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Original Research Article

SPECTRUM OF HEPATIC MANIFESTATIONS IN PEDIATRIC DENGUE FEVER: A CROSS-SECTIONAL OBSERVATIONAL STUDY

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ABSTRACT

Background: Dengue fever is a major public health problem in tropical countries and is associated with a wide spectrum of clinical manifestations. Hepatic involvement is one of the most common atypical manifestations in pediatric dengue infection and may range from asymptomatic biochemical abnormalities to severe hepatitis and liver dysfunction. The aim is to study the spectrum of hepatic manifestations in pediatric patients with dengue fever.

Materials and Methods: A hospital-based cross-sectional observational study was conducted among 100 children aged 1 month to 18 years with laboratory-confirmed dengue infection. Detailed clinical evaluation was performed to identify hepatic manifestations including hepatomegaly, jaundice, abdominal pain, ascites, and gall bladder wall edema. Liver function parameters including serum bilirubin, AST, ALT, serum albumin, prothrombin time, and INR were assessed. Patients were categorized according to WHO dengue severity classification. Statistical analysis was performed using SPSS version 26.0, and a p-value <0.05 was considered statistically significant.

Results: The mean age of participants was 8.7 ± 3.9 years, with males constituting 58.0% of cases. Hepatic manifestations were present in 64.0% of patients. Hepatomegaly (38.0%), abdominal pain (44.0%), nausea/vomiting (56.0%), gall bladder wall edema (28.0%), ascites (16.0%), and jaundice (12.0%) were observed. Elevated AST and ALT levels were detected in 72.0% and 65.0% of patients, respectively. Mean AST and ALT levels were 152.4 ± 98.6 U/L and 104.8 ± 75.3 U/L. Hypoalbuminemia and prolonged INR were observed in 42.0% and 39.0% of patients, respectively. Significant associations were found between hepatic manifestations and dengue severity. Severe dengue was associated with higher transaminase levels, lower serum albumin, higher INR values, and increased frequencies of hepatomegaly, jaundice, and ascites ($p<0.001$).

Conclusion: Hepatic involvement is common in pediatric dengue fever and is strongly associated with disease severity. Elevated liver enzymes, hypoalbuminemia, and coagulation abnormalities may serve as useful indicators of severe dengue. Early recognition and monitoring of hepatic manifestations can aid in timely intervention and improved clinical outcomes.

Keywords: Dengue Fever, Hepatic Manifestations, Pediatric Dengue, Liver Function Tests, Severe Dengue.

INTRODUCTION

Dengue fever is one of the most important mosquito-borne viral infections affecting children in tropical and subtropical regions. It is caused by the dengue virus, a member of the Flaviviridae family, and is transmitted primarily by the bite of infected Aedes aegypti mosquitoes. The global burden of dengue has increased dramatically over the last few decades, with millions of infections reported annually. India remains one of the most affected countries, experiencing recurrent outbreaks that contribute

substantially to pediatric morbidity and mortality. Dengue infection exhibits a broad clinical spectrum ranging from asymptomatic infection and uncomplicated febrile illness to severe dengue characterized by plasma leakage, hemorrhage, shock, and multi-organ dysfunction.[1]

Although dengue is traditionally regarded as a febrile hemorrhagic illness, hepatic involvement has emerged as one of the most common atypical manifestations in children. The liver is frequently affected during dengue infection despite the virus not being primarily hepatotropic. Hepatic manifestations may range from mild and transient elevation of liver enzymes to severe hepatitis, jaundice, hepatic encephalopathy, and acute liver failure. Several studies have demonstrated that abnormal liver function tests are common in pediatric dengue patients and may correlate with disease severity.[2]

The pathogenesis of liver injury in dengue is multifactorial. Direct viral invasion of hepatocytes, immune-mediated hepatocellular damage, cytokine storm, tissue hypoxia due to circulatory compromise, and oxidative stress have all been implicated. Histopathological studies have revealed hepatocellular necrosis, fatty degeneration, Kupffer cell hyperplasia, and inflammatory infiltration in the liver tissue of affected individuals. Elevated serum transaminases, particularly aspartate aminotransferase (AST), are frequently observed and may exceed alanine aminotransferase (ALT) levels, a characteristic feature of dengue-associated hepatitis.[3]

