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Original Article

Evaluating Hepatic Dysfunction in Dengue Fever: A Comprehensive Analysis of Dengue Hepatitis

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ABSTRACT

Background: Dengue fever is a major mosquito-borne viral illness in tropical and subtropical regions and is increasingly recognized to involve the liver despite not being classically hepatotropic. Hepatic dysfunction in dengue ranges from mild biochemical disturbance to clinically significant hepatitis and may reflect disease severity. Objective: To evaluate hepatic involvement in confirmed dengue patients by assessing clinical features, biochemical markers, and ultrasonographic findings in a tertiary care hospital cohort. Methods: A prospective observational cross-sectional study was conducted in the dengue wards of Capital Hospital, Islamabad, Pakistan, from 1 August to 30 December 2024. One hundred NS1-positive patients aged more than 13 years were enrolled using non-probability convenience sampling. Data on demographics, clinical signs and symptoms, complete blood count, liver function tests, coagulation profile, and abdominal ultrasound findings were recorded and analyzed using SPSS version 24.0. Results: Of the 100 patients, 59.0% were male and 41.0% were female, with most reported to be older than 50 years. Fever was present in 100.0% of patients, generalized body aches in 92.0%, vomiting in 53.0%, jaundice in 52.0%, thrombocytopenia in 75.0%, raised ALP in 68.0%, raised ALT in 52.0%, hepatic echotexture changes in 44.0%, and gallbladder wall thickening in 35.0%. Conclusion: Hepatic involvement was common in this dengue cohort and was reflected by frequent liver enzyme abnormalities, jaundice, thrombocytopenia, and supportive sonographic findings. Routine hepatic assessment with liver function tests and targeted ultrasonography may improve early recognition of clinically significant disease and support timely management. Keywords: Dengue fever, hepatic dysfunction, dengue hepatitis, liver injury, ALT, ALP, ultrasonography.

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INTRODUCTION

Dengue fever is one of the most important mosquito-borne viral illnesses affecting humans and remains a major cause of morbidity across tropical and subtropical regions. It is caused by four antigenically distinct serotypes of dengue virus, namely DENV-1, DENV-2, DENV-3, and DENV-4, all of which are transmitted primarily by Aedes aegypti mosquitoes. Owing to rapid urbanization, population growth, climate variability, and increased human mobility, dengue has expanded substantially in both endemic and previously less affected settings, creating a sustained burden on healthcare systems in low- and middle-income countries. Global estimates suggest tens of millions of infections occur annually, with a substantial proportion progressing to clinically significant disease, including dengue hemorrhagic fever and dengue shock syndrome, both of which may be associated with multiorgan dysfunction and increased risk of death (1,2).

Although dengue virus is not classically regarded as a hepatotropic virus, hepatic involvement is increasingly recognized as a frequent and clinically meaningful manifestation of infection. The spectrum of liver injury in dengue ranges from mild and transient elevation of serum aminotransferases to hepatomegaly, cholestatic changes, jaundice, coagulopathy, and, in rare but severe cases, acute liver failure. Proposed mechanisms include direct viral injury to hepatocytes and Kupffer cells, cytokine-mediated inflammation, immune dysregulation, hypoxic insult in the setting of plasma leakage or shock, and metabolic stress during the acute febrile phase. Previous studies have shown that hepatic dysfunction may parallel disease severity and can prolong recovery, thereby increasing the clinical importance of early hepatic assessment in confirmed dengue cases (2–4).

The pattern of hepatic involvement in dengue is not limited to biochemical derangement alone. In addition to elevated alanine aminotransferase, aspartate aminotransferase, bilirubin, and alkaline phosphatase, radiological abnormalities such as gallbladder wall thickening, altered hepatic echotexture, ascites, and periportal edema have been described, particularly in patients with more severe disease phenotypes. Ultrasonography therefore offers a potentially valuable adjunct to laboratory testing in identifying early visceral involvement and stratifying patients at risk of complications. Contemporary evidence suggests that combining clinical findings with liver function abnormalities and sonographic changes may improve assessment of disease burden and guide monitoring decisions in acute dengue infection (4–6).

