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SPECTRUM OF LIVER DYSFUNCTIONS IN DENGUE INFECTION

General Medicine

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ABSTRACT

Background: Dengue fever is the most common arboviral infection worldwide, particularly prevalent in tropical regions like India. It poses significant clinical and economic burdens due to its potential to cause severe disease forms, including liver dysfunctions. Liver involvement in dengue can range from mild elevations in liver enzymes to severe hepatic failure, affecting the clinical outcomes. **Objective:** This study aims to delineate the spectrum of liver dysfunctions in adults diagnosed with dengue fever in a regional Indian setting, emphasizing the prevalence, patterns, and severity of hepatic dysfunction. **Methods:** We conducted a hospital-based, retrospective analysis at a tertiary care center in India. The study included adults over 18 years diagnosed with dengue via immunochromatographic tests and, in some cases, IgM ELISA. We excluded patients with other causes of liver disease. Data on liver function tests, clinical severity, and outcomes were analyzed for 281 inpatients, with a focus on those exhibiting significant laboratory abnormalities. **Results:** The study found high frequencies of liver enzyme abnormalities, particularly transaminases, in dengue patients. Severe liver involvement was correlated with more advanced stages of dengue, such as dengue haemorrhagic fever (DHF) and dengue shock syndrome (DSS). Statistical analysis showed significant differences in liver function parameters between uncomplicated dengue fever and its severe forms. **Conclusion:** Hepatic dysfunction is a common complication of dengue fever and is more pronounced in severe disease forms. Elevated liver enzymes, particularly transaminases, are prevalent and can serve as early markers for disease severity. Understanding these patterns helps in the prognostication and management of dengue, emphasizing the need for careful monitoring of liver function in infected individuals.

KEYWORDS

Dengue Fever, Liver Dysfunction, Transaminase Elevation, Hepatic Complications, Clinical Severity

INTRODUCTION:

At present, dengue fever stands as the most frequently occurring arboviral infection across the globe, making it a leading cause of fever and hospitalization in numerous tropical regions [1,2]. In India and other nations with tropical climates, dengue fever is notably one of the primary causes of febrile illnesses. Current estimates suggest that approximately 2.5 billion individuals residing in over 100 endemic countries are at risk of infection. In addition, large numbers of international travelers visiting these endemic areas also face considerable exposure to the virus. Various elements have contributed to the ongoing increase in the disease's prevalence within tropical zones as well as its spread worldwide. Key factors include inadequate sanitation practices, compromised healthcare infrastructures, and the exponential growth in global travel—each factor fostering an environment conducive to sustained viral transmission [1,2].

Dengue virus was initially isolated in India in 1945, marking a pivotal event in understanding the disease's presence in the country. The earliest confirmed evidence of dengue fever in India emerged in 1956 from Vellore district in Tamil Nadu. Subsequently, the first recorded outbreak of dengue haemorrhagic fever (DHF) occurred in 1963 in Calcutta (now Kolkata), West Bengal, underscoring the virus's capacity to cause severe manifestations [3]. Today, dengue is pervasive throughout India, affecting both densely populated urban centres and more remote rural areas. According to recent data, as of 25 October 2015, there were 64,058 reported cases and 135 fatalities attributable to dengue in India. Additionally, economic analyses have revealed a significant financial burden; a particular study estimated the country's direct annual medical costs for dengue at approximately US $548 million. Within these expenditures, outpatient (ambulatory) treatment constituted 67% of cases and contributed to 18% of costs, while hospitalized patients, making up 33% of cases, accounted for a disproportionately high 82% of the expenses [4]. These figures highlight not only the clinical but also the substantial economic impact that dengue exerts on healthcare systems. Clinically, most individuals infected with dengue virus remain either completely asymptomatic (about 80%) or exhibit mild nonspecific symptoms, predominantly low-grade fever. However, a smaller subset of patients (approximately 5%) may progress to more severe forms of the disease, and an even smaller fraction can experience fatal outcomes if not managed promptly [5,6]. Among the notable complications of dengue infection is hepatic involvement, frequently manifesting as disturbances in liver function tests. These hepatic abnormalities range widely—from mild elevations in transaminases and bilirubin levels to clinical pictures resembling acute viral hepatitis or even acute liver failure. For example, markedly elevated transaminase levels (over 10 times the upper limit of normal) and deranged coagulation parameters (such as an increased INR) may be observed. While liver impairment in many dengue cases remains subclinical and without overt jaundice, more severe instances can lead to pronounced jaundice and acute liver failure, particularly in individuals with severe dengue forms [7]. The pathogenesis of this hepatic involvement is multifactorial. It may result from direct viral invasion and injury to hepatocytes, or it may arise indirectly through an abnormal immune response mounted by the host against the virus [8]. Furthermore, factors such as pre-existing liver conditions and the concurrent use of drugs with hepatotoxic potential may exacerbate liver injury [9]. Importantly, elevated liver enzymes in dengue patients not only serve as early indicators of infection but can also be employed as predictive markers for gauging disease severity [10]. Despite the escalating number of affected patients annually, the existing body of research on dengue-related liver dysfunction remains limited.

