

ORIGINAL ARTICLE

Assessment of liver dysfunction in children with dengue

Rajesh Pachoria

Associate Professor, Department of Paediatrics, Major S D Singh Medical College & Hospital, Farrukhabad, Uttar Pradesh, India

ABSTRACT:

Background: Dengue fever is a viral illness caused by the dengue virus, which is transmitted to humans through the bite of infected female mosquitoes. The present study was conducted to assess liver dysfunction in children with dengue. **Materials & Methods:** 56 NS1 Ag-positive or dengue IgM-positive confirmed cases of dengue fever in children of both genders were carefully examined and subjected to complete blood count, serum IgM and IgG antibodies, prothrombin time, international normalized ratio, liver function tests such as alanine transaminase (ALT) and aspartate transaminase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), serum bilirubin, prothrombin time (PT), the international normalized ratio (INR), total protein and albumin. etc. **Results:** Out of 56 patients, males were 26 and females were 30. The maximum cases of dengue without warning signs were seen in age group >6 years, with warning signs in age group 1-5 years and severe cases (9) in age group 1-5 years. The difference was significant ($P < 0.05$). Liver function tests in cases with dengue without warning signs, with warning signs and with severe dengue in reference to total bilirubin was 0.61, 1.4 and 1.9 respectively. The mean serum protein was 6.2, 6.5 and 6.8. albumin was 4.8, 4.6 and 4.2, AST was 113.5, 168.4 and 405.2, ALT was 94.2, 148.4 and 402.6 and alkaline phosphatase level was 120.6, 132.8 and 48.5 respectively. The difference was significant ($P < 0.05$). **Conclusion:** There were alterations in liver function tests in children with dengue fever. Hence early and prompt treatment is required to prevent complications.

Key words: Dengue fever, alanine transaminase, liver function tests

Corresponding author: Rajesh Pachoria, Associate Professor, Department of Paediatrics, Major S D Singh Medical College & Hospital, Farrukhabad, Uttar Pradesh, India

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INTRODUCTION

Dengue fever is a viral illness caused by the dengue virus, which is transmitted to humans through the bite of infected female mosquitoes, primarily the *Aedes aegypti* mosquito. Dengue is a significant public health concern in many tropical and subtropical regions of the world, including parts of Southeast Asia, the Pacific Islands, the Caribbean, Central and South America, and Africa.¹

Dengue is caused by one of four closely related viruses, known as Dengue virus serotypes 1, 2, 3, and 4 (DENV-1, DENV-2, DENV-3, and DENV-4).² Infection with one serotype does not provide immunity against the others and can, in some cases, lead to more severe disease upon subsequent infections with a different serotype. Dengue is primarily transmitted to humans through the bite of infected *Aedes* mosquitoes, particularly *Aedes aegypti*. These mosquitoes are often found in urban and semi-urban areas and are known for their daytime feeding habits.³

Liver dysfunction is a common feature of dengue fever, and it can range from mild to severe. Dengue virus infection can affect the liver, leading to a variety of liver-related symptoms and laboratory abnormalities. One of the most common signs of liver involvement in dengue is the elevation of liver

enzymes, particularly alanine transaminase (ALT) and aspartate transaminase (AST).⁴ Elevated liver enzymes are often seen during the acute phase of the illness and can be an indicator of liver inflammation. Dengue virus can directly infect liver cells, causing hepatic injury. This injury can lead to hepatomegaly (enlargement of the liver) and tenderness in the right upper abdomen.⁵ The present study was conducted to assess liver dysfunction in children with dengue.

MATERIALS & METHODS

The present study consisted of 56 cases of dengue fever in children of both genders. Parents gave their written consent to participate in the study. All were NS1 Ag-positive or dengue IgM-positive cases.

Data such as name, age, gender etc. was recorded. All were carefully examined and subjected to complete blood count, serum IgM and IgG antibodies, prothrombin time, international normalized ratio, liver function tests such as alanine transaminase (ALT) and aspartate transaminase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), serum bilirubin, prothrombin time (PT), the international normalized ratio (INR), total protein and albumin. etc. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 56		
Gender	Male	Female
Number	26	30

Table I shows that out of 56 patients, males were 26 and females were 30.

Table II Distribution of cases based on severity

Age group	Dengue without warning signs	With warning signs	Severe dengue	P value
<1 year	1	9	7	0.05
1-5 years	3	12	9	0.02
>6 years	4	6	5	0.9

Table II shows that maximum cases of dengue without warning signs were seen in age group >6 years, with warning signs in age group 1-5 years and severe cases (9) in age group 1-5 years. The difference was significant ($P < 0.05$).

