Int J Pharm Bio Sci 2013 Jan; 4(1): (B) 429 - 433

Research Article **Bio chemistry**

International Journal of Pharma and Bio Sciences **ISSN 0975-6299**

ROLE OF SERUM TRANSAMINASES IN DENGUE FEVER

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ABSTRACT

Dengue fever is an arboviral infection with the largest incidence worldwide, transmitted by *Aedes aegypti*. The aim of the study is to study the role of serum transaminases on dengue fever. The serum glutamic oxaloacetic transaminases (SGOT) and serum glutamic pyruvic transaminases (SGPT) were estimated by enzymatic (kinetic) methods for 50 dengue positive cases. Overall, 80% of dengue fever cases of this study showed increased values of serum transaminases. This study suggested that most of the dengue fever cases had elevated serum transaminases reflecting early liver damage. Early alterations of biochemical markers can predict dengue fever in patients with acute fever caused by dengue.

KEYWORDS: **Dengue fever, aspartate aminotransferase, alanine aminotransferase, hepatotoxicity.**

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INTRODUCTION

Dengue fever is a dreadful viral disease in developing countries like India with high mortality, without early and adequate intervention. It is an acute febrile disease diagnosed clinically by high fever. Clinical manifestation of dengue viral infection varies widely from no symptoms to dengue shock syndrome.[1,2] It is characterised by weak rapid pulse, narrow pulse pressure (<20mmHg) & cold clammy skin and restlessness. Nearly 100 million cases of dengue fever and between 250,000 and 500,000 cases of dengue fever are annually reported to World Health Organisation (WHO).[2,3] The incidence of hepatic dysfunction in dengue fever is high. The serum glutamic oxaloacetic transaminases (SGOT) and serum glutamic pyruvic transaminases (SGPT) were found to be increased 5-10 times in dengue fever due to liver parenchymal damage caused by the virus. Dengue fever is an acute febrile illness diagnosed clinically by high fever, constant headache, eye pain[1,2,4], hemorrhagic tendency (positive tourniquet test, spontaneous bruising etc,)[1,2], thrombocytopenia (<1,00,000 platelets/mm^3 or estimated on < 3 platelets/high power field) [1,2,5,6], evidence of plasma leakage (hematocrit >10%), pleural effusion, ascitis etc.[1,2,3.5,6] Dengue is caused by a Flavivirus with 4 serotypes (DENV1,2,3,4)[1,2,7], inoculated into humans by females *Aedes aegypti* or rarely by *Aedes albopictus* mosquito. Liver dysfunction in Dengue is due to direct effects of the virus on liver cells and an adverse effects of host immune reaction against the virus. Virus has been identified in liver tissue and dengue antigen identified within liver cells of affected individuals[6] leading to kupffer cell hyperplasia. Dengue viral antigens are mostly found in the liver cells surrounding necrotic areas of the liver. Apoptotic hepatocytes are found to be co-localised with dengue virus infected hepatocytes, suggesting that hepatocytes are the major site of dengue virus replication in the liver. Therefore, the aim of the present study is to analyse the role of liver enzymes in Dengue viral infection.

MATERIALS & METHODS

This study was done in a tertiary care hospital, Chennai. The study included 50 positive cases of dengue fever. The liver diseases with elevated serum transaminases other than dengue fever, history of concomitant diseases such as diabetes mellitus, acquired immunodeficiency syndrome (AIDS), hematologic disorders, cancer or cardiac disease, history of consumption of drugs which are hepatotoxic & history of fever excluding dengue fever were excluded in this study. The study was approved by the institutional ethical committee. Dengue fever was confirmed by immune-chromatographic method identifying the antibodies against dengue virus – IgM and IgG and dengue viral antigen – NS1. The SGOT, SGPT levels were estimated by enzymatic (kinetic) method. The SGOT, SGPT levels were estimated by kinetic method by adding 100µl of patient's serum with 1000µl of reagent and the concentration was read immediately in semi-automated analyser at 340nm wavelength with 1cm optical path at 37^{o}C temperature.

