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A STUDY OF HEPATIC PROFILE IN DENGUE

General Medicine

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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), prM (matrix), B (envelope) and seven non. structural (N - S) proteins. NSl 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.

INTRODUCTION:

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 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.

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]. 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.

Aims and Objectives:

To study the hepatic profile in dengue.

MATERIALS AND METHODS:

METHODOLOGY

The present study was conducted in the Department of General Medicine, at Shree Siddhartha medical College agalakote, Tumkur .

120 patients were chosen for the study who were confirmed cases of Dengue.

The study was done from at August 2014 to April 2015.

Inclusion Criteria:

1. Cases confirmed with Dengue with Antigen antibody reaction test with specificity of more than 90.
2. Cases with 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.
3. Previous Dengue infections who have reinfection.

All the statistical analysis is done using the ANNOVA.

RESULTS:

Table 1: Age

Total	Mean Age	SD
120	53.81 years	± 11.28 years

Table 2: Sex Distribution

Total	Male	Female
120	76	44

Table 3: Spectrum of Dengue related to Hepatic Disfunction

Spectrum	Frequency
Dengue without Hepatic Disfunction	78
Dengue with Hepatic Disfunction	42

Table 4: Clinical Signs and symptoms related to Hepatic Disfunction

Spectrum	Frequency
Tender Hepatomegaly	21
Hepatomegaly	19
Frank Jaundice	02

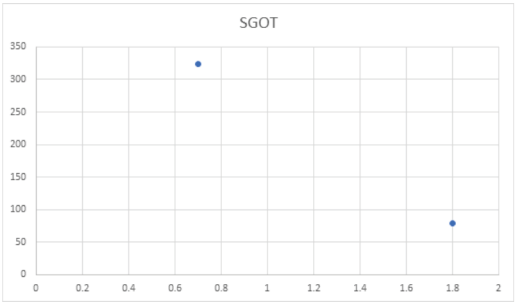
Table 4: Enzyme

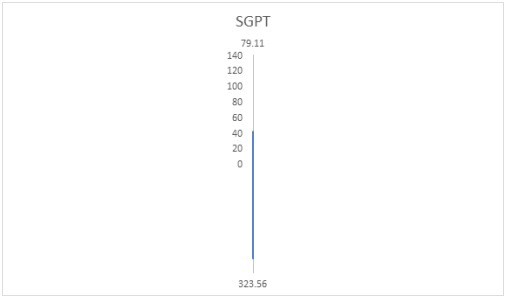
Spectrum	SGOT (Mean)	SGPT (Mean)
Dengue without Hepatic signs	79.11	41.87
Dengue with Hepatic Signs	323.56	120.81

Table 5: Significance of rise in enzymes

Value of SGOT	X-Value	Significance
323.56	6.4	0.00054

Graph 1: Enzymes





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. Sore throat, injected pharynx and conjunctival injection in some patients. Anorexia, nausea and vomiting associated with High fever may cause febrile seizures in children. In some cases, temperature may decrease to nearly normal after 3-4 days and other symptoms disappear; this remission lasts for 2 days and is followed by return of fever and other symptoms. This is known as "saddleback fever". Tenderness upon pressure on eyeball. A positive tourniquet test may be present. Mild haemorrhagic manifestations like petechiae and mucosal membrane bleeding may be seen. Earliest laboratory abnormality is a progressive decrease in total white cell count, which should alert the physician to a high probability of dengue. Critical Phase and the time of effervescence of fever, an increase in capillary permeability along with increasing haematocrit of critical phase. The degree of increase above the baseline haematocrit often levels may occur. This marks beginning reflects severity of plasma leakage. The period of clinically significant plasma leakage usually lasts for 24-48 hours. Progressive leukopenia followed by a rapid decrease in platelet count usually precedes plasma leakage. Patients Without an increase in capillary permeability will improve, while those with increased capillary permeability may become worse as a result of lost plasma volume. Pleural effusion and ascites may be detectable by ultrasound. Shock occurs when a critical volume of plasma is lost through leakage. It is often preceded by warning signs.

Body temperature may be subnormal when shock occurs. With prolonged shock, organ hypoperfusion results in progressive organ impairment, metabolic acidosis and DIC. This in turn leads to severe haemorrhage causing the haematocrit to decrease in severe shock. Instead of leucopenia, usually seen during this phase of dengue, leucocytosis may occur in patients with severe bleeding. Severe organ impairment such as severe hepatitis, encephalitis, myocarditis and severe bleeding may also develop Without obvious plasma leakage or shock. Cases of dengue with warning signs usually recover with early rehydration. However, some cases will deteriorate to severe dengue. Recovery Phase If the patient survives the 24-48 hours critical phase, a gradual reabsorption of extravascular fluid takes place in the following 48-72 hours. General well-being improves appetite returns, gastrointestinal symptoms abate, haemodynamic status stabilises and diuresis ensues. Some patients may have a rash of "isles of white in the sea of red". Some patients may experience pruritus, particularly on hands and feet. Bradycardia and ECG changes are common during this stage. The haematocrit stabilises or may be lower due to dilutional effect of reabsorbed fluid. White blood cell count usually starts to rise soon after effervescence but recovery of platelet count is typically later than that of white blood cell count. Respiratory distress from massive pleural effusion and ascites will occur at any time if excessive intravenous fluids have been administered. Severe Dengue is defined by one or more of the following:

1. Plasma leakage that may lead to shock (dengue shock) and/or fluid accumulation, with or without respiratory distress
2. Severe bleeding.
3. Severe organ impairment.

Increasing vascular permeability results in shock. It usually takes place around defervescence, usually on day 4 or 5 (range days 3-7) of illness, preceded by warning signs.

During the initial stage of shock, compensatory mechanisms produce tachycardia and peripheral vasoconstriction with reduced skin perfusion, resulting in cold extremities and delayed capillary refill time. Diastolic pressure rises towards the systolic pressure and the pulse pressure narrows (20 mmHg) as the peripheral vascular resistance increases. Patients in dengue shock often remain conscious and lucid. Later, patient decompensates and both systolic and diastolic pressures disappear abruptly. Prolonged hypotensive shock, may lead to multiorgan failure. Hypotension is usually associated with major bleeding as shock in combination with thrombocytopenia, hypoxia and acidosis which can lead to DIC. Unusual manifestations include acute liver failure, encephalopathy and cardiomyopathy. Most deaths from dengue occur in patients with profound shock, particularly if it is complicated by fluid overload.

CONCLUSION:

The liver is definitely involved in majority of the times in dengue and also has been reported in our study as has been in other studies. But for the first time an effort has been made to study both clinical and biochemical patterns of the disease. The study has a future scope of continuation where an effort can be made to find the relation between the clinical and the biochemical enzyme levels. Liver involvement is more common in dengue hemorhagic fever and dengue shock syndrome.

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