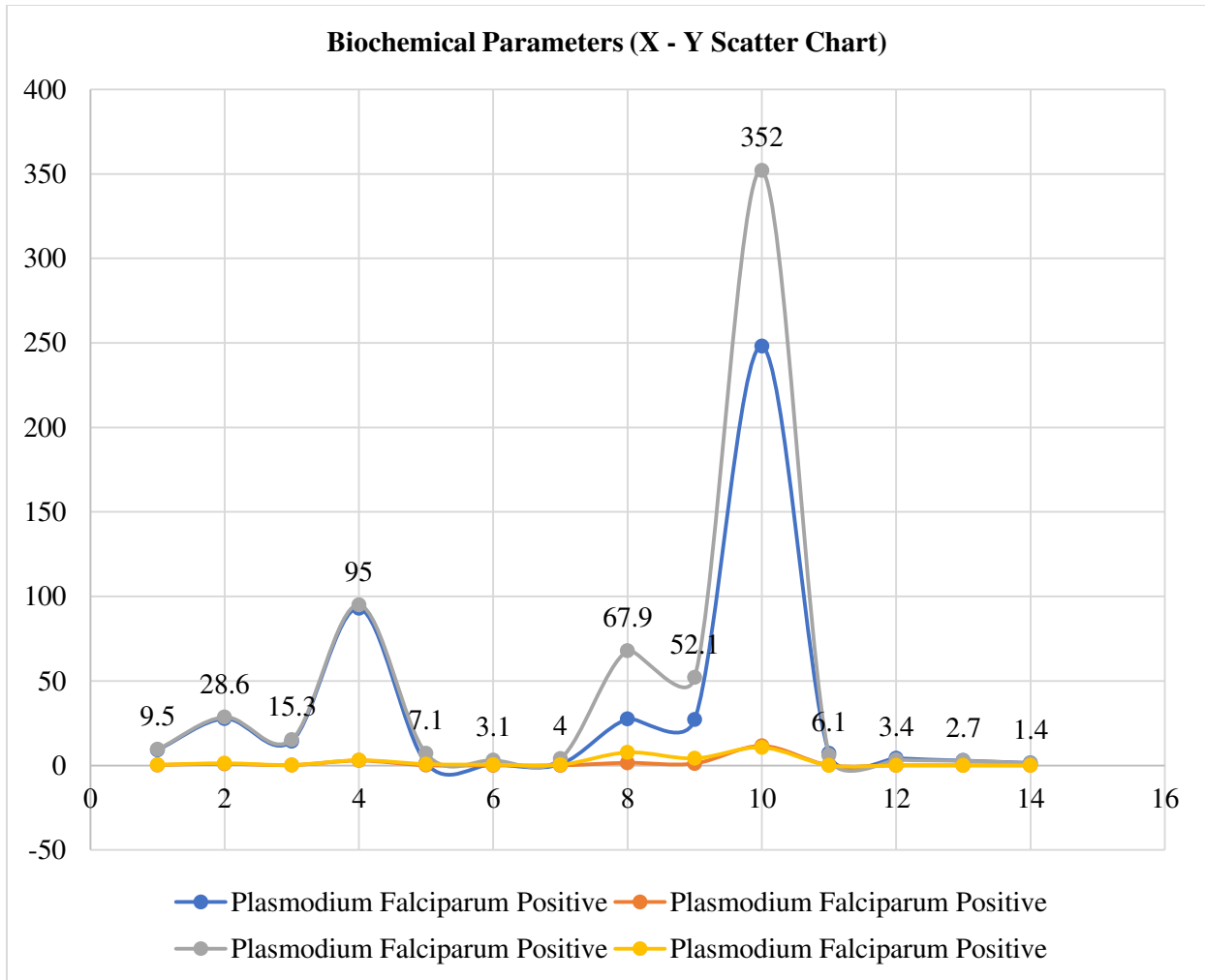
Detailed outcomes analysis has been depicted in the given tabular data.

