International Archives of BioMedical and Clinical Research
E-ISSN:2454-9894 P-ISSN: 2454-9886
(An Official Publication of "Ibn Sina Academy of Medieval Medicine & Sciences")
WWW.IABCR.ORG

Original Article

Study of Malaria Parasite with Special Reference to Liver Function Test of the Patient Attending at TMU Hospital Moradabad (U.P)

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ABSTRACT

Background: *Malaria is an important infectious vector borne disease caused by a Plasmodium species. Liver involvement in severe Plasmodium falciparum infection is commonly a significant cause of morbidity and mortality among humans.* ***Objective:*** *In this study, we show the correlation of malaria positive cases with liver derangement.* ***Methods:*** *The present study had been conducted in Teerthankar Mahaveer Medical college Hospital and Research Center, Moradabad, U.P. from February 2014 to January 2015 on both IPD & OPD patients .****Results:*** *The present study included 200 clinically suspected cases of Malaria with derange Liver function. 67.5% patients show increased level of Total and indirect bilirubin followed by 45% of patient shows increased level of direct bilirubin. 27.5% patient's shows increased level SGPT followed by 40% of SGOT.* ***Conclusions:*** *Deranged liver functions are commonly seen as a complication of severe malarial infection.*

Key words: *: plasmodium, liver function, malaria*

Section – Microbiology

INTRODUCTION

Malaria is an important infectious vector borne disease caused by a *Plasmodium species* and it has been estimated that worldwide there are 300-500 million cases of malaria per year and 1.5-2.7 million deaths due to it.

Malaria is the most important protozoal parasitic disease of humans affecting more than one billion people worldwide and causing between 1 and 3 million deaths each year.[1] Malaria is caused by obligate intracellular parasites, which live in host erythrocytes and remodel these cells to provide optimally for their own needs.[2] Malaria parasite belongs to the genus Plasmodium. There are 156 named species of Plasmodium which infect various species of vertebrates. Four are known to infect humans: P. Falciparum, P. vivax, and P. malariae and P. ovale. P. vivax has widest distribution, extending throughout the tropics, subtropics, subtropics and temperate zones. Plasmodium falciparum is the most dangerous form of malaria, with the highest rates of complications and mortality. It is much more prevalent in sub- Saharan Africa than in many other regions of the world; is most African countries, over 75%of cases were due to P. Falciparum, whereas in most other countries with malaria transmission, other, less virulent plasmodial species predominate. P. falciparum is the most pathogenic of the human plasmodium species. It causes; sub-tertian fever, malignant fever, pernicious malaria, cerebral malaria and algid malaria.[3]

Plasmodium. Vivax (P. vivax) malaria is a public health problem that puts billions of the world's populations at risk of infection as highlighted in the World Malaria Report 2010.[4] P. vivax has a greater ability to survive in non-conducive environments; however, it is still considered to run a relatively benign disease course as compared to Plasmodium falciparum.

Access this article online	
Website: www.iabcr.org	Quick Response code
DOI: 10.21276/iabcr.2017.3.1.10	

Received:03.02.17| **Revised**:16.02.17| **Accepted**:17.02.17

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P. vivax malaria is commonly called "benign tertian malaria"; however, features of acute presentation are confounded by multiple relapses occurring because of the activation of dormant hypnozoites residing in the liver. The classical clinical presentation in a non-immune patient causes high-grade fever with headaches and prostration with a regular 48-hour periodicity. Most patients are treated as outpatients in endemic areas, with chloroquine being the cornerstone of treatment.[5-7] Malarial transmission to the human host is established by sporozoites infection to the liver.[8] The malarial sporozoites once injected in blood by the bite of female Anopheles mosquitoes are attached to hepatocytes through receptor for thrombospondin and properdin.[9] Here these sporozoites become mature to form tissue schizonts or become dormant hypnozoites.

