



Hepatic Dysfunction And Its Correlation With Platelet Count In Dengue Infection: A Hospital-Based Case–Control Study From Eastern India.

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Abstract

Background: Dengue fever is an important mosquito-borne viral infection causing substantial morbidity in tropical and subtropical countries. Hepatic involvement is a recognized manifestation of dengue infection and may range from mild elevation of liver enzymes to significant hepatic dysfunction. Monitoring liver function parameters may provide important insights into disease severity. **Objective:** To evaluate liver dysfunction in patients with dengue infection and to determine its correlation with platelet count.

Methods: This hospital-based case–control study was conducted in the Department of Biochemistry in collaboration with the Departments of General Medicine and Pediatrics at Saheed Laxman Nayak Medical College and Hospital, Koraput, Odisha. A total of 160 participants were included, comprising 80 confirmed dengue patients and 80 age- and sex-matched healthy controls. Liver function tests including serum bilirubin, SGOT, SGPT, total protein, albumin, and platelet counts were analyzed. Statistical analysis was performed using appropriate tests, and correlation between platelet count and liver enzymes was assessed using Pearson correlation. **Results:** Dengue patients demonstrated significantly elevated levels of SGOT and SGPT compared with the control group ($p < 0.001$). Platelet counts were significantly lower among dengue cases than controls. A significant negative correlation was observed between platelet count and liver enzyme levels, indicating that declining platelet counts were associated with increasing SGOT and SGPT levels. **Conclusion:** Hepatic dysfunction is a common finding in dengue infection and is associated with thrombocytopenia. Monitoring liver function parameters alongside platelet counts may help in assessing disease severity and improving clinical management of dengue patients.

Keywords: Dengue infection; Liver function tests; Thrombocytopenia; SGOT; SGPT; Hepatic dysfunction; Platelet count.



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INTRODUCTION

Dengue fever is one of the most important mosquito-borne viral infections affecting tropical and subtropical regions of the world. The disease is caused by the dengue virus (DENV), which is transmitted primarily by *Aedes aegypti* mosquitoes. Although dengue infection is often self-limiting, the illness can lead to significant morbidity due to high fever, severe body aches, and systemic manifestations that may result in considerable physical debility. In severe cases, the infection may progress to dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS), which are associated with increased complications and mortality. Despite the growing global burden of dengue, effective antiviral therapy remains unavailable, and management largely relies on supportive care and careful clinical monitoring.¹

Hepatic involvement is a well-recognized manifestation of dengue virus infection and may range from mild elevation of liver enzymes to severe hepatic dysfunction. Previous studies have demonstrated that hepatocytes and Kupffer cells are important targets for dengue virus replication, as evidenced by histopathological findings in liver biopsy and autopsy specimens of severe cases.² Infection of hepatocytes by dengue virus can lead to cellular injury and programmed cell death through apoptosis. The apoptotic process has been demonstrated in both in-vivo and in-vitro studies and results in characteristic histological changes such as the formation of Councilman bodies.

Several mechanisms have been proposed to explain dengue-related liver injury. These include direct viral cytopathic effects, immune-mediated hepatocellular damage, mitochondrial dysfunction due to hypoxia, and cellular stress responses involving the endoplasmic reticulum. Additionally, inflammatory mediators such as tumor necrosis factor- α (TNF- α), tumor necrosis factor-related apoptosis-inducing ligand (TRAIL), and Fas signaling pathways have been implicated in the

induction of hepatocyte apoptosis during dengue infection. Dysfunction of mitochondrial pathways further contributes to cellular injury and amplification of hepatocellular damage.³

Liver dysfunction in dengue infection is often reflected by abnormalities in liver function tests, particularly elevated levels of serum transaminases. These biochemical changes may also be associated with hematological abnormalities such as thrombocytopenia, which is a common finding in dengue infection. Understanding the relationship between hepatic dysfunction and platelet count may therefore provide useful insights into disease severity and clinical progression.