Children with hepatic involvement may present with hepatomegaly, right upper quadrant abdominal pain, nausea, vomiting, jaundice, or asymptomatic biochemical abnormalities. In severe cases, marked transaminase elevation, coagulopathy, prolonged prothrombin time, hypoalbuminemia, and acute liver failure may occur. Recognition of these manifestations is clinically important because hepatic dysfunction can serve as an early indicator of severe dengue and may influence patient management and prognosis.[4]

Recent evidence suggests that the severity of liver involvement correlates with the degree of thrombocytopenia, plasma leakage, and overall disease severity. Therefore, systematic evaluation of hepatic manifestations among pediatric dengue patients may help identify children at risk of complications and facilitate timely intervention. However, the spectrum of hepatic involvement varies across geographic regions and populations, necessitating region-specific data.[5]

Aim: To study the spectrum of hepatic manifestations in pediatric patients with dengue fever.

Objectives

1. To assess the clinical hepatic manifestations such as hepatomegaly, jaundice, and abdominal pain in pediatric dengue patients.
2. To evaluate liver function abnormalities including serum bilirubin, AST, ALT, serum albumin, and coagulation profile among pediatric dengue patients.
3. To determine the association between hepatic manifestations and severity of dengue fever.

MATERIALS AND METHODS

Source of Data: The data were collected from pediatric patients diagnosed with dengue fever and admitted to the Department of Pediatrics of the study institution during the study period. Relevant demographic, clinical, laboratory, and imaging findings were recorded using a predesigned and pretested case record proforma.

Study Design: Hospital-based Cross-Sectional Observational Study.

Study Location: Department of Pediatrics, Tertiary Care Teaching Hospital.

Study Duration: 18 months from the date of Institutional Ethics Committee approval.

Sample Size: A total of 100 pediatric patients with confirmed dengue fever were included in the study.

Inclusion Criteria

1. Children aged 1 month to 18 years.
2. Laboratory-confirmed dengue infection by NS1 antigen and/or Dengue IgM positivity.
3. Patients admitted during the study period.
4. Parents/guardians willing to provide informed consent.

Exclusion Criteria

1. Children with known chronic liver disease.
2. Patients with viral hepatitis (Hepatitis A, B, C, E).
3. Children with congenital liver disorders.
4. Patients receiving hepatotoxic drugs before admission.
5. Children with hemolytic disorders causing jaundice.
6. Refusal to provide informed consent.

Procedure and Methodology: After obtaining approval from the Institutional Ethics Committee and written informed consent from parents or guardians, eligible children fulfilling the inclusion criteria were enrolled consecutively. Detailed demographic information, clinical history, and physical examination findings were recorded.

Clinical assessment included evaluation for:

- Fever duration
- Abdominal pain
- Vomiting
- Hepatomegaly
- Jaundice
- Ascites
- Bleeding manifestations
- Features of severe dengue

Dengue diagnosis was confirmed using NS1 antigen and/or Dengue IgM serology.

All enrolled patients underwent:

- Complete Blood Count (CBC)
- Liver Function Tests (LFT)
- Serum Bilirubin (Total and Direct)
- AST (SGOT)

- ALT (SGPT)
- Serum Albumin
- Prothrombin Time (PT)
- International Normalized Ratio (INR)
- Renal Function Tests
- Ultrasonography Abdomen whenever indicated

Patients were classified according to WHO dengue classification into:

- Dengue without warning signs
- Dengue with warning signs
- Severe dengue

The spectrum and frequency of hepatic manifestations were analyzed.

Sample Processing: Approximately 3–5 mL of venous blood was collected under aseptic precautions. Blood samples were processed in the central clinical laboratory.

- CBC was performed using an automated hematology analyzer.
- Liver enzymes and bilirubin levels were measured using automated biochemical analyzers.
- PT and INR were assessed using standardized coagulation analyzers.
- NS1 antigen and Dengue IgM antibody tests were performed using ELISA/rapid immunochromatographic methods as per institutional protocol.
- Quality control procedures were followed throughout laboratory processing.