Tissue schizonts amplify the infection by producing large number of merozoites (10,000 to 30,000). Each merozoite released from the liver is capable of invading a human red blood cell and establishing the asexual cycle of replication in that red cell with the release of 24 to 32 merozoites at the conclusion of 48 to 72 hours asexual cycle.[10] Malaria causes abnormalities in the liver however, opinions differ about the clinical importance of this damage.[11]

Liver involvement in malaria is common in patients of severe malaria and may manifest as jaundice, hepatomegaly and elevated liver enzymes like aspartate and alanine transaminases.[12] The factors leading to severe anemia in malaria are multiple. It may be due to hemolysis, bone marrow dysfunction etc and is proportional to the level of parasitaemia.[13]

METHODS

The present study was conducted in the research lab, department of microbiology and biochemistry, at Teerthanker Mahaveer medical college hospital and research center, Moradabad U.P.

Sample size: -Total 200 patient samples were included in my study.

Methodology: -

Diagnosis of malaria is done by microscopy is done by peripheral blood smear examination. It remains gold standard for conformation to diagnosis of malaria. Thick and Thin blood smears were prepared and both are stained with Leishman's stain. Than the smear are examined to the different stages of malaria parasites under oil immersion lens.

Diagnosis of liver function by the semi autoanalyzer of, Semmens Company using commercially prepared reagent of liver function (bilirubin, SGPT, SGOT, ALP) using Erba bio diagnostic kit.

Collection of sample: -

Under aseptic conditions, from each patient 5ml of blood will be collected which are divided separately in Ethylene Diamine Tetra Acetic Acid (EDTA) bulb for the diagnosis of malaria & Non EDTA bulb for liver function diagnosis.

RESULTS

The present study was conducted in Teerthanker Mahaveer Medical College Hospital and Research Center, Moradabad, U.P. Total 200 clinically suspected cases of Malaria with derange Liver function were taken from February 2014 to January 2015. 160 out of 200 malaria positive cases were found to have deranged Liver function test.

DISCUSSION

Malarial hepatitis is a term commonly used to describe hepatocytic dysfunction in severe and complicated Malaria. Malarial hepatitis is characterized by a rise in serum bilirubin along with the rise in serum glutamate pyruvate transaminase.

In this present study, Malaria positive patient were taken of different age group in which (52.5%) were female and (47.5%) were males. Out of total Malaria positive females, (80.95%) females have got deranged LFT and in total Malaria positive males, (78.9%) have got deranged LFT. Total 80% of malaria positive cases shows mean level of Bilirubin (Total, Direct, Indirect), ALT, AST were 2.52 ± 3.96mg/dl, 0.89 ±1.38 mg/dl, 1.65±2.67mg/dl, 39.05±28.38 IU/L, 53.39±49.00 IU/L, respectively.