Aims & Objectives:

Aim: To investigate the extent of liver involvement and its clinical significance in adults with dengue fever, particularly focusing on the prevalence, patterns, and severity of hepatic dysfunction in a regional Indian population.

Objectives:

1. To determine the prevalence and degree of liver enzyme abnormalities in adults diagnosed with dengue fever.
2. To assess the relationship between the extent of hepatic dysfunction and the clinical severity of dengue infection.
3. To identify potential factors—such as underlying liver conditions or medication use—that may influence the magnitude and progression of liver injury in dengue-affected individual

MATERIALS AND METHODS: This hospital-based, retrospective study was carried out at a tertiary care centre in India, encompassing records from January 2015 to May 2017. All inpatients above 18 years of age diagnosed with dengue infection, confirmed either by a rapid

immunochromatographic test (ICT) for NS1 antigen or IgM antibodies and, in some instances, additionally by IgM ELISA, were included. Patients with pre-existing or concurrent liver diseases attributable to any other cause (such as chronic liver disease of known etiology, recent or co-existing acute viral hepatitis, malaria, typhoid, leptospirosis, intake of hepatotoxic drugs, alcoholic or non-alcoholic fatty liver disease, Wilson's disease, autoimmune hepatitis, or heart failure) were excluded to ensure that any hepatic involvement could be solely attributed to dengue. Patients were classified into classical dengue fever (DF), dengue haemorrhagic fever (DHF), or dengue shock syndrome (DSS) based on the WHO criteria. For each included patient, clinical details, routine laboratory investigations (complete blood counts, liver function tests, coagulation profiles including PT and APTT), and abdominal ultrasonography findings were extracted from the medical records. Of the 281 eligible inpatients, comprehensive laboratory and imaging data were available for 110 patients, and this subset was used for detailed analysis. Data were anonymized, tabulated, and evaluated using standard descriptive statistical methods. As this was a retrospective analysis of hospital records, formal patient consent was waived, and institutional ethical clearance was obtained.

RESULTS:

Table 1. Mean Values and Standard Deviations of Various Liver Function Tests (LFTs)

Parameter	Mean Value	Standard Deviation (SD)
Total Bilirubin (mg/dL)	0.95	0.72
SGOT (AST) (U/L)	687.28	3037.94
SGPT (ALT) (U/L)	293.65	1041.89
Serum Albumin (g/dL)	3.39	0.45
Alkaline Phosphatase(U/L)	112.14	82.17
INR	1.3	0.28

Table 2: Percentage of Patients with Various Levels of SGOT & SGPT Elevation

Parameter	Level	Frequency	Percent (%)
SGOT/SGPT	<40	3	1.1
SGOT/SGPT	>40	278	98.9
SGOT/SGPT	Total	281	100
SGOT	<40	4	1.4
SGOT	41–120	128	45.6
SGOT	121–400	103	36.7
SGOT	>400	46	16.4
SGOT	Total	281	100
SGPT	<40	11	3.9
SGPT	41–120	78	27.8
SGPT	121–400	167	59.4
SGPT	>400	25	8.9
SGPT	Total	281	100

