International Journal of Advanced Research in Medicine

E-ISSN: 2706-9575
P-ISSN: 2706-9567
IJARM 2020; 2(2): 124-126
Received: 28-05-2020
Accepted: 29-06-2020

Dr. Mohammed Mirvaz Zulfikar
Assistant Professor, Department of Medicine, Azeezia Institute of Medical Sciences and research, Kollam, Kerala, India

Dr. Sheeba Mariyam
Junior Resident, Azeezia Institute of Medical Sciences and research, Kollam, Kerala, India

Corresponding Author:
Dr. Sheeba Mariyam
Junior Resident, Azeezia Institute of Medical Sciences and research, Kollam, Kerala, India

A study of hepatic profile in dengue

Dr. Mohammed Mirvaz Zulfikar and Dr. Sheeba Mariyam

DOI: https://doi.org/10.22271/27069567.2020.v2.i2b.59

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. 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. 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

Total	Mean Age	SD
120	34.82 years	± 08.74 years

Table 2: Sex Distribution

Total	Male	Female
120	58	62

Table 3: Spectrum of Dengue related to Hepatic Disfunction

Spectrum	Frequency
Dengue without Hepatic Disfunction	81
Dengue with Hepatic Disfunction	39

Table 4: Clinical Signs and symptoms related to Hepatic Disfunction

Tender Hepatomegaly	44
Hepatomegaly	31
Frank Jaundice	06

Table 4: Enzyme

Spectrum	SGOT (Mean)	SGPT (Mean)
Dengue without Hepatic signs	81.16	42.56
Dengue with Hepatic Signs	313.76	122.12

Table 5: Significance of rise in enzymes

Value of SGOT	X-Value	Significance
313.76	7.1	0.00062

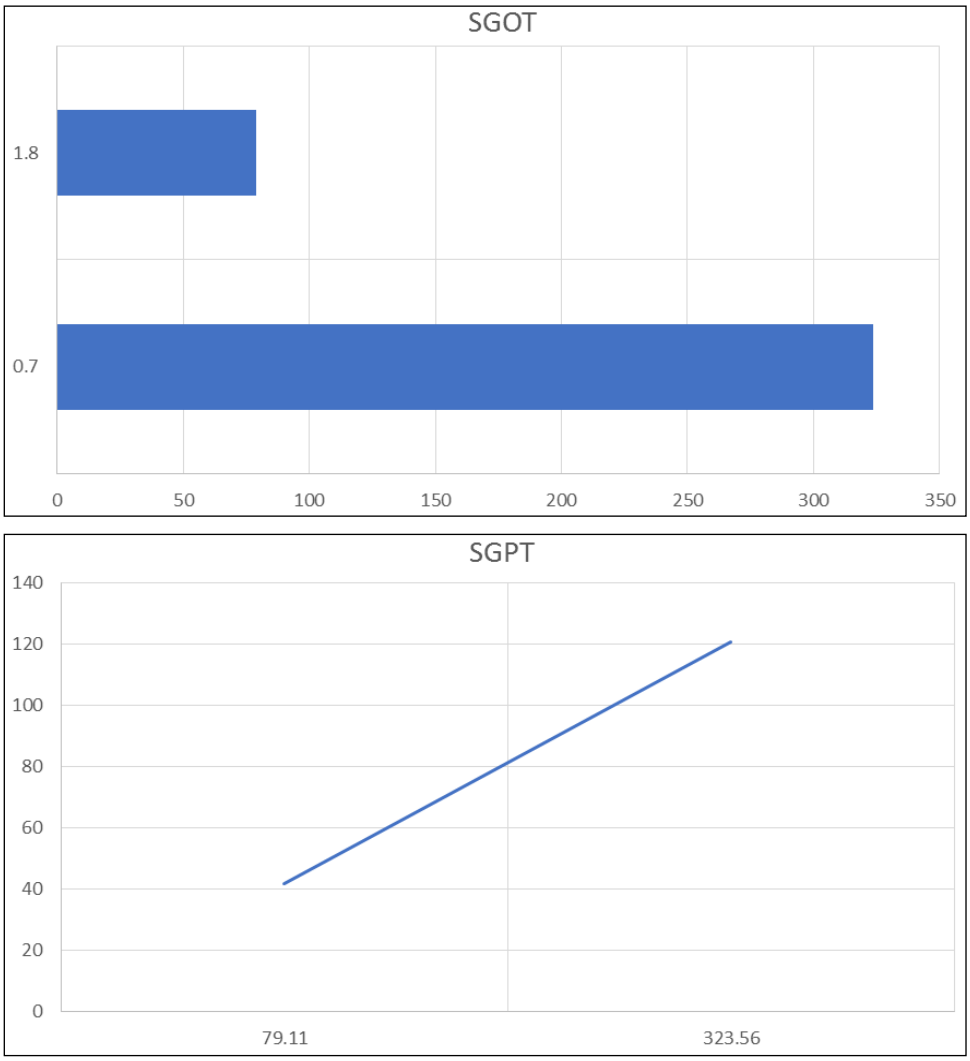

Fig 1: Enzymes

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 haemo concentration). 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

1. George R, Lum LCS. Clinical spectrum of dengue infection in Gubler DJ, Kuno G. Eds. Dengue and Dengue hemorrhagic fever. Washington Cab International 1997.
2. Viroj Wiwanitkit. Liver dysfunction in dengue Infection, an analysis of previously published Thai cases. J Ayub. Med Coll. Abbottabad 2007;19(1):10-12.
3. Srivenu Itha, Rajesh Kashyap, Narendra Krishnani, Viveka Saraswat, Gourdas Choudhari, Rakesh Aggarwal. Profile of liver involvement in dengue virus infection. The National Medical Journal of India 2005, 18(3).
4. Rashmi Kumar, Piyush Tripathi, Sanjeev Tripathi, Alok Kanodia, Vimala Venkatesh. Prevalence of dengue infection in North Indian Children with acute hepatic failure, Annals of Hepatology 2008;7(1):59-62.
5. Seneviratne SL, Malavige GN, De Silva HJ. Pathogenesis of liver involvement during dengue viral infections. Trans R Soc. Trop Med Hygl 2006;100:608-614.
6. Narayanan M, Arvind MA, Ambikapathy P, Prema R, Jeyapaul MP. Dengue fever Clinical and laboratory parameters associated with complications. Dengue Bulletin 2003, 27.
7. Jagadish K, Puja Jain *et al*. Hepatic involvement in dengue fever in children Iran J Pediatr. 2012;22(2):231-236.
8. Ashok S. Kapse. Dengue Illnesses. A Parthasarathy, MKC Nair, PSN Menon. IAP Textbook of Pediatrics 3rd Edition 2006, 247-254.