A study of hepatic profile in dengue

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Abstract
Dengue fever is a painful, debilitating mosquito-borne disease caused by any one of four closely related dengue viruses. These viruses are related to the viruses that cause West Nile infection and yellow fever. An estimated 400 million dengue infections occur worldwide each year, with about 96 million resulting in illness. Most cases occur in tropical areas of the world, with the greatest risk occurring in our own country. Clinically Liver is often enlarged and tender. There are many articles which has reported the involvement of liver in this disease. The changes can be noted both clinically and also biochemically in which the enzymes are quoted elevated. These features occur in both severe and non-severe dengue cases. Therefore, monitoring for warning signs and other clinical parameters is crucial for recognising progression to critical phase. This study puts in an effort to find the hepatic profile of the patients both clinically and biochemically so as to be useful to the practising physicians.

Keywords: SGOT, SGPT, liver function, dengue

Introduction
An estimated 50 million dengue infections occur annually caused by four distinct subgroups of dengue viruses, types 1, 2, 3 and 4 (DEN 1-4) which are RNA viruses. The genome of DEN virus encodes different gene products: C (capsid), prM (matrix), B (envelope) and seven non. Structural (N-S) proteins. NS1 protein is secreted in plasma and is useful in early diagnosis. Dengue infection of humans occurs from bites of Aedes aegypti mosquitoes. The mosquito feeds during the day and has a propensity for man-made habitats containing water. Dengue viral infection can present as three broad clinical patterns: Classic dengue, Haemorrhagic fever and undifferentiated fever. Clinically Liver is often enlarged and tender. Mildly elevated liver enzymes have been reported in dengue infection [1, 2, 3]. The enzymes can be used as a predictor for assessing the disease severity [4, 5]. In view of this biochemical pattern, it is possible to confuse liver involvement in dengue infection with typical acute viral hepatitis [6, 7]. The presence of thrombocytopenia and persistence of fever with elevated hepatic enzymes should help [7, 8]. This study puts in an effort to find the hepatic profile of the patients both clinically and biochemically so as to be useful to the practising physicians.

Aims and Objectives
To study the hepatic enzyme profile in dengue.

Materials and Methods
Methodology
The present study was conducted in the Department of General Medicine, Azeezia Institute of Medical Sciences and research, Kollam, Kerala 120 patients were chosen for the study who were confirmed cases of Dengue. The study was done from July 2017 to March 2019.

Inclusion criteria
1. Cases confirmed with Dengue with Antigen antibody reaction test with specificity of more than 90.
2. Cases with confirmed clinical hepatomegaly, tender hepatomegaly and elevated liver enzymes.
Exclusion criteria
1. Alcoholics and other known hepatocellular disease.
2. Patients on Hepatotoxic drugs, corticosteroid and other immunosuppressant therapy.

All the statistical analysis is done using the ANNOVA. SPSS California 2016.

Results

Table 1: Age

<table>
<thead>
<tr>
<th>Total</th>
<th>Mean Age</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>120</td>
<td>34.82 years</td>
<td>± 08.74 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>120</td>
<td>58</td>
<td>62</td>
</tr>
</tbody>
</table>

Table 3: Spectrum of Dengue related to Hepatic Dysfunction

<table>
<thead>
<tr>
<th>Spectrum</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dengue without Hepatic Dysfunction</td>
<td>81</td>
</tr>
<tr>
<td>Dengue with Hepatic Dysfunction</td>
<td>39</td>
</tr>
</tbody>
</table>

Table 4: Clinical Signs and symptoms related to Hepatic Dysfunction

<table>
<thead>
<tr>
<th>Spectrum</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Tender Hepatomegaly</td>
<td>44</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>31</td>
</tr>
<tr>
<td>Frank Jaundice</td>
<td>06</td>
</tr>
</tbody>
</table>

Table 4: Enzyme

<table>
<thead>
<tr>
<th>Spectrum</th>
<th>SGOT (Mean)</th>
<th>SGPT (Mean)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dengue without Hepatic signs</td>
<td>81.16</td>
<td>42.56</td>
</tr>
<tr>
<td>Dengue with Hepatic Signs</td>
<td>313.76</td>
<td>122.12</td>
</tr>
</tbody>
</table>

Table 5: Significance of rise in enzymes

<table>
<thead>
<tr>
<th>Value of SGOT</th>
<th>X-Value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>313.76</td>
<td>7.1</td>
<td>0.00062</td>
</tr>
</tbody>
</table>

Discussion
Dengue fever is caused by a flavivirus, is also known as breakbone fever (because it causes severe muscle and joint pains), is a mosquito-borne infection characterized by fever, muscle and joint pains, lymphadenopathy, and rash. The name ‘dengue’ is derived from Swahili ki denga pepo, meaning ‘a sudden seizure by a demon’. Dengue Virus is widely distributed in tropics and subtropics. Four types of dengue virus (DEN) exist—DEN 1, DEN 2, DEN 3 and DEN 4. Recovery from infection by one type does not provide complete immunity against infection by other types. Transmitted to man by Aedes aegypti mosquitoes. Humans
and monkeys are reservoir hosts. In humans, clinical disease begins 2-5 days after an infective mosquito bite. Dengue fever presents typically as a fever of sudden onset with headache, chills, malaise, retrobulbar pain, conjunctivitis, pain in back and limbs (break bone fever), lymphadenopathy, maculopapular rash. Fever typically begins on the 3rd day and lasts for 5-7 days and is typically biphasic (saddle back), coinciding with absence of virus in blood, followed by recovery. Dengue may also occur in more serious forms, with haemorrhagic manifestations (dengue haemorrhagic fever) or with shock (dengue shock syndrome characterized by shock and haemocoagulation).

Pathogenesis of these severe syndromes involves pre-existing dengue antibody. It is postulated that virus-antibody complexes are formed within a few days of second dengue infection and non-neutralizing antibodies promote infection of higher numbers of mononuclear cells, followed by the release of vasoactive mediators and procoagulants, leading to disseminated intravascular coagulation seen in haemorrhagic fever. Control of dengue is by vector control. No vaccine is available.

Laboratory diagnosis of Arboviral Infections: Specimens of Blood, CSF, brain tissue inoculated into suckling mice intracerebrally. Animals develop fatal encephalitis; tissue cultures such as chick embryo fibroblast or vero or HeLa cell lines; yolk sac of embryonated eggs. Isolate is identified by hemagglutination and IF. Serodiagnosis is by demonstration of a rise in titre of antibodies in patient’s serum by HI, CFT, IF, ELISA, immunodiffusion and neutralization tests are suggestive of infection. Molecular methods such as RT-PCR can be used to detect viral RNA from blood or other samples.

Liver is often enlarged and tender. There are many articles which has reported the involvement of liver in this disease. The changes can be noted both clinically and also biochemically in which the enzymes are quoted elevated. These features occur in both severe and non-severe dengue cases. Therefore, monitoring for warning signs and other clinical parameters is crucial for recognising progression to critical phase.

**Conclusion**
The liver enzymes are deranged in this disease. The values of which can both be taken as a point to check the prognosis and also effectiveness for the treatment of the disease.

**References**