Table 3: Comparison of Various Parameters Between Male and Female Dengue Patients

Characteristic	Male (Mean ± SD)	Female (Mean ± SD)	p-value
Age (in Years)	42.799 ± 15.811	43.804 ± 14.93	0.611
Platelets (per cu.mm)	0.554 ± 0.363	0.981 ± 3.711	0.118
T. Bilirubin (mg/dl)	0.959 ± 0.663	0.933 ± 0.835	0.774
SGOT (U/L)	720.603 ± 3517.682	618.837 ± 1681.11	0.793
SGPT (U/L)	318.443 ± 1230.32	242.717 ± 457.187	0.568
S. Albumin (g/dl)	3.729 ± 3.04	3.668 ± 3.433	0.881
S. Alkaline Phosphatase(U/L)	107.106 ± 76.895	122.478 ± 91.646	0.141
INR	1.294 ± 0.253	1.327 ± 0.335	0.913

Table 4: Comparison of Biochemical Liver Test Derangements Among Dengue Groups

Characteristic	DF (n=248)	DHF (n=21)	DSS (n=12)	p-value (DF vs DHF)	p-value (DF vs DSS)	p-value (DHF vs DSS)
Mean SGOT (U/L)	218.528	1747.762	8519.083	0.008	0	0
Mean SGPT (U/L)	125.805	856.42	2777.5	0	0	0
Mean S. Albumin (g/dl)	3.735	4.01	2.65	0.704	0.248	0.237
Mean Bilirubin (mg/dl)	0.78	2.01	2.69	0	0	0
Mean INR	1.25	1.449	2.19	0	0	0

DISCUSSION:

In this hospital-based observational study, a comprehensive analysis of liver function test (LFT) parameters was performed among 281 patients diagnosed with dengue infection. Among these individuals, the majority (88.3%) presented with classical Dengue Fever (DF), while Dengue Haemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS) were observed in 7.5% and 4.3% of cases, respectively. This distribution closely mirrors the findings of a study conducted in Delhi, where DF was predominant, and DHF and DSS were noted in 9.3% and 2.2% of patients, respectively [11].