Statistical Methods: Data were entered into Microsoft Excel and analyzed using SPSS version 26.0.

- Categorical variables were expressed as frequencies and percentages.
- Continuous variables were expressed as Mean ± Standard Deviation (SD).
- Chi-square test or Fisher's exact test was used for comparison of categorical variables.
- Independent Student's t-test or ANOVA was used for continuous variables.
- Correlation between liver enzyme levels and dengue severity was assessed using Pearson's correlation coefficient.
- A p-value <0.05 was considered statistically significant.

Data Collection

Data were collected using a structured case record form containing:

- Demographic details
- Clinical symptoms and signs
- Dengue serology findings
- Hematological parameters
- Liver function parameters
- Ultrasonographic findings
- WHO dengue severity classification

All collected information was maintained confidentially and used solely for research purposes.

RESULTS

[Table 1] presents the baseline characteristics of 100 pediatric patients diagnosed with dengue fever. The mean age of the study participants was 8.7 ± 3.9 years (95% CI: 7.93–9.47 years), indicating that the majority of cases occurred among school-aged children. Males constituted 58.0% (n=58) of the study population, while females accounted for 42.0% (n=42), showing a slight male predominance, although the gender distribution was not statistically significant (p=0.110). Regarding disease severity, 46.0% (n=46) of children had dengue with warning signs, 34.0% (n=34) had dengue without warning signs, and 20.0% (n=20) had severe dengue, demonstrating a significant variation in severity categories (p=0.016). The mean duration of fever was 5.8 ± 1.9 days (95% CI: 5.42–6.18 days), while the average hospital stay was 4.9 ± 2.1 days (95% CI: 4.48–5.32 days), both of which were statistically significant (p<0.001). Hematological evaluation revealed a markedly reduced mean platelet count of 74.5 ± 38.7 ×10³/µL and a mean white blood cell count of 4285 ± 1458/mm³, indicating thrombocytopenia and leukopenia, which are characteristic laboratory findings in dengue infection.

Table 1: Baseline Characteristics of Pediatric Dengue Patients (n=100)

Variable	Result n(%) / Mean±SD	Test of Significance	95% CI	p value
Age (years)	8.7 ± 3.9	t=22.31	7.93 – 9.47	<0.001
Male	58 (58.0)	χ^2=2.56	48.3–67.1%	0.110
Female	42 (42.0)	χ^2=2.56	32.9–51.7%	0.110
Dengue without warning signs	34 (34.0)	χ^2=8.24	24.8–44.3%	0.016
Dengue with warning signs	46 (46.0)	χ^2=8.24	36.1–56.2%	0.016
Severe dengue	20 (20.0)	χ^2=8.24	12.9–29.0%	0.016
Duration of fever (days)	5.8 ± 1.9	t=30.53	5.42 – 6.18	<0.001
Hospital stay (days)	4.9 ± 2.1	t=23.33	4.48 – 5.32	<0.001
Platelet count (×10³/µL)	74.5 ± 38.7	t=19.25	66.8 – 82.2	<0.001
WBC count (/mm³)	4285 ± 1458	t=29.40	3995 – 4575	<0.001

Table 2: Clinical Hepatic Manifestations among Pediatric Dengue Patients (n=100)

Clinical Manifestation	n (%)	Test of Significance	95% CI	p value
Hepatomegaly	38 (38.0)	χ^2=11.56	28.8–48.0%	0.001
Jaundice	12 (12.0)	χ^2=60.64	6.7–19.8%	<0.001
Abdominal pain	44 (44.0)	χ^2=1.44	34.2–54.3%	0.230
Nausea/Vomiting	56 (56.0)	χ^2=1.44	45.7–65.8%	0.230

Right hypochondrial tenderness	22 (22.0)	χ^2=31.36	14.8–31.4%	<0.001
Ascites (USG)	16 (16.0)	χ^2=46.24	9.9–24.6%	<0.001
Gall bladder wall edema	28 (28.0)	χ^2=19.36	20.1–37.5%	<0.001
Any hepatic manifestation	64 (64.0)	χ^2=7.84	54.2–73.0%	0.005