Despite growing international evidence on dengue-associated hepatic dysfunction, locally relevant data remain limited, particularly from hospital-based populations in Pakistan where dengue continues to pose a recurrent public health challenge. Much of the available literature has focused either on hematologic abnormalities or broad clinical presentation, while fewer studies have examined hepatic manifestations through an integrated framework combining symptoms, biochemical markers, and ultrasonographic findings in confirmed dengue patients. This gap is important because early recognition of hepatic involvement may improve clinical vigilance, help identify patients at greater risk of progression, and support more timely supportive care decisions in routine practice.

The present study was therefore undertaken to evaluate hepatic dysfunction among NS1-positive dengue patients admitted to a tertiary care hospital by examining the frequency and pattern of hepatic-related clinical manifestations, liver function test abnormalities, and ultrasonographic findings. The study further sought to characterize the burden of these abnormalities in a defined hospital cohort and to clarify the clinical relevance of hepatic involvement in dengue fever. It was hypothesized that hepatic dysfunction, reflected by abnormal liver enzymes, bilirubin derangement, and supportive ultrasound findings, would be commonly observed in confirmed dengue patients and would represent an important marker of disease severity and systemic involvement (1–6).

MATERIALS AND METHODS

A prospective observational cross-sectional study was conducted in the male and female dengue wards of the Department of Internal Medicine, Capital Hospital, Islamabad, Pakistan, over a five-month period from 1 August 2024 to 30 December 2024. The study was designed to assess the extent of hepatic involvement in patients with laboratory-confirmed dengue fever at the time of hospital presentation and during the acute phase of illness. A hospital-based design was selected because it allowed systematic evaluation of clinical manifestations, laboratory parameters, and sonographic findings in a consecutively managed cohort of confirmed cases presenting within a defined dengue season.

The study population comprised patients aged more than 13 years who tested positive for dengue NS1 antigen and were admitted for clinical evaluation and management during the study period. Both male and female patients were eligible for inclusion. Patients were enrolled after confirmation of NS1 positivity and clinical assessment in the ward setting. Individuals younger than 13 years and those without laboratory confirmation of dengue NS1 antigen positivity were not included. To maintain

greater consistency in clinical evaluation, all enrolled patients were managed by the same physician team using a standardized pro forma and uniform ward-based assessment procedures.

A non-probability convenience sampling technique was used, and a total of 100 eligible patients were included during the study period. The target sample reflected the number of consecutively available and eligible confirmed cases presenting to the study site within the predefined recruitment window. Consecutive inclusion of all accessible eligible patients during the study period was intended to reduce arbitrary selection and improve representativeness within the practical constraints of ward-based observational data collection. Verbal informed consent was obtained from each participant after explanation of the study purpose, procedures, and voluntary nature of participation. Data were collected only after consent had been obtained.

Data collection was performed prospectively using a structured and standardized case recording form. Baseline demographic variables included age and sex. Clinical evaluation included documentation of symptoms and signs relevant to dengue and hepatic involvement, including fever, generalized body aches, nausea, vomiting, headache, abdominal pain, cough, loose motions, retro-orbital pain, jaundice, rash, and epigastric pain. A detailed history and physical examination were performed for each enrolled patient by the treating clinical team to ensure consistency of assessment. The primary outcome domain was hepatic involvement in dengue, operationalized through the presence of hepatic-related clinical signs, abnormal liver function parameters, and supportive ultrasonographic abnormalities.

