INTRODUCTION:
Dengue fever is caused by a flavivirus, also known as breakbone fever (because it causes severe 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, chillis, 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 haemoconcentration). 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.

Clinical, Hepatic, Profile, Biochemical.

A STUDY OF HEPATIC PROFILE IN DENGUE

ABSTRACT
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), E (matrix), B (envelope) and seven non-structural (NS1) 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. 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
Clinical, Hepatic, Profile, Biochemical.

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>53.81 years</td>
<td>± 11.28 years</td>
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</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>76</td>
<td>44</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>78</td>
</tr>
<tr>
<td>Dengue with Hepatic Dysfunction</td>
<td>42</td>
</tr>
</tbody>
</table>

Table 4: Clinical Signs and symptoms related to Hepatic Dysfunction
<table>
<thead>
<tr>
<th>Spectrum</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tender Hepatomegaly</td>
<td>21</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>19</td>
</tr>
<tr>
<td>Frank Jaundice</td>
<td>02</td>
</tr>
</tbody>
</table>
permeability will improve, while those with increased capillary
precedes plasma leakage. Patients Without an increase in capillary
leukopenia followed by a rapid decrease in platelet count usually
significant plasma leakage usually lasts for 24-48 hours. Progressive
beginning reflects severity of plasma leakage. The period of clinically
with increasing haematocrit of critical phase. The degree of increase
effervescence of fever, an increase in capillary permeability along
membrane bleeding may be seen. Earliest laboratory abnormality is a
present. Mild haemorrhagic manifestations like petechiae and mucosal
Tenderness upon pressure on eyeball. A positive tourniquet test may be
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disappear; this remission lasts for 2 days and is followed by return of
decrease to nearly normal after 3-4 days and other symptoms
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flushing, skin erythema, generalised body ache, myalgia, arthralgia,
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**DISCUSSION:**
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.

Pathogenesis of Severe Dengue occurs in persons who were infected with one serotype of dengue virus previously and therefore have antibodies against that particular serotype. A second infection by a different serotype causes immunologic enhancement of antibody acquired from a previous infection. Antibody-virus complex taken up by macrophages. Production of vascular permeability factors by macrophages. These vascular permeability factors induce plasma leakage, resulting in DHF and ultimately, DSS. Clinical features are as follows, after the incubation period of 5-8 days, the illness begins abruptly and is followed by the three phases-febrile, critical and recovery. Febrile Phase in patients typically develop high-grade fever suddenly that usually lasts for 2-7 days. Often accompanied by facial flushing, skin erythema, generalised body ache, myalgia, arthralgia, severe back ache (“breakbone” fever), retro-orbital pain and headache.

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