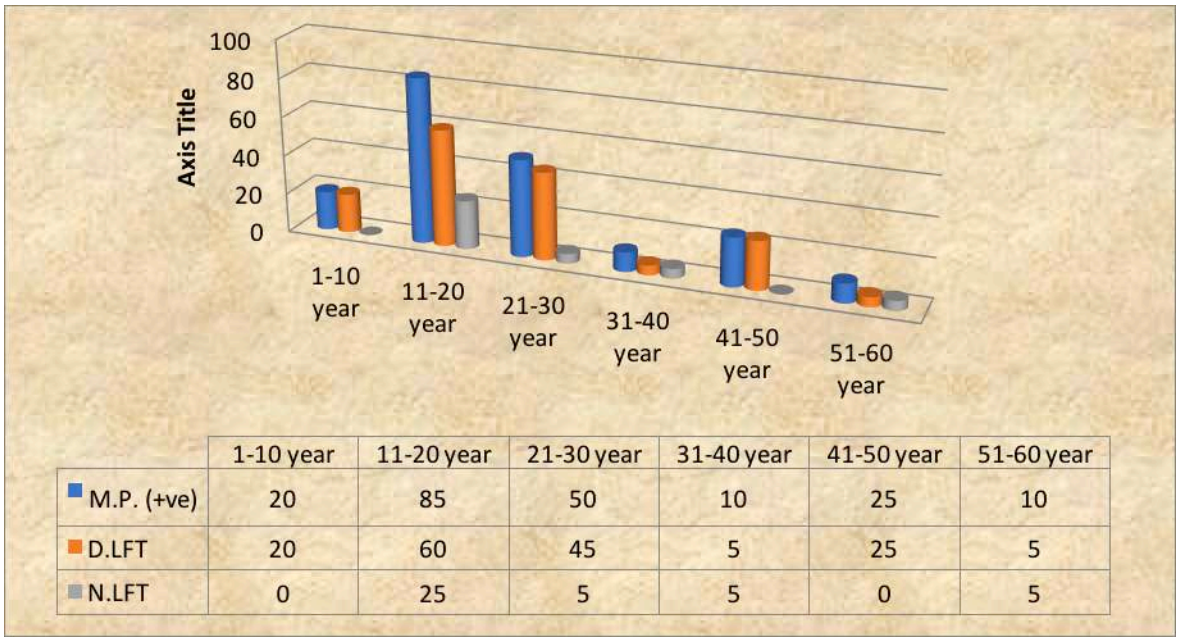
Kochar et al[14] observed that patients of malaria hepatitis had linear increase in AST and ALT levels with increasing bilirubin level. They observed that AST and ALT in patients with S. bilirubin less than 10 mg% were in the range of 163.11±76.79 and 202.98 ± 120 IU/L respectively, whereas patients with S. bilirubin more than 10 mg% had AST and ALT levels in the range of 563.03 ± 303.13 and 669.83 ± 368.08 IU/L respectively.

The study done by U.E. Uzuegbu et, all in 2010 in Anambra State University, Uli, Nigeria, Liver function test marker's assayed in 230 patients, age range: 0-50 years, presenting with acute, Uncomplicated Falciparum Malaria infection and 234 subjects without malaria infection.

In this study, it was observed that the values for liver function profiles among patients with the malaria were elevated when compared with those without infection. Previous studies have documented liver dysfunction in Plasmodium falciparum malaria (Anad et al., 1992; Premaratna et al., 2001).

