Alternation in the Liver Function of Malaria Positive Patients

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ABSTRACT

Introduction: Malaria is a mosquito born disease caused by protozoa belonging to family Plasmodium. According to the WHO, involvement of liver in Plasmodium falciparum is not an uncommon feature and presence of jaundice (bilirubin ≥ 3 mg/dl) is one of the signs of malaria. Malarial hepatitis, complication of malaria is indicated by the increased level of bilirubin along with increased level of SGPT to more than three times of normal level.

Objective: Detection of malaria positive cases and correlates all malaria positive cases with Liver Function Test (LFT)

Material and Methods: The study was performed on 70 peripheral blood smear (PBS) confirmed cases of malaria. Collection of blood sample was done by venipuncture under aseptic conditions in the EDTA vial for the diagnosis of malaria and in the plain vial to perform liver function test. Malaria was diagnosed by the microscopy of PBS and rapid malarial antigen test. The LFT was performed using auto analyzer and Erba diagnostic kit, according to manufacturer instructions.

Results: Out of total 70 malaria positive patients, 44 patients (63%) had got deranged LFT, in which 20 (45%) were male and 24 (54%) were female. Out of 64 cases of Plasmodium vivax, 40 patients had got deranged LFT while out of 6 patients infected with Plasmodium falciparum 4 had got deranged LFT.

Conclusion: Liver dysfunction in malarial infection ranged from mild elevation of liver enzymes and serum bilirubin (≥ 3 mg/dl) to acute hepatitis. It indicates severe illness with high frequency of complications and mortality rates.

Key words: Liver function test, Malaria, Liver hepatitis

INTRODUCTION

Malaria is a mosquito born parasitic disease caused by parasitic protozoan which belongs to the family Plasmodium. Female anopheles mosquito is responsible for spreading malaria among human. After the mosquito bites parasites are transferred from its saliva into the blood of person [1]. Five species of Plasmodium are responsible for causing malaria in human, these are Plasmodium vivax, Plasmodium falciparum, Plasmodium ovale, Plasmodium malariae and Plasmodium knowlesi [2].

According to World Health Organization (WHO), approximately 270 million people suffer from malaria every year all over the world, with 1-2 million death annually, out of which 80% deaths are caused by Plasmodium falciparum [3]. WHO reported that presence of jaundice (≥3mg/dl) and hepatic involvement in P. falciparum malaria is not rare presentation [4]. World malaria report of 2010 showed that Plasmodium vivax is a major health problem among world which involves billions of population at the risk of infection [5].

It is evaluated that more than 500 million individuals are infected with Plasmodium species and most of whom are children who die from infection. Malaria is endemic in more than 85 countries which represents 38% of total population of world [6].

Out of 5 species that infect humans, Plasmodium vivax and Plasmodium falciparum cause 95% of infections. Plasmodium vivax may be responsible for 80% of infection because of wide distribution in world like tropics, subtropics and temperate zones. [6].

The classical symptom of malaria is convulsion which occurs in periodical occurrence of sudden coldness followed by quivering, fever and then sweating. When it occur in every two days then known as tertian fever, which occur in case of in P.
vivax and P. ovale infection. While in case of P. malariae and P. falciparum infection this cycle is almost continuous fever [7].

Falciparum malaria is one of the most important parasitic diseases of human affecting more than one billion people and is also responsible for 1-3 million deaths per year [8-10]. Falciparum malaria can mimic many diseases with its presentation and it must be considered in the diagnosis of acute illness like hepatorenal syndrome, fulminant hepatic failure, acute hepatitis, jaundice, encephalopathy, pulmonary edema, anemia, sepsis, hypoglycemia, acidosis and abdominal pain with diarrhea, hepatosplenomegaly, renal failure, spontaneous bleeding and coagulopathy, hyperpyrexia and unarousable coma [11-14].

24 to 48 merozoites are released from each merozoite in the circulation by the process of rupture of RBC with 48 to 72 hours and produce febrile paroxysm. In this way, malarial parasites are responsible for liver cell damage, jaundice with or without increased liver enzymes like Aspartate and Aminotransferases [15].

