ORIGINAL ARTICLE

Assessment of hepatic dysfunction in patients with dengue fever: A cross-sectional study

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ABSTRACT

Background: Dengue fever, the most significant arthropod-borne illness, is transmitted by Aedes mosquitoes and caused by four DENV serotypes, leading to symptoms ranging from mild fever to severe forms like dengue hemorrhagic fever and dengue shock syndrome. This study aimed to assess hepatic dysfunction in patients with dengue fever and identify clinical and laboratory predictors of liver-related complications.

Material and Methods: This cross-sectional study was carried out over six months (June to December 2024) at the Department of Medicine, Bacha Khan Medical College and Mardan Medical Complex. Dengue-positive patients aged 17 years and above (confirmed via NS-1, IgG, or IgM) were enrolled using non-probability consecutive sampling. Patients under 17 or with malaria co-infection or chronic liver disease were excluded. Clinical evaluation included complete blood count, liver function tests, and abdominal ultrasound. Associations between liver complications and variables such as age, platelet count, bilirubin levels, and hepatomegaly were analyzed using SPSS version 29.

Results: Most patients (58.8%) were male, with a mean age of 34.2 ± 9.3 years. Hepatomegaly was present in 31.8%, while elevated ALT and bilirubin were noted in 38.3% and 45.9% respectively. ALT levels were significantly higher in patients with hepatomegaly (p=0.004).

Conclusion: The study underscores the need to monitor liver function in dengue patients, especially those with hepatomegaly, high bilirubin, and low platelets, as these indicate hepatic dysfunction and disease severity. Early detection and management may enhance outcomes.

Keywords: Dengue, Hepatic dysfunction, Liver enzymes, Hepatomegaly, Platelet count, Total bilirubin, ALT, Pakistan

BACKGROUND

Mosquitoes of the *Aedes* family transmit *dengue virus (DENV)* to humans leading to dengue fever (DF), the most significant arthropod-borne illness.[1] The disease can be caused by all four *DENV* serotypes viz. *DENV-1*, *DENV-2*, *DENV-3*, and *DENV-4*, which can manifest as a moderate self-limiting condition, DF, or as the more severe manifestations of the disease, dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS).[2] Different manifestations of DENV infection include conventional DF, acute febrile sickness, DHF, and asymptomatic infection.[3] World Health Organization (WHO) has classified the disease into dengue without warning signals, dengue with warning signs, and severe dengue based on severity of infection. This classification assists in correlating clinical severity with hepatic involvement and stratifying the patient population.[4]

Globally, *DENV* infection is becoming more common, which is a serious public health concern. Approximately 400 million people worldwide are afflicted with dengue each year.[5] In Pakistan, the disease is consistently prevalent in all four provinces throughout the year, although the number of cases tends to rise from August to October, during monsoon season. In 2022, the WHO reported that there were 25,932 cases overall in Pakistan, with 62 fatalities.[6] The National Vector Borne Disease Control Program recorded over 150 thousand laboratory-verified dengue cases during 2019.[7] Approximately 20–40% of people with dengue symptoms have hepatic problems.[8]

In patients with *DENV* infection, elevated alanine aminotransferase (ALT), aspartate aminotransferase (AST) and bilirubin suggest that hepatic function is often affected from mild inflammation to severe hepatitis or failure. Such markers play a key role in assessing the severity of dengue, as they are often raised in severe cases.[9] Hepatic dysfunction may be evident as modest damage with raised transaminases to overt hepatocyte damage. Which can be due to direct toxicity or altered host immunological response. Although there

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have been sporadic occurrences of fulminant hepatic failure, abnormalities in the transaminase levels are typically self-limiting and can assist in evaluating severity of the illness.[10,11] In this regard, regional studies elucidating the pattern of liver involvement are scarce.[12] Hence, our aim is to estimate the frequency and range of hepatic involvement by assessing clinical, biochemical and radiological profile in patients with *DENV* infection.