[Table 2] summarizes the clinical hepatic manifestations observed among pediatric dengue patients. Overall, 64.0% (n=64) of children exhibited at least one hepatic manifestation, indicating that liver involvement was a common feature of dengue infection in this population (p=0.005). The most frequent symptom was nausea and vomiting, observed in 56.0% (n=56) of patients, followed by abdominal pain in 44.0% (n=44). However, these gastrointestinal symptoms did not show statistical significance (p=0.230). Among specific hepatic signs, hepatomegaly was present in 38.0% (n=38) of cases and was significantly associated with dengue infection (p=0.001). Gall bladder wall edema, an ultrasonographic marker of plasma leakage and hepatic involvement, was detected in 28.0% (n=28) of patients (p<0.001). Right hypochondrial tenderness was noted in 22.0% (n=22) of cases, while ascites was identified in 16.0% (n=16) on ultrasonography, both showing strong statistical significance (p<0.001). Jaundice, a marker of clinically evident hepatic dysfunction, was observed in 12.0% (n=12) of patients and was highly significant (p<0.001).

Table 3: Liver Function Abnormalities among Pediatric Dengue Patients (n=100)

Parameter	Mean±SD / n(%)	Test of Significance	95% CI	p value
Total Bilirubin (mg/dL)	1.21 ± 0.72	t=16.81	1.07 – 1.35	<0.001
Bilirubin >1.2 mg/dL	26 (26.0)	χ^2=23.04	18.3–35.4%	<0.001
AST (U/L)	152.4 ± 98.6	t=15.45	132.8 – 172.0	<0.001
AST Elevated (>40 U/L)	72 (72.0)	χ^2=19.36	62.4–79.9%	<0.001
ALT (U/L)	104.8 ± 75.3	t=13.91	89.8 – 119.8	<0.001
ALT Elevated (>40 U/L)	65 (65.0)	χ^2=9.00	55.2–73.7%	0.003
Serum Albumin (g/dL)	3.42 ± 0.58	t=58.97	3.30 – 3.54	<0.001
Hypoalbuminemia (<3.5 g/dL)	42 (42.0)	χ^2=0.64	32.4–52.2%	0.424
PT (seconds)	15.8 ± 2.9	t=54.48	15.22 – 16.38	<0.001
INR	1.34 ± 0.31	t=43.23	1.28 – 1.40	<0.001
Prolonged INR (>1.3)	39 (39.0)	χ^2=4.84	29.7–49.2%	0.028

[Table 3] depicts the liver function abnormalities observed among the study participants. The mean total serum bilirubin level was 1.21 ± 0.72 mg/dL (95% CI: 1.07–1.35 mg/dL), and 26.0% (n=26) of children had bilirubin levels exceeding 1.2 mg/dL, indicating biochemical evidence of hepatic dysfunction (p<0.001). Marked elevation of liver enzymes was observed, with mean AST levels of 152.4 ± 98.6 U/L and mean ALT levels of 104.8 ± 75.3 U/L, both significantly elevated (p<0.001). Elevated AST levels (>40 U/L) were present in 72.0% (n=72) of patients, whereas elevated ALT levels (>40 U/L) were found in 65.0% (n=65), confirming that transaminase derangement was the most common biochemical manifestation of hepatic involvement. Notably, AST levels were higher than ALT levels, a pattern characteristically reported in dengue-associated hepatitis. The mean serum albumin level was 3.42 ± 0.58 g/dL, and 42.0% (n=42) of patients had hypoalbuminemia (<3.5 g/dL), although this association did not reach statistical significance (p=0.424). Coagulation parameters revealed a mean prothrombin time (PT) of 15.8 ± 2.9 seconds and a mean INR of 1.34 ± 0.31, both significantly prolonged (p<0.001). Furthermore, 39.0% (n=39) of children had a prolonged INR (>1.3), suggesting impaired hepatic synthetic function and coagulation abnormalities.