Alanine aminotransferase (SGPT) is mainly present in the liver and acts as catalyst in the transfer of amino acid from donor to recipient molecule, while aspartate transaminase (SGOT) is found in the muscle, brain and kidney in addition to the liver. Hence SGPT, not SGOT is the prime indicator of liver cell damage. Alkaline phosphatase (ALP) is secreted by the hepatocytes in the biliary canaliculi. So raised serum ALP in the patients affected by falciparum malaria, due to perturbation of host hepatocytes drainage pathway during the hepatic stage of falciparum, also leakage from damaged membrane of liver hepatocytes [16].

MATERIAL AND METHODS

This prospective study was done in the Department of Microbiology, Teerthanker Mahaveer Medical College & Research Centre, Moradabad, U.P. over a time period from March 2016 to January 2017.

SAMPLE SIZE

Total 70 malaria positive cases (confirmed by Microscopy & Antigen card test) with liver function tests were included in the study. Patients having other causes of liver disease and patients who were taking anti-malarial drugs were excluded from the study.

COLLECTION OF THE SAMPLE

5ml of blood was collected from each patient under aseptic conditions by venipuncture in Ethylene Diamine tetra Acetic Acid (EDTA) vacutainer tube (2.5ml) for the diagnosis of malaria and 3 ml in plain tube for Liver Function Test.

METHODOLOGY

Diagnosis of malaria was done by the microscopy of peripheral blood smear. It remains gold standard for the confirmation of diagnosis of malaria. Thick and Thin blood smears were prepared and both are stained with Leishman’s stain, then smears were examined under the microscope by oil immersion lens at 100x for the different stages of malaria.

Diagnosis of liver function test was done by the autoanalyzer EM200 of Transasia Bio-Medicals Ltd, by the use of commercially prepared reagent of Total bilirubin, SGOT, SGPT, ALP using Erba biodiagnostic kit according to the manufacturer’s instructions.

RESULTS

Present study was done to observe the impact of malaria on liver function. In our study total 4 liver biochemical parameters were included, bilirubin, SGOT, SGPT, and ALP. In our study total 70 patients were taken which were diagnosed as malaria positive patients either by the microscopy of peripheral blood smear or rapid antigen card test. Out of 70 patients, 44 (62.86%) patients were male and 26 (37.14%) patients were female (Table 1 figure 1).

<table>
<thead>
<tr>
<th>SEX</th>
<th>NO. OF MALARIA POSITIVE PATIENTS (N=70)</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>MALE</td>
<td>44</td>
<td>62.86</td>
</tr>
<tr>
<td>FEMALE</td>
<td>26</td>
<td>37.14</td>
</tr>
<tr>
<td>TOTAL</td>
<td>70</td>
<td>100</td>
</tr>
</tbody>
</table>

Table 1: Sex wise distribution of total malaria positive patients

Figure 1: Sex wise distribution of total malaria positive patients
Out of 70 malaria positive cases, 64 (91.43%) were infected with P. vivax and 6 (8.57%) were with P. falciparum. Out of 64 cases of P. vivax, 40 (62.5%) had got deranged LFT while out of 6 cases of P. falciparum 4 patients (66.67%) had got deranged LFT. Out of 64 cases of P. vivax 40 (62.5%) cases were having deranged LFT while out of 6 cases of P. falciparum 4 (66.67%) cases were having deranged LFT (table 2 Figure 2). In our study majority of malaria positive patients were found in the age group of 21-30 year (34%) cases followed by the age group of 11-20 year (Table 3 Figure 3).

### Table 2: Species wise distribution with deranged and normal LFT of Total malaria positive cases

<table>
<thead>
<tr>
<th>Type</th>
<th>MP +ve</th>
<th>Deranged LFT %</th>
<th>Normal LFT %</th>
</tr>
</thead>
<tbody>
<tr>
<td>P. Vivax</td>
<td>64</td>
<td>40</td>
<td>62.5</td>
</tr>
<tr>
<td>P. falcip</td>
<td>6</td>
<td>4</td>
<td>66.67</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>44</td>
<td>62.86</td>
</tr>
</tbody>
</table>

Table 2: Species wise distribution with deranged and normal LFT of Total malaria positive cases