MATERIAL AND METHODS

An observational cross-sectional study design was employed to identify liver-related complications in patients with *DENV* infection and their association with various clinical and laboratory parameters. The study was conducted at the Department of Medicine, Bacha Khan Medical College & Mardan Medical Complex, Mardan, Pakistan from 1st June to 31st December 2024. This hospital was selected due to its accessibility to a significant number of dengue cases during the study period. A non-probability consecutive sampling technique was used to enroll participants in the study. All participants underwent a comprehensive clinical examination to assess their symptoms. As part of the routine diagnostic workup, each participant had blood samples taken for a complete blood count (CBC), dengue NS-1, IgG, IgM, and malaria parasite (MP) test. These tests were conducted to confirm the diagnosis of *DENV* infection and to rule out any co-infections such as malaria. The CBC results were used to evaluate hematological parameters, including hemoglobin levels, hematocrit, white blood cell count, and platelet count. All participants also underwent liver function tests (LFTs), including measurements of total bilirubin, ALT (SGPT), AST (SGOT), alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT) levels to assess hepatic dysfunction. An abdominal ultrasound was performed to detect liver-related complications such as hepatomegaly, splenomegaly, ascites, and pleural effusion, which are commonly associated with severe *DENV* infections.

The study adhered to ethical guidelines, ensuring participant privacy, voluntary consent, and the right to withdraw from the study at any time without penalty. All data were stored securely, and only authorized personnel had access to the research data. Ethical approval for this study was obtained from the Institutional Review Board of our institution (Ref # 690/BKMC - 18th April 2024).

A sample size of 85 was calculated using WHO sample size calculator, with a confidence level of 95%, margin of error of 7% and a 53.3% prevalence of hepatic complications in dengue cases.[13]

Patients who were ≥17years of age with laboratory-confirmed DENV infection were included in the study whereas, those who tested positive for MP using immunochromatographic method were excluded. Additionally, patients <17years of age and/or with a known history of chronic liver disease were also excluded.

Data were analyzed using Statistical Package for Social Sciences (SPSS) version 29. The quantitative variables, including age, number of symptoms, liver enzyme levels, hospitalization duration, and duration of liver-related symptoms, were presented as mean ± standard deviation. The distribution of categorical variables such as gender, residence area, history of travel, primary symptoms at diagnosis, pre-existing medical conditions, method of diagnosis, liver-related symptoms during dengue, imaging test findings, liver complications, treatment prescribed, specific liver treatment, and the impact of liver complications on infection severity were presented as frequencies and percentages. The independent sample t test was used for mean comparison of SGPT levels in patients with and without hepatomegaly. The p-value of ≤ 0.05 was considered as statistically significant.

RESULTS

Total 85 patients with *DENV* infection were examined during the study period. Age group of 26–35 years (28.2%) constituted most of the study population, followed by 17–25 years (21.2%) and 36—45 years (20%). There were more male patients (58.8%) compared to female patients (41.2%). Fever was present in all patients (100%), followed by body aches (82.4%), headache (57.6%), nausea or vomiting (54.1%), and abdominal discomfort (44.7%) (Table-I).

Hepatomegaly (31.8%) was the most frequent complication of the disease, followed by bleeding symptoms (24.7%), splenomegaly (22.4%), ascites (21.2%), and pleural effusion (18.8%). Whereas, 37.6% of patients showed no major complications (Table-II).

Regarding laboratory results, all patients were thrombocytopenic (100%) whereas, 23.5% had low

haemoglobin levels and 62.4% had leukopenia. ALT (SGPT) was elevated in 75.3% of the patients, ALP in 31.8%, GGT in 38.8% and total bilirubin in 45.9% respectively (Table-III).

Patients with hepatomegaly revealed a statistically significant difference in ALT (SGPT) levels compared to those without hepatomegaly (p=0.004).

Table-I: Demographic characteristics of dengue patients (n = 85).

Variable	Category	Frequency (n)
Age Group (years)	17–25	18 (21.2%)
	26–35	24 (28.2%)
	36–45	17 (20.0%)
	46–55	14 (16.5%)
	56–65	12 (14.1%)
Gender	Male	50 (58.8%)
	Female	35 (41.2%)
Presenting Complaints	Fever	85 (100.0%)
	Body Aches	70 (82.4%)
	Nausea/Vomiting	46 (54.1%)
	Abdominal Pain	38 (44.7%)
	Headache	49 (57.6%)

Table-II: Clinical Complications in Dengue Patients (n = 85).