RESULTS

Table 1

Sample	No. of patients	% of patients
Male	26	52
Female	24	48

Table 2

Parameters	No. of males (%)	No. of females (%)	Total(%)
SGPT	16 (32%)	19 (38%)	35 (70%)
SGOT	20 (40%)	20 (40%)	40 (80%)

The serum glutamic pyruvic transaminases (SGPT) levels were found to be elevated more in females when compared with males. The serum glutamic oxaloacetic transaminases (SGOT) levels were found to be similar in both sexes. Overall, 80% of dengue fever cases of this study showed increased values of both SGOT and SGPT. SGOT was increased more compared to SGPT.

Table – 3

Cases	SGOT		Total	Chi-Square Test	P - value
	Normal	Increased			
Dengue patients	10	40	50	66.667	0.000

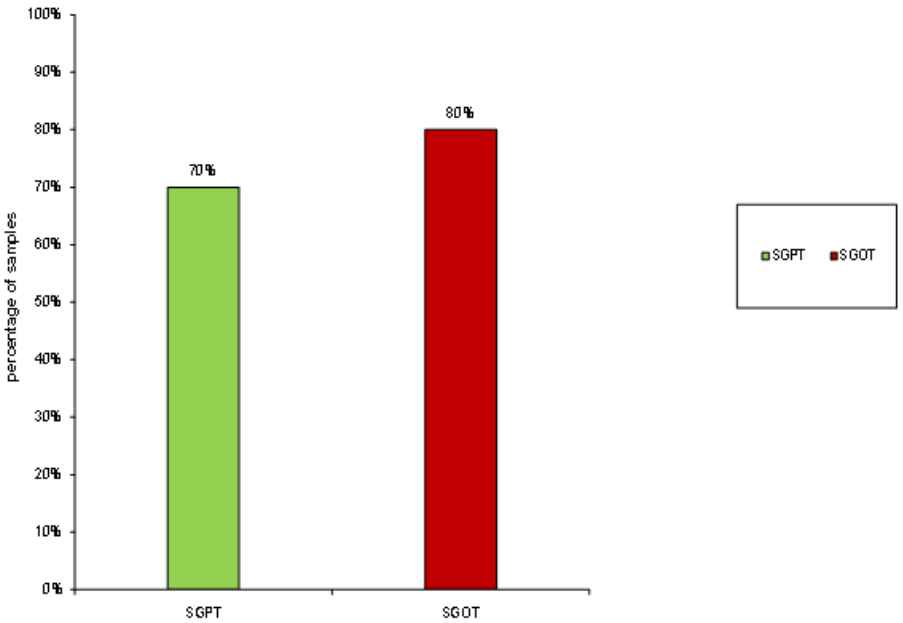
Note: Overall, 80% of dengue fever (40 cases) had increased SGOT levels.

Table – 4

Cases	SGPT		Total	Chi-Square Test	P – value
	Normal	Increased			
Dengue patients	15	35	50	53.846	0.000

Note: A 70% (35 cases) of dengue fever had increased SGPT.

Percentage of sample showing elevated levels of serum transaminases in Dengue fever (n=50)



DISCUSSION

Dengue is a dreadful viral infection common in tropical countries like India. The impact of the liver enzyme levels in the patients suffering from Dengue viral infection was studied in 50 cases attending a tertiary care hospital, Chennai. Here, none of the patients in our study had previous liver illness or abnormal SGOT and SGPT levels. This study supports the association between development of dengue fever and early alteration of the serum transaminases[8-12]. About 80% of the patients in our study had abnormal SGOT levels. This is in accordance with a study by Kuo *et al*[8] who had reported 90% of his study population had such abnormal SGOT levels. But other studies reported a higher percentage of Dengue cases with abnormal transaminases[9]. The SGPT level has been found to be elevated in 70% of the Dengue cases which is lesser than that reported in a study by Kuo *et al*[8]. These biomarkers have been proposed as indicators of severity in dengue patients.[4,8,9] Alteration in the serum levels of transaminases may be caused by damage of the liver parenchyma.[8,11,12.] In our study, Dengue was found to be more prevalent in males (52%) compared to female (48%), in contrast to the previous studies[13] where Dengue has been reported more in females. Previous studies suggest that biomarkers can predict a more severe form of dengue and could also be indicators of early tissue damage in the acute phase of dengue fever.[1,2] Such studies could facilitate in establishment of predictor biomarkers of Dengue severity that will help to decrease morbidity and mortality caused by this disease.