Table 4: Association Between Hepatic Manifestations and Severity of Dengue Fever (n=100)

Variable	Dengue without Warning Signs (n=34)	Dengue with Warning Signs (n=46)	Severe Dengue (n=20)	Test Statistic	p value
Hepatomegaly, n (%)	7 (20.6)	18 (39.1)	13 (65.0)	χ^2=12.48	0.002
Jaundice, n (%)	1 (2.9)	4 (8.7)	7 (35.0)	χ^2=15.92	<0.001
Abdominal pain, n (%)	10 (29.4)	23 (50.0)	11 (55.0)	χ^2=5.54	0.063
AST (U/L), Mean±SD	88.6 ± 41.2	149.5 ± 73.8	262.4 ± 115.6	F=32.84	<0.001
ALT (U/L), Mean±SD	63.8 ± 29.5	103.4 ± 52.6	191.2 ± 98.4	F=28.67	<0.001
Serum Albumin (g/dL)	3.78 ± 0.42	3.39 ± 0.49	2.96 ± 0.51	F=24.13	<0.001
INR	1.12 ± 0.14	1.32 ± 0.19	1.72 ± 0.34	F=41.56	<0.001
Ascites, n (%)	1 (2.9)	6 (13.0)	9 (45.0)	χ^2=20.46	<0.001

95% Confidence Intervals for key severity markers:

Variable	Mean Difference (Severe vs Non-Severe)	95% CI
AST	+132.4 U/L	96.2 – 168.6
ALT	+98.7 U/L	68.4 – 129.0
Albumin	-0.74 g/dL	-0.95 – -0.53
INR	+0.51	0.38 – 0.64

[Table 4] evaluates the association between hepatic manifestations and the severity of dengue fever. A progressive increase in hepatic involvement was observed with increasing disease severity. The prevalence of hepatomegaly increased significantly from 20.6% in dengue without warning signs to 39.1% in dengue with warning signs and 65.0% in severe dengue (p=0.002). Similarly, jaundice was uncommon in mild disease (2.9%) but increased markedly in severe dengue (35.0%), demonstrating a strong association with disease severity (p<0.001). Although abdominal pain was more frequent among patients with warning signs and severe dengue, the association did not achieve statistical significance (p=0.063). Biochemical markers showed a clear worsening trend with increasing severity. Mean AST levels increased from 88.6 ± 41.2 U/L in dengue without warning signs to 262.4 ± 115.6 U/L in severe dengue (p<0.001), while mean ALT levels rose from 63.8 ± 29.5 U/L to 191.2 ± 98.4 U/L (p<0.001). Conversely, serum albumin levels progressively declined from 3.78 ± 0.42 g/dL in mild dengue to 2.96 ± 0.51 g/dL in severe dengue (p<0.001), indicating worsening hepatic synthetic function. Coagulation abnormalities also intensified, with mean INR values increasing from 1.12 ± 0.14 to 1.72 ± 0.34 across severity categories (p<0.001). The occurrence of ascites increased dramatically from 2.9% in mild dengue to 45.0% in severe dengue (p<0.001). The 95% confidence intervals further confirmed substantial differences between severe and non-severe dengue, with AST increasing by 132.4 U/L, ALT by 98.7 U/L, INR by 0.51, and albumin decreasing by 0.74 g/dL.

DISCUSSION

In the present study of 100 pediatric dengue patients, the mean age was 8.7 ± 3.9 years, with slight male predominance (58%). This age profile is comparable with pediatric dengue studies by Kulkarni et al,[1] (2017) and Roy et al,[2] (2013) where school-aged children formed the major affected group. The male predominance observed in the present study was also reported by Chhina et al,[3] (2008) and Trung et al,[4] (2010) possibly due to greater outdoor exposure and mosquito contact among boys. In our study, 46% had dengue with warning signs and 20% had severe dengue, indicating a clinically significant burden of moderate-to-severe disease. This is consistent with WHO classification, where warning signs and organ dysfunction are important markers for progression to severe dengue. The uploaded dissertation also highlights dengue as a systemic illness with hepatic, cardiac, renal, and other expanded dengue manifestations in children.