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Considering the increasing incidence of dengue infection in eastern India, particularly in Odisha, it is important to evaluate the pattern of hepatic involvement in affected patients. Therefore, the present hospital-based study conducted at Saheed Laxman Nayak Medical College and Hospital aimed to assess liver dysfunction in patients diagnosed with dengue infection and to examine the correlation between liver function test abnormalities and platelet counts.

MATERIALS AND METHODS

This study was conducted as a hospital-based case-control study to evaluate liver dysfunction among patients with dengue infection and to analyze its association with platelet count. The study was carried out in the Department of Biochemistry in collaboration with the Departments of General Medicine and Pediatrics at Saheed Laxman Nayak Medical College and Hospital, Koraput, Odisha, India. This tertiary care teaching hospital caters to patients from Koraput district and surrounding tribal regions of southern Odisha.

The required sample size was determined based on an expected statistical power of 80% and a 5% level of significance. Considering feasibility and the availability of eligible patients during the study period, a total sample size of 160 participants was included in the study. The participants were divided into two groups with equal representation:

- Cases: 80 patients diagnosed with dengue fever
- Controls: 80 apparently healthy individuals matched for age and sex

This sample size was considered adequate to detect meaningful differences in biochemical parameters between the study groups.

Study population

Cases:

Patients diagnosed with dengue fever who were admitted to the hospital during the study period.

Controls:

Age- and sex-matched healthy individuals without any clinical or laboratory evidence of dengue infection.

Inclusion criteria

1. Patients of any age and either sex presenting with clinical features suggestive of dengue fever.
2. Laboratory-confirmed dengue infection with positive dengue NS1 antigen or dengue IgM antibody test.

Exclusion criteria

1. Patients presenting with fever but negative for dengue NS1 antigen or IgM antibody.
2. Patients with pre-existing chronic diseases such as chronic liver disease (CLD), chronic kidney disease (CKD), or coronary artery disease (CAD).
3. Individuals with a history of hepatotoxic drug intake or medications known to alter liver function tests.
4. Patients with dengue infection associated with other infections known to cause hepatic involvement, including viral hepatitis, leptospirosis, malaria, or enteric fever.

Collected data were compiled and entered into Microsoft Excel for organization and preliminary analysis. Statistical analysis was performed using appropriate statistical software. Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were summarized as frequencies and percentages. Suitable statistical tests were applied to determine the significance of differences between study groups, and a p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 160 participants were included in the study, consisting of 80 confirmed dengue patients (cases) and 80 healthy age- and sex-matched individuals (controls). Biochemical analysis revealed significant alterations in liver function parameters among dengue patients compared with the control group. Patients with dengue infection demonstrated marked elevation of serum transaminases (SGOT and SGPT) along with reduced serum albumin levels and lower platelet counts.

Correlation analysis showed a significant inverse relationship between platelet count and liver enzyme levels. The Pearson correlation coefficient between platelet count and SGOT was $r = -0.182$ ($p < 0.01$), indicating that declining platelet counts were associated with increasing SGOT levels. Similarly, platelet count showed a negative correlation with SGPT levels ($r = -0.169$, $p < 0.01$). These findings suggest that worsening thrombocytopenia in dengue infection may be associated with increasing hepatic involvement.

Table 1 General characteristics of study participants

Variable	Cases (n=80)	Controls (n=80)	p-value
Age (years)	27.12 ± 13.65	26.74 ± 14.01	0.84
Male : Female	52 : 28	49 : 31	0.61

The age distribution of participants in both groups was comparable, and no statistically significant difference was observed between cases and controls.