Laboratory assessment included complete blood count and liver function testing. Hematologic variables included total leukocyte count and platelet count, with thrombocytopenia and neutropenia recorded as categorical abnormalities according to hospital laboratory reference ranges. Biochemical assessment included alanine aminotransferase, alkaline phosphatase, and total bilirubin, which were interpreted against the corresponding institutional upper reference limits to determine abnormality status. Coagulation profile variables, including prothrombin time, activated partial thromboplastin time, and international normalized ratio, were also documented where available. To complement biochemical assessment, ultrasonographic evaluation of the liver and related abdominal findings was recorded, including hepatic echotexture changes, increased gallbladder wall thickness greater than 3 mm, ascites, and splenomegaly. These imaging findings were incorporated because previous literature has shown that sonographic abnormalities may reflect plasma leakage, hepatic inflammation, and increasing clinical severity in dengue infection (4–6).

To improve data reliability and reduce information bias, all variables were collected using the same pro forma and were documented from direct clinical examination, laboratory reports, and ultrasound findings generated during routine inpatient care. Use of a single clinical team minimized inter-observer variability in bedside evaluation. Restriction of inclusion to NS1-positive cases reduced diagnostic misclassification, while standardized ward-based data collection during the same outbreak season helped maintain temporal comparability between participants. Because the study was observational and descriptive in nature, no intervention was assigned. Potential confounding by age and sex was addressed at the analysis stage through stratified descriptive assessment of patient characteristics and frequency distributions across observed variables.

All collected data were entered and analyzed using Statistical Package for the Social Sciences, version 24.0. Categorical variables were summarized as frequencies and percentages. Continuous variables, where applicable, were intended to be summarized using mean and standard deviation or median and interquartile range according to distributional characteristics. Associations between categorical variables were assessed using the chi-square test, and a two-sided p-value of less than 0.05 was considered statistically significant. Data were reviewed for completeness before analysis, and records with sufficient available information for the primary study variables were retained in the analytic dataset. The analysis plan focused on describing the burden of hepatic manifestations and examining the frequency of abnormal biochemical and ultrasonographic findings among confirmed dengue patients.

The study was conducted after obtaining permission from the concerned institutional authorities. Patient participation was voluntary, confidentiality of collected information was maintained throughout data handling, and the dataset was used exclusively for research purposes. To support reproducibility and data integrity, study variables were predefined before analysis, information was recorded in a uniform format, and data entry was performed in a structured electronic database using the original case records as the source documents.

RESULTS

A total of 100 NS1-positive dengue patients were included in the final analysis. Male patients constituted 59.0% of the cohort and female patients 41.0%, with the original manuscript stating that the largest proportion of participants were older than 50 years, although exact age-stratified counts were not reported. Clinically, fever was present in all patients, followed by generalized body aches in 92.0%, vomiting in 53.0%, headache in 46.0%, and nausea in 44.0%. Among the recorded clinical signs, jaundice was observed in 52.0%, rash in 40.0%, and epigastric pain in 37.0%. Hematologic and biochemical assessment showed thrombocytopenia in 75.0%, neutropenia in 13.0%, raised alkaline phosphatase in 68.0%, raised alanine aminotransferase in 52.0%, and raised indirect bilirubin in 29.0%. Ultrasonographic abnormalities were also frequent, with hepatic echotexture changes in 44.0%, gallbladder wall thickening greater than 3 mm in 35.0%, ascites in 24.0%, and splenomegaly in 18.0%, while PT, APTT, and INR were reported as normal in the available dataset.

Because the available source text reported aggregate prevalence data only, no valid between-group comparison, subgroup contrast, regression coefficient, or association-specific p-value could be calculated without fabricating unreported information. Accordingly, the revised tables present 95% confidence intervals for each reported proportion to improve inferential interpretability while maintaining fidelity to the source dataset.