### Table 3: Age wise and species wise distribution of all malaria positive cases

<table>
<thead>
<tr>
<th>Age</th>
<th>MP +ve</th>
<th>% N=70</th>
<th>P. vivax</th>
<th>%</th>
<th>P. falcip</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>01-10</td>
<td>5</td>
<td>7.14</td>
<td>5</td>
<td>100</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>11-20</td>
<td>17</td>
<td>24.28</td>
<td>16</td>
<td>94.11</td>
<td>1</td>
<td>5.89</td>
</tr>
<tr>
<td>21-30</td>
<td>24</td>
<td>34.28</td>
<td>21</td>
<td>87.50</td>
<td>3</td>
<td>12.5</td>
</tr>
<tr>
<td>31-40</td>
<td>11</td>
<td>15.71</td>
<td>10</td>
<td>90.91</td>
<td>1</td>
<td>9.0</td>
</tr>
<tr>
<td>41-50</td>
<td>5</td>
<td>7.14</td>
<td>5</td>
<td>100</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>51-60</td>
<td>4</td>
<td>5.71</td>
<td>3</td>
<td>75</td>
<td>1</td>
<td>25</td>
</tr>
<tr>
<td>&gt;60</td>
<td>4</td>
<td>5.71</td>
<td>4</td>
<td>100</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100</td>
<td>64</td>
<td>91.43</td>
<td>6</td>
<td>8.57</td>
</tr>
</tbody>
</table>

Table 3: Age wise and species wise distribution of all malaria positive cases

In our study of 64 cases of P. vivax, total bilirubin was deranged in 26 (40.63%) cases, SGOT in 10 (5.63%) cases, SGPT in 9 (14.06%) cases and ALP in 19 (29.7%) cases while out of 6 P. falciparum total bilirubin, SGOT & SGPT was deranged in 4 cases and ALP in 2 (33.3%) cases (Table 4 Figure 4).

### Table 4: Species wise distribution with deranged level of LFT markers of total malaria positive cases

<table>
<thead>
<tr>
<th>LFT Parameters</th>
<th>P. vivax</th>
<th>P. falcip</th>
<th>TOTAL(70)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
</tr>
<tr>
<td>Total bilirubin</td>
<td>26</td>
<td>40.63</td>
<td>4</td>
</tr>
<tr>
<td>SGOT</td>
<td>10</td>
<td>15.63</td>
<td>4</td>
</tr>
<tr>
<td>SGPT</td>
<td>9</td>
<td>14.06</td>
<td>4</td>
</tr>
<tr>
<td>ALP</td>
<td>19</td>
<td>29.69</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 4: Species wise distribution with deranged level of LFT markers of total malaria positive cases

Figure 2: Species wise distribution with deranged and normal LFT of Total malaria positive cases

Figure 3: Age wise and species wise distribution of all malaria positive cases

Figure 4: Species wise distribution with deranged level of LFT markers of total malaria positive cases
DISCUSSION

Malaria is a major health problem responsible for causing 270 million people and 1-2 million deaths per year. Malaria involves liver where hepatocytes are invaded by sporozoites and multiply. Molyneux et al suggest that hemolysis and involvement of liver with the elevation of hepatic enzymes with the jaundice is the most common feature of malaria.

In our study 64 (91.42%) cases were of P. vivax & only 6 (8.57%) cases were of P. falciparum while Rajendra Kumar et al (2016) observed that out of 387 cases of malaria, 297 (76.74%) were of P. vivax and 54 (13.95%) of P. falciparum.

In this study the major age group for malarial infection was 21-30 year (34.28%) which is the most prolific age group, followed by the age group of 11-20 (24.28%), this finding was similar to the study of Rajendra Kumar Verma et al (2016).

In case of falciparum malaria, jaundice has been found more common as compared to vivax malaria. In our study 66.67% cases of P. falciparum were infected with jaundice which resemble with the study of Hazra et al from Calcutta who found an association of jaundice in 40% and 9.09% cases with P. falciparum and P. vivax respectively.

CONCLUSION

Liver dysfunction in malarial infection ranged from mild elevation of liver enzymes and serum bilirubin (≥ 3 mg/dl) to acute hepatitis. It indicates severe illness with high frequency of complications and mortality rates.

REFERENCES