CONCLUSION

The study suggested that an increased level of serum transaminases is possible in dengue fever due to liver parenchymal damage caused by the virus. So estimating the levels of serum transaminases in dengue fever may help in early detection of liver cell damage. Avoidance of hepatotoxic drugs in Dengue patients may prevent further liver damage. This may pave way for early recuperation, early discharge from hospital, decreased mortality and morbidity. Thus, detection of liver enzyme

levels may be used as early prognostic markers for monitoring illness and identifying those who may benefit future therapies. Application of these findings may help optimize resource allocation, leading to a more opportune and effective care of those patients with Dengue endemic areas. Similar studies are required for the establishment of predictor biomarkers of Dengue severity that will help to decrease morbidity and mortality caused by this disease.

REFERENCES

1. Gubler D. J. Dengue and dengue hemorrhagic fever. Clin Microbiol Rev 1998; 11(3): 480–496.
2. Rigau-Pérez JG, Clark GG, Gubler DJ, Reiter P, Sanders EJ, Vorndam AV. Dengue and dengue haemorrhagic fever. Lancet 1998;352: 971–977.
3. Siqueira JB Jr, Martelli CM, Coelho GE, Simplicio AC, Hatch DL. Dengue and dengue hemorrhagic fever, Brazil, 1981–2002. Emerg Infect Dis. 2005;11(1):48–53.
4. Kalayanarooj S, Vaughn DW, Nimmannitya S, Green S, Suntayakorn S, N. Kunentrasai et al. Early clinical and laboratory indicators of acute dengue illness. J Infect Dis. 1997;176:313–321.
5. Organización Panamericana de la Salud. Definiciones de casos. Dengue. Boletín Epidemiológico. 2000;21:14–15.
6. Rigau-Perez JG, Bonilla GL. An evaluation of modified case definitions for the detection of dengue hemorrhagic fever. Puerto Rico Association of Epidemiologists. P R Health Sci J. 1999;18:347–352.
7. Sivigila, 2002. Comportamiento por Regiones del Dengue en el 2001. Boletín Epidemiológico Semanal. Semana epidemiológica No. 02. Enero 06 a 12 de 2002.
8. Kuo CH, Tai DI, Chang-Chien CS, Lan CK, Chiou SS, Liaw YF. Liver biochemical tests and dengue fever. Am J Trop Med Hyg. 1992;47:265–270.
9. Alvarez ME, Ramirez-Ronda CH. Dengue and hepatic failure. Am J Med. 1985;79: 670–674.
10. Setiawan MW, Samsi TK, Wulur H, Sugianto D, Pool T. Epigastric pain and sonographic assessment of the pancreas in dengue hemorrhagic fever. J Clin Ultrasound. 1998;26:257–259.
11. Jusuf H, Sudjana P, Djumhana A, Abdurachman SA. DHF with complication of acute pancreatitis related hyperglycemia: a case report. Southeast Asian J Trop Med Public Health. 1998;29: 367–369.
12. Krippner R, Hanish G, Kretschmer H. Dengue fever with hemorrhagic manifestations after a stay in Thailand. Dtsch Med Wochenschr. 1990;115: 858–862.
13. Souza a LJ, Alves JG, Nogueira RM, Gicovate Neto C, Bastos DA, Siqueira EW, et al. Aminotransferase changes and acute hepatitis in patients with dengue fever: analysis of 1,585 cases. *Braz J Infect Dis*. 2004; 8(2): 156-163.