Complication	Frequency (%)
Bleeding Manifestations	21 (24.7%)
Ascites	18 (21.2%)
Hepatomegaly	27 (31.8%)
Splenomegaly	19 (22.4%)
Pleural Effusion	16 (18.8%)
No Major Complications	32 (37.6%)

Table-III: Laboratory Investigations – CBC and LFTs.

Parameter	Mean ± SD	Reference Range	Abnormal in n (%)
CBC			
Hemoglobin (g/dL)	12.6 ± 1.8	12.0–16.0 (F), 13.0–17.0 (M)	20 (23.5%)
Hematocrit (%)	40.5 ± 4.3	36.0–47.0	18 (21.2%)
WBC (×10³/µL)	3.4 ± 1.1	4.0–11.0	53 (62.4%)
Platelets (×10³/µL)	78.0 ± 13.0	150.0–400.0	85 (100%)
LFTs			
Total Bilirubin (mg/dL)	1.9 ± 0.6	0.3–1.2	39 (45.9%)
SGPT (ALT) (U/L)	108.0 ± 25.0	<40.0	64 (75.3%)
ALP (U/L)	168.0 ± 28.0	40.0–130.0	27 (31.8%)
GGT (U/L)	82.0 ± 22.0	9.0–48.0 (F), 9.0–60.0 (M)	33 (38.8%)

DISCUSSION

Our study revealed that the proportion of patients with *DENV* infection without warning signs (55%) was greater than that of those with warning signs (35%) and severe dengue (10%). Most of our patients were males as compared to females, similar to several other regional studies.[11,12.13] One possible explanation for this might be the decrease in self-reporting of cases among women in Asian populations.

Mean age of our patients was 34.8 years, 50.8% of whom were between 18-30 years of age. Gandhi *et al.* found comparable mean age among patients with *DENV* infection [34.30±15.0 years].[14] Regarding disease severity, we found notable variation in mean values of LFTs. When compared to dengue with and without warning signs, severe cases had considerably higher mean SGOT, SGPT ALP, total bilirubin, and direct bilirubin as well as significantly lower albumin levels. Therefore, we inferred that rising transaminases, total and direct bilirubin, ALP levels, hypoalbuminemia, and A:G reversal are all indicators of disease severity. The baseline platelet count levels and SGOT/SGPT levels

were found to be significantly correlated negatively in a study.[15] The SGOT and SGPT levels rise when the platelet count falls, as demonstrated by the Pearson correlation between the two variables.[15] Another study found a linear correlation between the severity of the disease and the degree of SGOT elevation, with a significant p value of less than 0.05.[16] Our research revealed that SGOT and SGPT levels rose with rising dengue severity, as suggested by drop in platelet count as they are inversely related.[17]

Liver damage in *DENV* infection is caused by several pathways including direct virus cytopathic effects, immune mediated damage and hypo perfusion. Alterations observed in human post mortem research include micro vesicular steatosis, hepatic necrosis, Kupffer cell hyperplasia and death, Councilman bodies, and inflammatory cell infiltrates. Immunohistochemistry investigations have also revealed CD4+ and CD8+ T cell infiltration of the hepatic acini together with increased expression, hence implicating the Th1 cells. Irrespective of the existence of hypotension, dengue is also known to generate microcirculatory dysfunction owing venular or sinusoidal endothelial damage, which might lead hepatocyte ischaemia. In endemic locations, dengue can aggravate chronic liver disease and lead to acute on chronic liver failure.[18,19] Jamil *et al*. concluded that there is significant correlation between higher LFTs and severe dengue outcomes, highlighting their predictive utility in determining severity and directing treatment.[20]

The relationship between hypoalbuminemia and hepatic dysfunction in dengue has not been extensively researched. Gandhi *et al*. and Ahmad *et al*. found hypoalbuminemia in 12.9% and 16.5% of *DENV* cases, respectively.[14,21] Those with severe dengue had notably low mean serum albumin, according to our findings. In this regard, a study found that the average blood albumin levels in people with severe dengue were significantly lower than those found in the general population.[22] According to another study, those who had died from dengue had a stronger correlation with severe dengue.[23]