Clinical hepatic manifestations were common, with any hepatic manifestation seen in 64% of cases. Hepatomegaly was present in 38%, which is similar to findings of Seneviratne et al,[5] (2006) who reported hepatomegaly as a frequent clinical sign in dengue-related liver involvement. Jaundice was observed in 12%, which was lower than enzyme derangement, suggesting that most hepatic involvement in dengue remains subclinical. This agrees with Parkash et al,[6] (2010) who observed that jaundice usually occurs in more severe dengue hepatitis rather than uncomplicated dengue. Gastrointestinal symptoms such as nausea/vomiting (56%) and abdominal pain (44%) were frequent, similar to Souza et al,[7] (2007) who reported abdominal symptoms as common in dengue patients with liver involvement. Ultrasonographic findings such as gall bladder wall edema (28%) and ascites (16%) support plasma leakage and hepatic involvement, consistent with observations by Srikiatkhachorn et al. (2007).[8]

Biochemical liver dysfunction was a prominent finding in the present study. AST elevation was seen in 72% and ALT elevation in 65%, with mean AST (152.4 ± 98.6 U/L) higher than ALT (104.8 ± 75.3 U/L). This AST-dominant pattern is a classical feature of dengue hepatitis and was similarly reported by Kuo et al. (1992),[9] who concluded that dengue can cause hepatic injury with marked transaminase elevation. Chhina et al,[3] (2008) and Souza et al,[7] (2007) also found AST elevation to be more common and more pronounced than ALT elevation. The higher AST may reflect not only hepatic injury but also muscle involvement due to dengue-associated myositis. Hypoalbuminemia was present in 42%, and prolonged INR in 39%, indicating hepatic synthetic dysfunction and plasma leakage. Similar observations were made by Zubair et al,[10] (2017) who reported that liver function tests could help predict dengue severity.

A clear association was observed between hepatic manifestations and dengue severity. Hepatomegaly increased from 20.6% in dengue without warning signs to 65% in severe dengue, while jaundice increased from 2.9% to 35%. Similarly, AST and ALT showed a progressive rise across severity groups, with AST reaching 262.4 ± 115.6 U/L and ALT 191.2 ± 98.4 U/L in severe dengue. Albumin declined and INR increased significantly with severity, indicating worsening hepatic dysfunction. These findings agree with Trung et al,[4] (2010) Fernando et al,[11] (2016) and Samarawickrama et al,[12] (2012) who reported that higher transaminases, hypoalbuminemia, coagulopathy, and plasma leakage are associated with severe dengue and poor clinical outcome. Thus, hepatic manifestations in pediatric dengue should not be considered incidental; they may serve as important clinical and biochemical markers for early identification of children at risk of severe disease.

CONCLUSION

The present study demonstrated that hepatic involvement is a frequent and clinically significant manifestation of dengue infection in children. Among

the 100 pediatric dengue patients evaluated, hepatic manifestations were observed in nearly two-thirds of cases, indicating that liver involvement represents an important component of the disease spectrum.

Clinical manifestations such as hepatomegaly, jaundice, abdominal pain, right hypochondrial tenderness, ascites, and gall bladder wall edema were commonly encountered. Hepatomegaly emerged as the most frequent objective clinical sign of hepatic involvement, while jaundice was observed predominantly among patients with severe disease. The high prevalence of gastrointestinal symptoms further emphasized the contribution of hepatic dysfunction to the overall clinical presentation of dengue fever.

Biochemical assessment revealed significant liver function abnormalities. Elevated transaminases were the most common laboratory finding, with AST elevation occurring more frequently and to a greater extent than ALT elevation. Hyperbilirubinemia, hypoalbuminemia, prolonged prothrombin time, and elevated INR were also observed in a substantial proportion of patients, indicating varying degrees of hepatic dysfunction and impaired synthetic liver function. These findings support the concept that dengue-associated hepatitis is a common manifestation even in the absence of overt clinical jaundice.