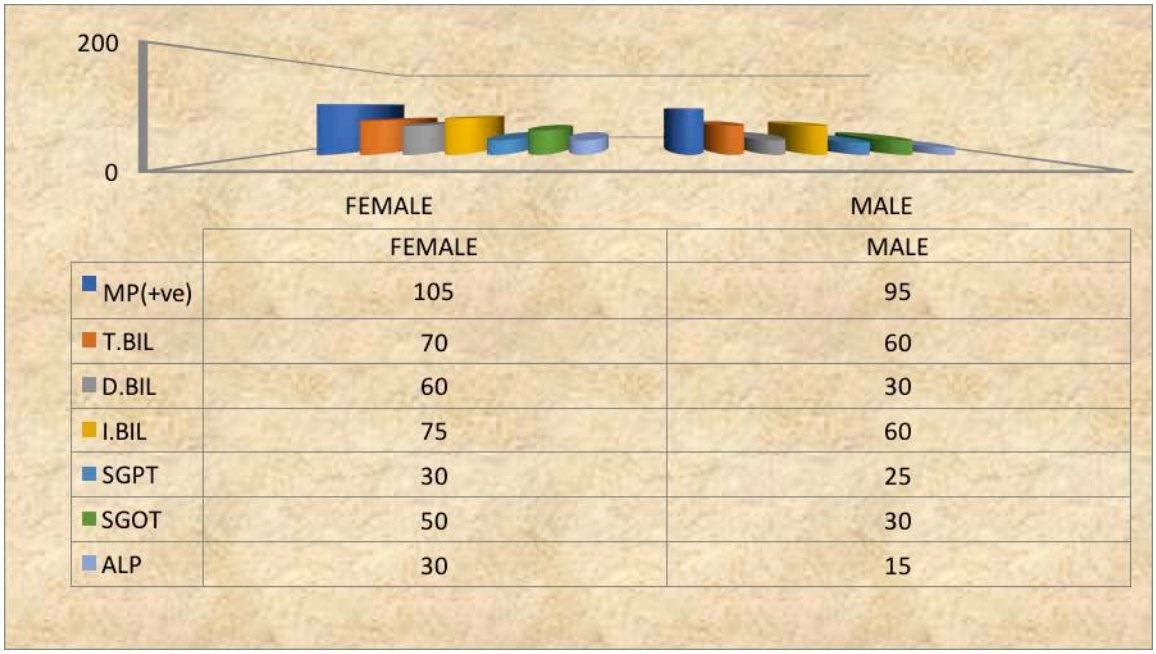
The observed increase ($p<0.005$) in serum liver enzymes (AST, ALT and ALP) could be due to leakage from hepatic cells that were killed or injured by the auto immune progress and/or y abnormal cell activation induced by the parasites. This finding supports previous reports (Guthrow et al., 2007) as judged by the changes in liver function markers, there appears to be a measure of liver dysfunction and compromise in P. falciparum malaria infected patients, which seems to be more severe among male patients irrespective of age .The changes in liver function markers induced by other forms of human malaria parasites (*P. vivax*, *P. malariae* and *P. ovale*) have been observed to be mild and usually reversible after few weeks of anti - malaria treatment (Wilaratna *et al.*, 1994).

The changes induced by *P. falciparum,* the commonest form of malaria infection have been reported to be complicated by Previous studies (Murthy *et al*., 1998) and further confirmed by this research findings. Yet, information on treatment follow - up is scarcely documented in Nigeria. Since the severity of *P. Falciparum* malaria infection on liver is becoming fully established, then documentation on post - treatment periods is desirable to provide the scientific basis for advising health care providers.



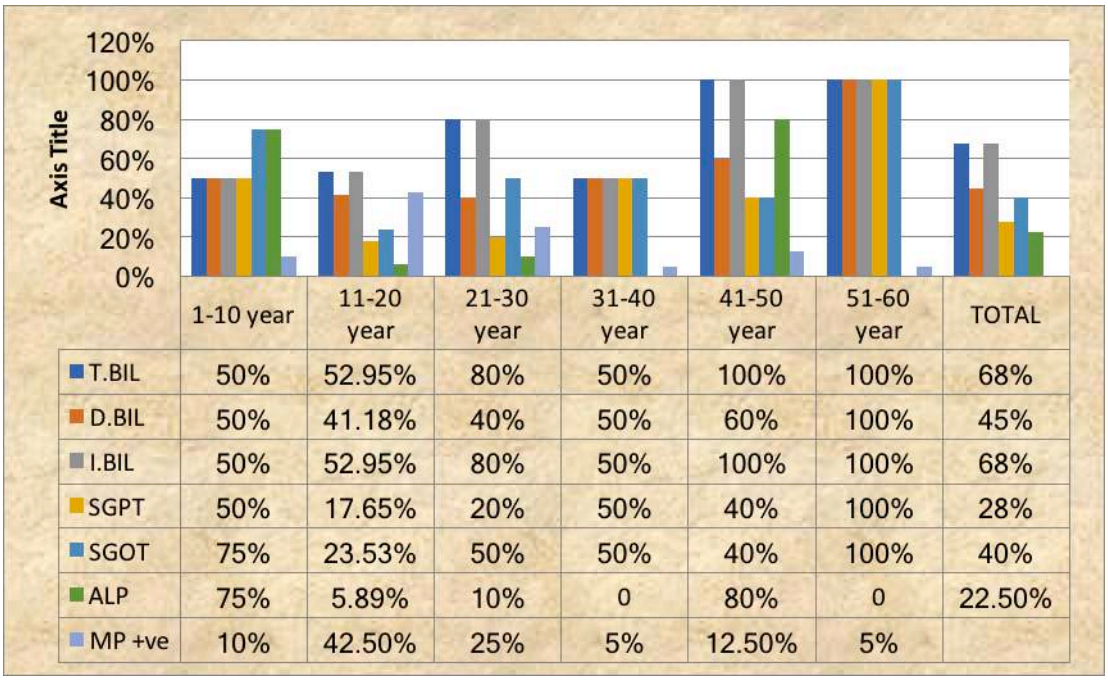
	1-10 year	11-20 year	21-30 year	31-40 year	41-50 year	51-60 year
M.P. (+ve)	20	85	50	10	25	10
D.LFT	20	60	45	5	25	5
N.LFT	0	25	5	5	0	5