Hypoalbuminemia was also more frequent in our patients who arrived in shock. Patients with severe dengue (27.3%) had a substantially greater A: G ratio of less than 1 than those with dengue with warning signals (7.3%) and dengue without warning signs (1.6%). This may be explained by the difference in molecular size of albumin and globulin. Albumin, a smaller molecule leaks out more readily than globulin during the early stages of the disease, therefore reversing the A:G ratio. Patients with shock had much higher mean SGOT, SGPT, and ALP levels than those without shock in our study. Although it has been suggested that hepatic impairment could occur even without hypotension because of microcirculatory malfunction, the damage seems to be much higher in the context of shock.[24,25]

Significant liver dysfunction was indicated by the severe hepatic abnormalities seen in our patients. Elevated liver enzymes, which indicate hepatocellular injury, and indications of biliary involvement or hepatic stress were common findings. Additionally, hyperbilirubinemia was common, suggesting that hemolysis or liver injury was the likely reason of defective bilirubin metabolism. These findings emphasize the necessity of routinely checking liver function in patients with dengue in order to properly manage any potential consequences. The result has significant clinical implications since it shows that patients with severe illness had greater levels of SGPT, bilirubin, ALP, and GGT. Therefore, severely deranged LFTs may indicate a severe case of dengue. The development of DHF is suggested to be positively predicted by liver injury.[26] According to Chhina *et al*., liver dysfunction is a predictor of complications and a sign of the severity of disease.[27]

Patients with no bleeding manifestations had relatively worse prognosis than those with bleeding symptoms in terms of SGOT, SGPT, ALP, and serum bilirubin. In patients with raised liver enzymes, greater mean haemoglobin and haematocrit indicated hemo-concentration that was statistically significant (p=0.028 and p=0.04, respectively). A study reported significant treatment-related problems in patients with severe hepatic dysfunction with bleeding occurring in 50% of patients.[28] The study discovered that every warning sign aside from cutaneous and mucosal bleeding (epistaxis and gum bleeding) was strongly correlated with severe dengue. Notably, severe illness was substantially linked to gastrointestinal bleeding or melena.[29] Moreover, severe dengue is associated with shock due to severe plasma leakage which leads to tachycardia, reduced pulse (<20 mmHg), delayed refill of capillaries and low blood pressure. These patients may also develop

pulmonary fluid overload, serious bleeding or damage to the CNS, liver and heart.[30]

Our study had a few limitations. Firstly, it was not possible to establish causal links between dengue-related factors and hepatic dysfunction due to the cross-sectional study design. Secondly, possible confounders including age, sex, and concomitant diseases were not considered when adjusting for findings such as the association between hepatomegaly and high SGPT levels, which might have affected the outcomes. Lastly, our study lacked follow-up information of the patients, which makes it difficult to evaluate how hepatic dysfunction resolves or advances and how it affects the prognosis of patients over a long period of time. To address these limitations, future studies incorporating longitudinal designs and confounding variable modifications are required.

CONCLUSION.

Our findings suggest that older patients, with lower platelet counts, and those exhibiting hepatomegaly or elevated bilirubin are more likely to experience severe hepatic involvement as a result of dengue fever. These markers can help clinicians identify patients at higher risk for complications and guide monitoring and management strategies. The results also emphasize the need for close monitoring of liver function and hematological parameters to improve patient outcomes.

CONFLICT OF INTEREST

None

GRANT SUPPORT & FINANCIAL DISCLOSURE

Declared none

AUTHOR CONTRIBUTION

Fazli Rabbi: Concept and design of study, acquisition, analysis and interpretation of data, drafting and revising article critically for important intellectual content, final approval, agreement to be accountable for all aspects of the work

Mohammad Sohrab Khan: Design of study, acquisition, analysis and interpretation of data, drafting and revising article critically for important intellectual content, final approval, agreement to be accountable for all aspects of the work

Shah Zeb: Design of study, acquisition, analysis and interpretation of data, drafting and revising article critically for important intellectual content, final approval, agreement to be accountable for all aspects of the work

Murad Ali: Analysis and interpretation of data, drafting and revising article critically for important intellectual, content final approval, agreement to be accountable for all aspects of the work

Waleed Ahmad Khan and Asim Ali: Analysis and interpretation of data, drafting and revising article critically for important intellectual content, final approval, agreement to be accountable for all aspects of the work

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