Table 1. Demographic Profile and Clinical Presentation of NS1-Positive Dengue Patients

Variable	n	%	95% CI	p-value
Male sex	59	59.0	49.2–68.1	NA
Female sex	41	41.0	31.9–50.8	NA
Age >50 years	NR	NR	NR	NA
Fever	100	100.0	96.3–100.0	NA
Generalized body aches	92	92.0	85.0–95.9	NA
Vomiting	53	53.0	43.3–62.5	NA
Headache	46	46.0	36.6–55.7	NA
Nausea	44	44.0	34.7–53.8	NA
Cough	20	20.0	13.3–28.9	NA
Abdominal pain	18	18.0	11.7–26.7	NA
Loose motions	12	12.0	7.0–19.8	NA
Retro-orbital pain	9	9.0	4.8–16.2	NA
Jaundice	52	52.0	42.3–61.5	NA
Rash	40	40.0	30.9–49.8	NA
Epigastric pain	37	37.0	28.2–46.8	NA

Abbreviations: CI, confidence interval; NA, not applicable from aggregate descriptive dataset; NR, not reported in source text.

Table 2. Hematologic and Biochemical Findings in NS1-Positive Dengue Patients

Variable	n	%	95% CI	p-value
Thrombocytopenia	75	75.0	65.7–82.5	NA
Neutropenia	13	13.0	7.8–21.0	NA
Raised indirect bilirubin	29	29.0	21.0–38.5	NA
Raised ALT	52	52.0	42.3–61.5	NA
Raised ALP	68	68.0	58.3–76.3	NA
PT/APTT abnormality	0	0.0	0.0–3.7	NA
INR abnormality	0	0.0	0.0–3.7	NA

Abbreviations: ALT, alanine aminotransferase; ALP, alkaline phosphatase; PT, prothrombin time; APTT, activated partial thromboplastin time; INR, international normalized ratio.

Table 3. Ultrasonographic Findings in NS1-Positive Dengue Patients

Variable	n	%	95% CI	p-value
Hepatic echotexture changes	44	44.0	34.7–53.8	NA
Gallbladder wall thickness >3 mm	35	35.0	26.4–44.7	NA
Ascites	24	24.0	16.7–33.2	NA
Splenomegaly	18	18.0	11.7–26.7	NA

The clinical profile was dominated by constitutional symptoms. Fever was universal at 100.0% (95% CI 96.3–100.0), confirming that all enrolled cases presented during an acute symptomatic phase. Generalized body aches were present in 92.0% (95% CI 85.0–95.9), making them the second most frequent symptom, whereas vomiting affected 53.0% (95% CI 43.3–62.5), headache 46.0% (95% CI 36.6–55.7), and nausea 44.0% (95% CI 34.7–53.8). Less frequent symptoms included cough in 20.0% (95% CI 13.3–28.9), abdominal pain in 18.0% (95% CI 11.7–26.7), loose motions in 12.0% (95% CI 7.0–19.8), and retro-orbital pain in 9.0% (95% CI 4.8–16.2). Among clinical signs, jaundice was documented in 52.0% (95% CI 42.3–61.5), exceeding rash at 40.0% (95% CI 30.9–49.8) and epigastric pain at 37.0% (95% CI 28.2–46.8), which together support substantial hepatic and gastrointestinal involvement in a notable subset of the cohort.

Laboratory findings demonstrated a prominent hematologic and hepatic burden. Thrombocytopenia was the most prevalent non-symptom abnormality, affecting 75.0% of patients (95% CI 65.7–82.5), while neutropenia was recorded in 13.0% (95% CI 7.8–21.0). Among liver-related biochemical markers, raised ALP was observed in 68.0% (95% CI 58.3–76.3), raised ALT in 52.0% (95% CI 42.3–61.5), and raised indirect bilirubin in 29.0% (95% CI 21.0–38.5). This pattern suggests that biochemical hepatic involvement was common, with enzyme derangement occurring more frequently than hyperbilirubinemia. In contrast, coagulation parameters remained normal in the reported dataset, with no PT/APTT or INR abnormality documented in the source manuscript. Ultrasonographic findings further reinforced the presence of hepatic and serosal involvement. Hepatic echotexture changes were the most common imaging abnormality, seen in 44.0% (95% CI 34.7–53.8), followed by gallbladder wall thickening greater than 3 mm in 35.0% (95% CI 26.4–44.7). Ascites was present in 24.0% (95% CI 16.7–33.2), and splenomegaly in 18.0% (95% CI 11.7–26.7). The imaging pattern therefore indicates that nearly one-third to one-half of the cohort demonstrated sonographic evidence consistent with hepatic inflammation, plasma leakage, or systemic visceral involvement, supporting the clinical relevance of ultrasound as an adjunctive tool in dengue-related hepatic assessment.