Table 2 Comparison of biochemical parameters between cases and controls

Variable	Cases (n=80)	Controls (n=80)	p-value
Serum total bilirubin (mg/dL)	0.82 ± 0.12	0.78 ± 0.09	0.28
SGOT (IU/L)	158.64 ± 76.82	42.75 ± 5.90	0.001
SGPT (IU/L)	91.37 ± 44.21	43.89 ± 4.12	0.001
Total protein (g/dL)	6.39 ± 0.64	6.48 ± 0.55	0.33
Albumin (g/dL)	3.69 ± 0.31	3.92 ± 0.28	0.01
Platelet count (lakh/mm ³)	1.38 ± 0.41	2.08 ± 0.87	0.001

Patients with dengue infection demonstrated significantly elevated SGOT and SGPT levels compared with healthy controls. In addition, platelet counts were significantly reduced in dengue cases.

DISCUSSION

Dengue infection remains one of the most significant mosquito-borne viral illnesses affecting tropical and subtropical regions. India has experienced a steady increase in dengue incidence over recent decades, with outbreaks reported in multiple states including Odisha.^{4,5} The present hospital-based study conducted at Saheed Laxman Nayak Medical College and Hospital evaluated hepatic dysfunction in patients with dengue infection and examined its relationship with platelet counts.

In the present study, dengue patients showed significantly elevated serum transaminases compared with the control group. These findings indicate hepatic involvement during dengue infection, which may occur due to direct viral injury to hepatocytes or immune-mediated inflammatory responses. Similar observations have been reported in previous studies conducted in different regions of India.

Elevated SGPT levels were observed in a large proportion of dengue patients in our study. Comparable findings have been reported by Srivenu Itha et al., who documented significant elevation of liver enzymes in dengue infection.⁶ Brij Mohan et al. also reported increased SGPT levels among dengue patients, indicating hepatic involvement as a common clinical feature.⁷

Studies conducted in other populations have similarly demonstrated abnormal liver function tests in dengue infection. Kuo et al. reported elevated ALT levels in approximately 82% of dengue cases, suggesting that hepatic injury is a frequent manifestation of the disease.⁸ Faridi et al. observed raised ALT levels in approximately 64% of dengue patients, further supporting the presence of hepatic dysfunction during dengue infection.⁸

The present study also demonstrated a significant inverse correlation between platelet count and liver enzyme levels, indicating that increasing hepatic injury may be associated with worsening thrombocytopenia. Similar findings were reported by Chandrasekar et al., who observed a negative correlation between platelet counts and transaminase levels in dengue infection.⁹

These findings suggest that hepatic dysfunction may reflect the severity of dengue infection. As platelet counts decline, elevated transaminase levels may indicate increased hepatocellular injury resulting from viral replication, immune-mediated damage, and systemic inflammatory responses.¹⁰ Another important observation in the present study was the reduced serum albumin level in dengue patients compared with controls. Hypoalbuminemia may occur due to increased vascular permeability, hepatic dysfunction, and altered protein synthesis during dengue infection.¹¹

Although this study provides valuable insights into the relationship between hepatic dysfunction and thrombocytopenia in dengue infection, certain limitations should be acknowledged.¹² The study was conducted in a single tertiary care hospital, which may limit the generalizability of the findings to the wider population. Additionally, the cross-sectional design restricts the ability to assess temporal changes in liver function during the course of the illness. Despite these limitations, the present study provides useful evidence that hepatic dysfunction is a common manifestation in dengue infection and that liver enzyme abnormalities may correlate with thrombocytopenia.

CONCLUSION

The present hospital-based study demonstrated a significant association between elevated liver enzymes and reduced platelet counts in dengue infection. Patients with dengue showed marked elevation of serum transaminases along with thrombocytopenia compared with healthy controls.

These findings suggest that hepatic dysfunction is a frequent manifestation of dengue infection and may reflect disease severity. Routine monitoring of liver function tests in patients with dengue infection may therefore help in early identification of hepatic involvement and facilitate timely clinical management.

It is recommended that patients diagnosed with dengue infection should be carefully evaluated for liver dysfunction. Furthermore, abnormalities in liver function tests, when interpreted alongside clinical features and hematological parameters, may aid clinicians in assessing the severity and progression of dengue infection.

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