Table III Assessment of liver function tests

LFT	Dengue without warning signs	With warning signs	Severe dengue	P value
Total bilirubin	0.61	1.4	1.9	0.01
Serum protein	6.2	6.5	6.8	0.02
Albumin	4.8	4.6	4.2	0.97
AST	113.5	168.4	405.2	0.03
ALT	94.2	148.4	402.6	0.02
Alkaline phosphatase	120.6	132.8	48.5	0.01

Table III, graph I shows that LFT in cases with dengue without warning signs, with warning signs and with severe dengue in reference to total bilirubin was 0.61, 1.4 and 1.9 respectively. The mean serum protein was 6.2, 6.5 and 6.8. albumin was 4.8, 4.6 and 4.2, AST was 113.5, 168.4 and 405.2, ALT was 94.2, 148.4 and 402.6 and alkaline phosphatase level was 120.6, 132.8 and 48.5 respectively. The difference was significant ($P < 0.05$).

DISCUSSION

Liver dysfunction in dengue can manifest with symptoms such as abdominal pain, jaundice (yellowing of the skin and eyes), dark urine, and pale stools.⁶ These symptoms are more likely to occur in severe cases. The severity of liver dysfunction in dengue can vary. In mild cases, there may be only a slight increase in liver enzymes with no significant symptoms.^{7,8} In severe cases, however, liver involvement can contribute to the development of dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS), which are life-threatening complications of dengue fever.^{9,10} The present study was conducted to assess liver dysfunction in children with dengue.

We found that out of 56 patients, males were 26 and females were 30. Mittal et al¹¹ found cases of dengue fever (8%), dengue hemorrhagic fever (51%) and dengue shock syndrome (42%). The mean age (\pm SD) of children were 8.3 ± 3.5 y with male: female ratio 1.32. Mean duration of fever (\pm SD) was $6.3 \text{ d} \pm 3.7 \text{ d}$. The clinical features included fever (100%), headache (63%), abdominal pain (71%), petechia (35.5%), rash (26.6%) and bleeding manifestations (48.8%). On examination, Hess test (33.3%), signs of fluid retention (23%), pallor (13.3%), signs of circulatory failure (43%), hepatomegaly (31.1%) and splenomegaly (27%) were positive. Laboratory

investigations revealed mean (\pm SD) hemoglobin 11.5 g/dl (± 1.7), hematocrit 36.1(± 5.5), leucocyte count 7,551/mm³ and platelet count 38,800 mm³ on day of admission. A total of 92.6% of children had thrombocytopenia and 19.2% had abnormal leucocyte count. Deranged liver function tests were observed in 48 children. The mean (\pm SD) of hospitalization and platelet recovery were $4.2 \pm 2.3 \text{ d}$ and $3.6 \pm 1.3 \text{ d}$ respectively and did not vary according to disease category ($P > 0.05$). Bleeding manifestations were not related to platelet count ($P > 0.05$). There was no statistical difference in the demographic, clinical or laboratory observations according to disease category ($P > 0.05$). A total of 24 children had evidence of other co-infections and four had atypical complications. Dengue 2 virus was the strain reported in most of the cases.

We found that the maximum cases of dengue without warning signs were seen in age group >6 years, with warning signs in age group 1-5 years and severe cases (9) in age group 1-5 years. We observed that liver function tests in cases with dengue without warning signs, with warning signs and with severe dengue in reference to total bilirubin was 0.61, 1.4 and 1.9 respectively. The mean serum protein was 6.2, 6.5 and 6.8. albumin was 4.8, 4.6 and 4.2, AST was 113.5, 168.4 and 405.2, ALT was 94.2, 148.4 and 402.6 and alkaline phosphatase level was 120.6, 132.8 and 48.5

respectively. Kalayanarooj et al¹² found that patients with dengue had significantly lower platelet, white blood cell (WBC) and neutrophil counts, and a higher frequency of petechiae than OFI patients. Higher frequencies of myalgia, rash, hemorrhagic signs, lethargy/prostration, and arthralgia/joint pain and higher hematocrits were reported in adult patients with dengue but not in children. Most multivariable models included platelet count, WBC, rash, and signs of liver damage; however, none had high statistical validity and none considered changes in clinical features over the course of illness.

The limitation of the study is small sample size.

CONCLUSION

Authors found that there were alterations in liver function tests in children with dengue fever. Hence early and prompt treatment is required to prevent complications.

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