Table – I: Falciparum Malaria (Sex and Age Distribution)

Groups	Subjects	Age	Male		Female	
			Number	Percentage	Number	Percentage
G "I"	24	25.2 ± 3.33	14	58.3	10	41.7
G "II"	24	24.7 ± 2.71	16	66.7	8	33.3

**Table – II: Group I & II (Biochemical Parameters Comparison)**

Biochemical Parameters (Unit)	Plasmodium Falciparum Positive				P Value
	Group I (24)		Group II (24)		
	Mean	(±) SEM	Mean	(±) SEM	
Hemoglobin (g/dl)	9.2	0.31	9.5	0.44	0.578
Hematocrit (%)	27.7	0.97	28.6	1.31	0.598
Prothrombin time (Control: 11 to 16 sec)	14.4	0.23	15.3	0.31	0.016
Random Blood Glucose (mg/dl)	93	2.89	95	3.11	0.619
Bilirubin - Total (mg/dl)	1.4	0.13	7.1	0.83	0
Direct (mg/dl)	0.6	0.07	3.1	0.49	0.001
Indirect (mg/dl)	0.8	0.08	4	0.43	0.001
ALT (U/L)	27.5	1.59	67.9	7.72	0.001
AST (U/L)	27.2	1.19	52.1	4.21	0.001
Alkaline Phosphatase (U/L)	248	11.6	352	10.7	0.001
Total protein (g/dl)	7.2	0.14	6.1	0.06	0.001
Albumin (g/dl)	4.3	0.08	3.4	0.08	0.001
Globulin (g/dl)	2.9	0.1	2.7	0.1	0.072
A/G ratio	1.5	0.06	1.4	0.09	0.28



R-value was observed in both Groups as (0.99); which represents positive association to hemoglobin, (P < 0.01).

DISCUSSION:

Pakistan is observed with common malarial species of vivax and Plasmodium falciparum. Malaria includes liver; where infected sporozoites multiply and invade hepatocytes and in the stage of erythrocyte, infected cells of red blood are destroyed by merozoites [15]. Our research evaluated the falciparum malaria damage to acute hepatic function. Subjects were selected on the basis of physical and biochemical parameters respectively (fever, liver size, spleen size, parasite form in blood smear) and (bilirubin, protein and enzymes). Enlargement of liver was observed in 28 cases (38.9%) in the range of (0.5 – 4.0) cm; whereas, 27 cases had palpable spleen (37.5%) in the range of (0.5 – 3.0) cm. Above two centimeters liver enlargement cases underwent Ultrasonography and normal architecture without any drainage obstruction or dilation was observed. No complications were noticed in all the cases without any unconsciousness. Jaundice and Pallor was present respectively in 52.7 % and 63.8 % cases. No

case of ascites, bleeding or edema was observed. Low rate of hemoglobin in malaria can be attributed to acute destruction or hemolysis of infective and non-infective red blood cells and nutritional deficiency [1].

Hemoglobin outcomes as shown in Table – II can be compared with the outcomes of Samiullah and Bhalli; whereas, non-comparable in terms of mean value with the outcomes as observed by Nadeem (13.78 g/dl) [4 - 16]. There is a positive relation of hematocrit of both groups with mean hemoglobin. We observed 64.3 % cases of hyperbilirubinemia which is near to the outcomes of Abro who observed level of serum as (81%) [22]. Indirect higher mean bilirubin of second group has a relation with illness duration and it is comparable with the outcomes of Irfan [20]. In falciparum malaria cases abnormalities of coagulation are common. We observed prothrombin time mean values in Group I & II as 14.4 & 15.3; whereas, four cases in the total of forty-

eight who were infected by *Plasmodium falciparum* had prolonged prothrombin time and no bleeding was observed in these cases which is same as observed by Abro but contradictory to the findings of Premaratna as he observed a prothrombin time as eighteen seconds [14]. Vogetseder and our outcomes about coagulation system impairment including disease severity are same [17]. Malaria parasite erythrocyte stages relation with glycolysis in terms of supply of energy is well established [18]. Hypoglycemia can be attributed to reduced intake, liver glycogen depletion and consumption of glucose by parasites. Random blood glucose outcome was normal glycemic. Van Thien outcomes are contradictory about the quantification of the gluconeogenesis in the patients of severe malaria [19]. Elevated level of hepatic enzymes serum, alkaline phosphatase and SGPT and SGOT transaminases are indicators of liver abnormalities. In group "II" specific liver enzyme SGPT SGOT were increased, same has been forwarded by Shah and Premaratna [14, 21]. Variation in the enzymes mean value in both groups is significantly high when we compared the infected cases of *Plasmodium falciparum* in both I & II groups; these outcomes correlate with the outcomes of Ubom and Garba [13]. Poor significant association was observed about total protein with hemoglobin.

CONCLUSION:

As Fulminant hepatitis can be manifested by *Falciparum*; so, LFT is to be carried out along with *Plasmodium falciparum* malaria's early diagnosis for the possible prevention of complications to eradicate the chances of mortality. Valuable information can be extracted through the outcomes of this research about the relation between patients of *Falciparum* malaria and hepatic biochemical imbalance.

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