A Study of status of liver enzymes in malaria

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Abstract

Mildly elevated liver enzymes have been reported in dengue infection. Malaria destroys the liver tissue and the enzymes are elevated in blood that is absorbed partially or fully in the blood stream. The liver enzymes in malaria are elevated in early malaria infection and it’s a rule and natural history of the disease. The enzymes can be used as a predictor for assessing the disease severity and higher the levels of liver enzymes poorer is the prognosis of the disease. Most of the studies showed that unlike other viral infections, in dengue the rise of SGOT is usually more than SGPT and is believed to be due to release from the damaged myocytes. In view of this biochemical pattern, it is possible to confuse liver involvement in malaria infection with typical acute viral hepatitis, especially in countries where outbreaks of hepatitis A and E are common. So an attempt has been made to study the hepatic enzyme values and whether it reflects the prognosis is checked in this study.

Keywords: LFT, SGPT, SGOT, Malaria, Infection

Introduction

Surveys of falciparum malaria have reported lower rates of jaundice at presentation in endemic populations (2.6–5.3% of cases) compared with epidemic malaria (11.5–62% of cases) [1-7]. Other Plasmodium species have been associated with a relatively lower rate of jaundice compared with falciparum [8, 9]. Mild elevations of transaminases are common, although more significant elevations associated with multiorgan dysfunction have also been reported [10-14]. The syndrome of “malarial hepatopathy” has been recently proposed, being defined as a bilirubin level >2.5 times upper limit of normal (ULN) with associated transaminase elevation >3×ULN (where ALT is considered the more liver-specific enzyme) [1, 2, 15-19]. Although the clinical significance of malarial hepatopathy has not been fully elucidated, it represents an attempt to further describe malaria-associated liver injury and has been associated with more severe disease and other organ dysfunction [15]. The relationship of malarial hepatopathy to antimalarial treatment needs to be evaluated to differentiate it from clinically significant drug-induced liver injury meeting Hay's Law, which has a similar definition [20]. Malarial hepatopathy has been reported in 2.6–45% of all malaria cases and up to 87.5% of cases presenting with clinical jaundice [1, 2, 4, 13, 18, 21, 22]. Kupffer cell hyperplasia, hemozoin loading, and monocyte infiltration are the most frequently reported histological findings in malaria-associated liver injury; all seem to resolve after antimalarial treatment [1, 2, 12, 23-27]. So an attempt has been made to study the hepatic enzyme values and whether it reflects the prognosis is checked in this study.

Aims and Objectives: Hepatic Enzyme as a prognostic evaluator in malaria.

Materials and methods

Methodology: The present study was conducted in the Department of Medicine, in Srinivas Institute of Medical Sciences, Mangalore.

45 patients were chosen for the study.

The study was done in 45 patients who were admitted with Dengue Positive.

Inclusion Criteria

1. Cases confirmed peripheral smear.
2. Cases with elevated liver enzymes.
Inclusion Criteria
1. Cases confirmed peripheral smear
2. Cases with elevated liver enzymes.

Exclusion Criteria
1. Alcoholics.
2. Patients on Hepatotoxic drugs.

All the statistical analysis is done using the ANNOVA.

Results

Table 1: Age

<table>
<thead>
<tr>
<th>Total</th>
<th>Mean Age</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>45</td>
<td>41.39 years</td>
<td>± 5.39 years</td>
</tr>
</tbody>
</table>

Table 2: Sex Distribution

<table>
<thead>
<tr>
<th>Total</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>45</td>
<td>31</td>
<td>14</td>
</tr>
</tbody>
</table>

Graph 1: Enzyme

Table 5: Significance of rise in enzymes

<table>
<thead>
<tr>
<th>Value of SGOT</th>
<th>X-Value</th>
<th>Significance</th>
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<tbody>
<tr>
<td>375.29</td>
<td>9.38</td>
<td>0.0018</td>
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Discussion

Peaks in transaminase levels after treatment have also been recently reported; these could represent a distinct type of malaria-associated liver injury [28-31]. In a small retrospective comparative European study, asymptomatic transaminase elevation was observed in returned travellers hospitalized for malaria after treatment with the Artemisinin-based combination therapy arteether–lumefantrine. The authors postulated that the rapid parasiticidal effect of the antimalarial treatment may promote accelerated hepatic heme loading and oxidative stress [28]. Liver injury through this proposed mechanism is not exactly an adverse drug reaction but rather may be influenced by factors, such as parasite burden, rate of clearance, and host heme metabolism. In prospective trials using the induced blood stage malaria (IBSM) model in healthy malaria-naïve participants, asymptomatic moderate (2.5–5.0×ULN) to more rarely severe (>5.1×ULN) elevations of transaminase enzymes have been reported in a subset of participants. These isolated transaminase elevations are associated with an ALT/AST ratio >1 and a normal bilirubin level, peaking 4–12 days after treatment, and have been reported following multiple antimalarial compounds in both falciparum and vivax malaria models [29, 30]. Similar elevations have been observed after sporozoite malaria challenge [31, 32]. Delayed transaminase elevation may be attributed to the antimalarial compound, the use of acetaminophen for symptom relief [33] the malaria parasite, or host factors associated with parasite clearance. Although the relative contribution of each of these factors is uncertain, it is important to understand their relative contribution, given the implications that unexplained transaminase elevation may have on antimalarial drug development.

Conclusion

Both SGOT and SGPT are significantly elevated in severe dengue cases than SGPT. But the increase of SGOT is significantly higher.

References