Fig. 01. Age wise distribution of deranged & normal liver function test.



	FEMALE	MALE
MP(+ve)	105	95
T.BIL	70	60
D.BIL	60	30
I.BIL	75	60
SGPT	30	25
SGOT	50	30
ALP	30	15

Fig.02. Sex wise distribution of malaria positive cases with individual increased level of LFT markers in both sexes.



	1-10 year	11-20 year	21-30 year	31-40 year	41-50 year	51-60 year	TOTAL
T.BIL	50%	52.95%	80%	50%	100%	100%	68%
D.BIL	50%	41.18%	40%	50%	60%	100%	45%
I.BIL	50%	52.95%	80%	50%	100%	100%	68%
SGPT	50%	17.65%	20%	50%	40%	100%	28%
SGOT	75%	23.53%	50%	50%	40%	100%	40%
ALP	75%	5.89%	10%	0	80%	0	22.50%
MP +ve	10%	42.50%	25%	5%	12.50%	5%	

Fig 03. Age wise distribution of malaria positive cases with increased level individual LFT markers with percentage.

Table 01:- Age wise distribution of all Malaria positive cases with abnormal and normal Liver function test.

Age	Malaria positive	Percentage (n=200)	Deranged liver function	Percentage	Normal liver function	Percentage
1-10	20	10%	20	100% (n=20)	00	00
11-20	85	42.5%	60	70.59% (n=85)	25	29.42% (n=85)
21-30	50	25%	45	90% (n=50)	5	10% (n=50)
31-40	10	5%	5	50% (n=10)	5	50% (n=10)
41-50	25	12.5%	25	100% (n=25)	00	00
51-60	10	5%	5	50% (n=10)	5	50%
>60	00	00	00	00	00	00
TOTAL	200	100%	160	80%	40	20%

Table 02:- Sex wise Distribution of all Malaria positive cases with abnormal & normal liver function test.

Patient	M.P (+ve)	PER.% (N=200)	D.L FT	PER. %	N.L FT	PER. %
Female	105	52.5%	85	80.95% (N=105)	20	19.05% (N=105)
Male	95	47.5%	75	78.9% (N=95)	20	21.1% (N=95)
Total	200	100%	160	80%	40	20%

The study conducted by Rathod chirag, et, al in 2012 on 1093 patient out of 781 slides were positive, remaining 312 were treated on clinical ground. Of 781 cases, 443(56%) P. falciparum,327(42%) P. vivax and 11(2%) were mixed infection. Male to female ratio was 1.8:1 &0.8:1 in P. falciparum & P.vivax, respectively. Fever, Prodroms, GI symptoms, Liver- dysfunction (51% vs 47%),

The study conducted by V. Singh et, al in 2006 on 732 adult patient with falciparum malaria, only 39(5.3%) were noted to have jaundice and only 18 (2.45%) had evidence of malarial hepatitis.[15] In another study by Murthy et al, out of 95 patient admitted with falciparum malaria, 62% had jaundice but only 21% had evidence of malaria hepatitis.