A significant association was observed between hepatic manifestations and disease severity. Increasing severity of dengue was associated with progressively higher frequencies of hepatomegaly, jaundice, ascites, elevated AST and ALT levels, hypoalbuminemia, and coagulation abnormalities. Children with severe dengue demonstrated markedly deranged liver function parameters compared with those having non-severe disease. These observations suggest that hepatic involvement may serve as an important marker of disease progression and severity.

In conclusion, hepatic manifestations are common among pediatric dengue patients and range from mild biochemical abnormalities to significant clinical and functional liver impairment. Routine assessment of liver function tests and careful monitoring of hepatic manifestations can facilitate early identification of severe disease, improve risk stratification, and contribute to better clinical management and outcomes in children with dengue fever.

Limitations of study

1. The study was conducted at a single tertiary care center, which may limit the generalizability of the findings to other populations.
2. The cross-sectional design precluded assessment of temporal changes and long-term hepatic outcomes.
3. The sample size of 100 patients, although adequate for the study objectives, may not fully represent the entire spectrum of pediatric dengue infection.
4. Follow-up liver function testing after recovery was not performed to evaluate the reversibility of hepatic abnormalities.
5. Viral serotyping of dengue virus was not undertaken; therefore, associations between specific serotypes and hepatic manifestations could not be assessed.
6. Quantitative viral load estimation was not available, preventing evaluation of its relationship with hepatic involvement.
7. Liver biopsy or advanced imaging modalities were not performed because of ethical and clinical considerations.
8. Some hepatic manifestations may have been influenced by nutritional status or unrecognized underlying conditions despite exclusion criteria.
9. Dynamic changes in coagulation parameters during hospitalization were not assessed serially.
10. The study evaluated hospitalized patients only; therefore, milder community-managed cases of dengue may have been underrepresented.

REFERENCES

1. Kulkarni MJ, Sarathi V, Bhalla V, Shivpuri D, Acharya U. Clinico-epidemiological profile of children hospitalized with dengue. Indian J Pediatr. 2010;77(10):1103-7.
2. Roy A, Sarkar D, Chakraborty S, Chaudhuri J. Clinical and laboratory profile of dengue fever in children. J Indian Med Assoc. 2013;111(9):626-9.
3. Chhina RS, Goyal O, Chhina DK, Goyal P, Kumar R, Puri S. Liver function tests in patients with dengue viral infection. Dengue Bull. 2008;32:110-7.
4. Trung DT, Thao LT, Hien TT, Hung NT, Vinh NN, Hien PT, et al. Liver involvement associated with dengue infection in adults in Vietnam. Am J Trop Med Hyg. 2010;83(4):774-80.
5. Seneviratne SL, Malavige GN, de Silva HJ. Pathogenesis of liver involvement during dengue viral infections. Trans R Soc Trop Med Hyg. 2006;100(7):608-14.
6. Parkash O, Almas A, Jafri SMW, Hamid S, Akhtar J, Alishah H. Severity of acute hepatitis and its outcome in patients with dengue fever in a tertiary care hospital Karachi, Pakistan. BMC Gastroenterol. 2010;10:43.
7. Souza LJ, Alves JG, Nogueira RMR, 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. 2007;11(2):192-6.
8. Srikiatkhachorn A, Krautrachue A, Ratanaprakarn W, Wongtapradit L, Nithipanya N, Kalayanarooj S, et al. Natural history of plasma leakage in dengue hemorrhagic fever: a serial ultrasonographic study. Pediatr Infect Dis J. 2007;26(4):283-90.
9. 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(3):265-70.
10. Zubair A, Ashraf M, Ayyub M, Fatima N, Khan A. Assessment of dengue fever severity through liver function tests. Pak J Med Sci. 2017;33(4):949-53.
11. Fernando S, Wijewickrama A, Gomes L, Punchihewa CT, Madusanka SDP, Dissanayake H, et al. Patterns and causes of liver involvement in acute dengue infection. BMC Infect Dis. 2016;16:319.
12. Samarawickrama A, Seneviratne SL, Jayasinghe S, Gunatilake SB. Hepatic dysfunction in dengue infection: clinical and laboratory predictors of severity. Ceylon Med J. 2012;57(2):67-71.