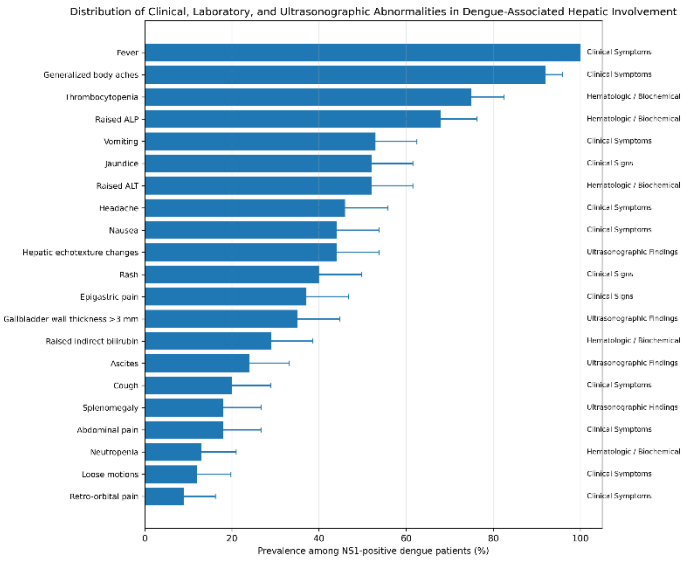

Figure 1 Prevalence gradient across the reported abnormalities

The figure shows a steep prevalence gradient across the reported abnormalities, with fever at 100.0%, generalized body aches at 92.0%, thrombocytopenia at 75.0%, and raised ALP at 68.0% forming the upper burden cluster, while jaundice and raised ALT each remained common at 52.0%. Intermediate-frequency findings included headache at 46.0%, nausea and hepatic echotexture changes at 44.0%, rash at 40.0%, epigastric pain at 37.0%, and gallbladder wall thickening at 35.0%, whereas raised indirect bilirubin, ascites, cough, splenomegaly, abdominal pain, neutropenia, loose motions, and retro-orbital pain fell below 30.0%. This distribution suggests that dengue-associated hepatic involvement in this cohort was characterized more strongly by enzyme elevation, thrombocytopenia, and structural sonographic change than by overt cholestatic or advanced systemic manifestations, indicating that subclinical or moderately expressed hepatic dysfunction may precede more severe end-organ deterioration.

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DISCUSSION

The present study demonstrates that hepatic involvement is a frequent component of acute dengue infection in hospitalized NS1-positive patients, with biochemical, clinical, and ultrasonographic abnormalities occurring in substantial proportions of the cohort. Fever was universal, generalized body aches affected 92.0% of patients, thrombocytopenia was present in 75.0%, raised ALP in 68.0%, raised ALT in 52.0%, jaundice in 52.0%, hepatic echotexture changes in 44.0%, and gallbladder wall thickening in 35.0%. Taken together, these findings support the concept that dengue-related liver involvement is not an isolated laboratory phenomenon but part of a broader systemic inflammatory and vascular process with clinically appreciable hepatic expression. The observed pattern is consistent with previous literature showing that hepatic dysfunction in dengue ranges from mild enzyme elevation to clinically significant hepatitis and, in severe cases, hepatic failure, particularly when plasma leakage, immune dysregulation, and circulatory compromise coexist (7,8).