The study conducted by Muhammad Waseem Kausar et al, in 2010 on 81 patients of different ages and both sexes suffering from acute malaria, were selected by convenient sampling. Nine patients, infected by Hepatitis B and C infections were excluded from the study. Among remaining 72 cases, 48 (70%) were suffering from infection by

Plasmodium falciparum and 24 (30%) from infection by Plasmodium vivax infection.

TABLE 03:- The mean value of measured parameters in malaria positive cases.

PARAMETERS	DERANGED LFT MEAN±S.D (N=200)
T.BIL	2.52 ± 3.96 mg/dl
D.BIL	0.89 ± 1.38 mg/dl
I.BIL	1.65 ± 2.67 mg/dl
SGPT	39.05 ± 28.38 IU/L
SGOT	53.39 ± 49.00 IU/L
ALP	392.71 ± 258.72

TABLE 04:- mean value of measured parameters in malaria patients stratified by gender

Parameters	Deranged LFT Mean±s.d	
	Male	Female
T.BIL	1.37 ± 0.76 mg/dl	3.57 ± 5.19 mg/dl
D.BIL	0.48 ± 0.26 mg/dl	1.26 ± 1.80 mg/dl
I.BIL	0.89 ± 1.50 mg/dl	2.34 ± 3.49 mg/dl
SGPT	36.13 ± 24.30 IU/L	41.69 ± 31.30 IU/L
SGOT	52.22 ± 49.24 IU/L	54.45 ± 48.70 IU/L
ALP	362.62±240.21 U/L	378.75±249.12 U/L

Ali Hassan Abro et al, in 2009 study included 105 adult patients who fulfilled the inclusion criteria. On clinical examination, 23% patients were found to be jaundiced. Serum alanine amino transferase (ALT) level was above the reference range in 67.6%, but in only 11.4%, ALT was more than 3 times of normal level. Serum bilirubin was found to be higher than normal level in 81%.

The study conducted by Singh R, Kaur M et al, in 2010 on the age group of patients ranged between16-56 year (mean 28.14±7.23). Serum bilirubin levels ranged from 1 to 32mg %(mean5.65). 41.46% had serum bilirubin of <3mg%, 40.24% had 3-10 mg% and 18.29% had >10 mg%.

Ali Hassan,et al in 2009 conduct study on 105 adult patients who fulfilled the inclusion criteria 23% patients were found to be jaundiced. Serum alanine amino transferase (ALT) level was above the reference range in 67.6%, but in only 11.4%, ALT was more than 3 times of normal level. Serum bilirubin was found to be higher than normal level in 81%, however, only in 23% of the patients, Serum bilirubin was >3mg/dl. Predominantly conjugated hyperbilirubinemia was observed in patients with high ALT.

CONCLUSION

Deranged liver functions are commonly seen as a complication of severe malarial infection; however, histologically severe hepatic inflammation has never been documented.[15] Presence of raised hepatic enzymes with near normal coagulation parameters, in presence of documented malarial infection should suggest presence of malarial hepatopathy. Severe hepatic dysfunction in malaria is usually associated with coexisting viral hepatitis or underlying chronic liver disease. Patients with malarial hepatopathy are more prone to complications; hence it should be promptly recognized. Since patients with malarial hepatopathy have a better outcome than those with other causes of hepatic failure, it should be aggressively treated.[17] Anand AC, Puri P. Jaundice in Malaria. J Gastroenterol Hepatol 2005;20:1322-32.)

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How to cite this article: Bisht R, Mishra M, Mishra S. Study of Malaria Parasite with Special Reference to Liver Function Test of the Patient Attending at TMU Hospital Moradabad (U.P). Int Arch BioMed Clin Res. 2017;3(1):33-37.DOI:10.21276/iabcr.2017.3.1.10

Source of Support: Nil, **Conflict of Interest:** None