A notable observation in our cohort was the high frequency of transaminase elevation, with 98.9% of patients exhibiting elevated SGOT/SGPT levels (transaminase, defined as SGOT or SGPT > upper limit of normal). This aligns with the report by Shukla et al. [12], who documented 100% elevation in SGOT and 91% elevation in SGPT in their dengue cohort. Comparable patterns of elevated aminotransferases have also been documented by other researchers [13,14]. However, elevated bilirubin or alkaline phosphatase (ALP) levels were less common. A key finding was that mean SGOT values were more than twice the mean SGPT values, contrasting the typical pattern of viral hepatitis, in which SGPT commonly exceeds SGOT. Similar results have been reported in studies from Kolkata, India, and Karachi, Pakistan, where SGOT levels consistently surpassed SGPT levels [15,16]. Although the exact underlying mechanism remains unclear, one hypothesis suggests that this pattern may result from excessive AST (SGOT) release by damaged monocytes during dengue infection. Thus, an SGOT-predominant elevation could help differentiate dengue from other conditions such as acute viral hepatitis, especially when initial presentation and laboratory features overlap. When comparing laboratory parameters between males and females in our study, no statistically significant differences were noted in the LFT values. This finding aligns with a large study conducted in Brazil, which also found no significant sex-based difference in transaminase levels. Interestingly, the Brazilian study noted that liver damage was more frequently reported in females, which differs from our findings [17]. It is worth mentioning that few investigations have specifically focused on comparing LFT values between male and female dengue patients. Our data revealed statistically significant differences in LFT abnormalities when comparing DF, DHF, and DSS groups. Patients with DHF demonstrated significantly higher bilirubin, SGOT, SGPT, ALP, and INR values compared to those with DF ($p<0.05$), although albumin levels remained similar ($p=0.704$). Similarly, those with DSS also showed higher values of bilirubin, SGOT, SGPT, ALP, and INR compared to DF patients ($p<0.05$), while albumin levels again remained unaffected ($p=0.248$). These trends clearly suggest that hepatic involvement intensifies as the severity of dengue increases. From a clinical standpoint, severely deranged LFTs, including elevated SGOT, SGPT, bilirubin, ALP, and INR, may serve as a valuable indicator of severe dengue infection. Previous literature supports the notion that hepatic injury is a positive predictive factor for DHF [18]. Kuo et al. [19] similarly reported that elevated aminotransferase levels correlate with disease severity, and Chhina et al. [20] documented that DHF patients experience more pronounced elevations in SGOT and ALP, while DSS patients exhibit significant increases across all biochemical liver parameters compared to DF. This pattern reinforces the association between escalating liver injury and worsening clinical status. Albumin levels generally remain stable in dengue because it is an acute illness rather than a chronic liver condition, where albumin synthesis is often impaired. The development of jaundice in DF, DHF, or DSS may be multifactorial. Potential contributors include direct hepatic injury by the dengue virus, as well as hypoxic and ischemic insult to liver tissues, particularly in cases involving dengue shock. Jaundice tends to be more frequent in complicated dengue cases and has been linked with fulminant hepatic failure. It also serves as a poor prognostic indicator [21]. In our investigation, 3.6% (10/281) of patients exhibited total bilirubin values exceeding 3 mg/dl, signifying a subset of patients with substantial liver involvement.

Hepatic encephalopathy, a serious manifestation, was observed in only six patients in our sample. LFT abnormalities in these individuals were

significantly more pronounced compared to those without encephalopathy, indicating that hepatic failure was a likely contributor to their altered mental status. Nevertheless, other mechanisms such as metabolic acidosis, disseminated intravascular coagulation (DIC), severe haemorrhage, cerebral edema, or hyponatremia induced by excessive fluid therapy can also play a role in neurological symptoms associated with dengue. Additionally, an abnormal prothrombin time (PT) suggests coagulopathy, more frequently evident in severe dengue cases involving acute hepatic failure, DIC, shock, and encephalopathy. In such severe scenarios, the extensive hepatocellular injury likely reduces production of clotting factors, prolonging PT [22]. In our study, significantly higher PT prolongation was noted among DSS cases compared to DF and DHF, reinforcing the link between hepatic dysfunction and severe disease states. It is important to acknowledge certain limitations of our study. As a hospital-based record analysis, our findings may not accurately represent the full spectrum of dengue infections in the community. Many dengue patients with milder symptoms receive outpatient treatment and thus remain underrepresented. Consequently, our cohort may be biased towards more symptomatic individuals or those with marked laboratory abnormalities. Moreover, some patients were included based solely on rapid immunochromatographic tests, whereas IgM capture ELISA remains the gold standard, as per current recommendations, for confirming dengue infection. Despite these constraints, our investigation provides valuable insights into the hepatic manifestations of dengue and their correlations with disease severity.

CONCLUSION:

We examined abnormalities in liver function tests (LFTs) in a large cohort of patients with dengue infection. Nearly all participants showed elevated transaminases (SGOT and SGPT), with SGOT levels generally rising more prominently than SGPT. Moreover, levels of SGOT/SGPT, alkaline phosphatase (ALP), international normalized ratio (INR), and bilirubin were significantly greater in patients suffering from dengue haemorrhagic fever (DHF) and dengue shock syndrome (DSS) compared to those with uncomplicated dengue fever (DF). These findings suggest that an early and substantial increase in SGOT may serve as an initial marker of dengue infection. Additionally, pronounced elevations in bilirubin, SGOT, SGPT, and ALP appear to be indicators of more severe disease and may be associated with a less favourable prognosis.

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