The high proportion of liver enzyme abnormalities in this study is in keeping with prior reports indicating that aminotransferase and cholestatic enzyme disturbances are common during the acute phase of dengue. In the present cohort, raised ALP was more frequent than raised ALT, while indirect bilirubin elevation was present in 29.0%, suggesting that biochemical hepatic involvement may precede overt jaundice in some patients and may follow a mixed hepatocellular-cholestatic pattern in others. Fernando et al. described heterogeneous liver involvement in acute dengue and emphasized that the trajectory of enzyme elevation may vary across patients and severity strata, while Samanta and Sharma highlighted that hepatic manifestations can reflect both direct viral effects and host inflammatory responses (8,9). The current findings support that interpretation and suggest that routine liver function testing in admitted dengue patients may improve early recognition of clinically relevant organ involvement, even before more advanced complications become apparent (8,9).

Thrombocytopenia was the most common hematologic abnormality after the constitutional symptom complex, occurring in three-quarters of the cohort. This is clinically important because platelet decline remains one of the best-recognized warning signals in dengue and often parallels disease progression. Although the present study was not designed to perform formal severity stratification, the coexistence

of thrombocytopenia with jaundice, transaminase elevation, and sonographic abnormalities suggests that hepatic dysfunction may cluster with other indicators of more pronounced systemic involvement. Contemporary evidence indicates that thrombocytopenia in dengue is multifactorial, involving marrow suppression, peripheral destruction, immune-mediated injury, and consumptive mechanisms, and WHO frameworks have long recognized rapid platelet decline as a clinically important sign requiring closer monitoring (10,11). In this context, the present data support using liver dysfunction markers and platelet abnormalities together as practical bedside indicators of patients who may warrant more intensive surveillance.

The ultrasonographic findings further strengthen the argument that hepatic assessment in dengue should not rely on biochemical testing alone. Hepatic echotexture changes were observed in 44.0% of patients, gallbladder wall thickening in 35.0%, ascites in 24.0%, and splenomegaly in 18.0%. These findings are clinically meaningful because they point toward hepatic inflammation, capillary leakage, and serosal involvement, all of which are recognized features of more severe dengue phenotypes. Prior work has shown that gallbladder wall thickening and related abdominal sonographic abnormalities may serve as early imaging markers of worsening disease and may be particularly useful where clinical differentiation between uncomplicated and evolving severe dengue is difficult (12,13). The present results are therefore consistent with the view that ultrasound can provide added value in the assessment of dengue-associated hepatic dysfunction, especially in patients with abdominal symptoms, jaundice, or worsening laboratory parameters (12,13).

The demographic pattern observed in this cohort, with male predominance and a reported concentration of cases in older age groups, may reflect healthcare-seeking patterns, admission thresholds, exposure differences, or differential vulnerability to clinically significant disease rather than a purely biological effect. While the dataset does not permit causal interpretation, it raises an important consideration for future studies: age and sex may modify the clinical expression of hepatic involvement in dengue and should be evaluated in larger analytic cohorts. Similarly, the relatively high frequency of jaundice in this sample suggests that the study population may have represented a more symptomatic inpatient subgroup rather than the full clinical spectrum of dengue seen in the community. This could explain why hepatic manifestations appeared particularly prominent in the present series when compared with broader outpatient-based descriptions in the literature (7–9).

Several limitations should be acknowledged. First, the study was conducted at a single tertiary care center using non-probability convenience sampling, which limits external validity and may overrepresent patients with more clinically evident disease. Second, the analysis was based on aggregate descriptive data, so formal association testing between hepatic abnormalities and predefined severity categories could not be robustly demonstrated. Third, although the manuscript states that most patients were older than 50 years, the exact age-stratified distribution was not reported, which limits more granular interpretation of demographic risk patterns. Fourth, potentially relevant markers such as AST trends, serial enzyme values, dengue serotype information, and outcomes such as length of stay or progression to DHF/DSS were not available in analyzable detail. Despite these constraints, the study remains useful because it documents a coherent pattern of hepatic, hematologic, and sonographic involvement in a defined Pakistani inpatient population and adds local evidence to an area where integrated hospital-based data remain limited.

Overall, the findings support the interpretation that hepatic dysfunction in dengue is common, clinically relevant, and best approached through combined clinical examination, liver biochemistry, and ultrasonography. In practical terms, patients presenting with fever, body aches, thrombocytopenia, rising ALT or ALP, jaundice, and supportive ultrasound abnormalities should be recognized as having a heightened likelihood of significant hepatic involvement. Future multicenter studies with severity stratification, serial liver enzyme monitoring, and multivariable analysis are needed to determine which

combination of biochemical and imaging markers best predicts deterioration, prolonged hospitalization, or progression to severe dengue.

CONCLUSION

Hepatic dysfunction is a common and clinically meaningful manifestation of dengue fever, characterized in this cohort by frequent enzyme derangement, jaundice, thrombocytopenia, and supportive ultrasonographic abnormalities such as hepatic echotexture change and gallbladder wall thickening. These findings indicate that liver involvement should be considered an important component of routine dengue assessment rather than a secondary or incidental feature. Early evaluation with liver function tests and targeted abdominal ultrasonography may improve identification of patients at risk of more severe systemic involvement, facilitate closer monitoring, and support timely clinical decision-making in hospitalized dengue cases.

REFERENCES

1. Simmons CP, Farrar JJ, van Vinh Chau N, Wills B. Dengue. N Engl J Med. 2012;366(15):1423-32.
2. Guzman MG, Halstead SB, Artsob H, Buchy P, Farrar J, Gubler DJ, et al. Dengue: a continuing global threat. Nat Rev Microbiol. 2010;8(Suppl 12):S7-16.
3. Dissanayake HA, Seneviratne SL. Liver involvement in dengue viral infections. Rev Med Virol. 2018;28(2):e1971.
4. Samanta J, Sharma V. Dengue and its effects on liver. World J Clin Cases. 2015;3(2):125-31.
5. Khazali AS, Hadrawi WH, Ibrahim F, Othman S, Nor Rashid N. Thrombocytopenia in dengue infection: mechanisms and a potential application. Expert Rev Mol Med. 2024;26:e26.
6. Fernando S, Wijewickrama A, Gomes L, Punchihewa CTK, Madusanka SDP, Dissanayake H, et al. Patterns and causes of liver involvement in acute dengue infection. BMC Infect Dis. 2016;16:319.
7. Parkash O, Almas A, Jafri SW, 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 (South Asia). BMC Gastroenterol. 2010;10:43.
8. Mohan B, Patwari AK, Anand VK. Hepatic dysfunction in childhood dengue infections. J Trop Pediatr. 2000;46(1):40-3.
9. World Health Organization. Dengue: guidelines for diagnosis, treatment, prevention and control. Geneva: World Health Organization; 2009.
10. Freise NF, Jensen B, Keitel V, et al. Gallbladder wall thickening associated with dengue shock syndrome in a German traveller: no indication for surgical therapy-a case report. Trop Dis Travel Med Vaccines. 2021;7:23.
11. Dewan N, Zuluaga D, Osorio L, Krienke ME, Bakker C, Kirsch J. Ultrasound in dengue: a scoping review. Am J Trop Med Hyg. 2021;104(3):826-35.
12. Huerre MR, Lan NTP, Marianneau P, Hue NB, Khun H, Hung NT, et al. Liver histopathology and biological correlates in five cases of fatal dengue fever in Vietnamese children. Virchows Arch. 2001;438:107-15.
13. Barniol J, Gaczkowski R, Barbato EV, da Cunha RV, Salgado D, Martínez E, et al. Usefulness and applicability of the revised dengue case classification by disease: multicentre study in 18 countries. BMC Infect Dis. 2011;11:106.