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HIGH-SENSITIVITY CARDIAC TROPONIN I AS A BIOMARKER OF MYOCARDIAL INJURY IN SEVERE DENGUE: A RETROSPECTIVE STUDY IN HOSPITALIZED PATIENTS

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ABSTRACT

Background: A major worldwide health problem, dengue fever has a broad spectrum of clinical severity. Although hematological and vascular problems are well known, cardiac involvement is underreported despite its major impact on morbidity and death. High-sensitivity cardiac troponin I (hs-Troponin I), a sensitive marker of myocardial injury, might assist to detect cardiac involvement in dengue.**Objective:** To evaluate the correlation between serum hs-Troponin I values and the degree of dengue infection. **Materials and Method:** From August 2024 to September 2025, this retrospective study was carried out at a tertiary care facility hospital. Enrolled were data of 100 adult patients (aged 18–70 years) with confirmed dengue infection (NS1 antigen or IgM positive). World Health Organization (WHO) 2009 criteria classified patients into dengue fever (DF), dengue hemorrhagic fever (DHF), and dengue shock syndrome (DSS). Using an automated immunoassay, serum hs-Troponin I levels were determined. Using one-way ANOVA followed by Bonferroni post-hoc test, statistical analysis was carried out; $p< 0.05$ was regarded significant. **Results:** With a small male predominance (52%), participants had a mean age of 39.67 ± 15.8 years. With illness severity, hs-Troponin I levels increased dramatically: DF (18.23 ± 19.49), DHF (179.27 ± 19.49), and DSS (674.05 ± 124.91) ($p< 0.05$). Post-hoc study revealed statistically significant differences ($p< 0.001$) among all the groups. The results show a gradual rise in hs-Troponin I aligned with growing dengue severity. **Conclusion:** High hs-Troponin I levels are strongly linked with serious forms of dengue and might provide a practical biomarker for early identification of cardiac damage. Regular evaluation of hs-Troponin I might help to refine risk stratification, direct clinical treatment, and lower negative consequences in dengue patients.

Keywords: Dengue fever, hs-Troponin I, Myocardial injury, Dengue shock syndrome, Biomarker.

INTRODUCTION

One of the most serious public health issues in tropical and subtropical countries is dengue fever, a mosquito-borne viral infection caused by the dengue virus (DENV), primarily transmitted by Aedes aegypti and Aedes albopictus.

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With manifestations spanning a spectrum from asymptomatic or minor flu-like symptoms to severe forms such as dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS), dengue infects nearly 390 million individuals annually and can lead to life-threatening plasma leakage, bleeding, and organ damage[1]. Although traditionally dengue infection is associated with haematological and vascular complications, emerging evidence highlights the under recognized role of cardiac involvement in exacerbating disease severity.

Many dengue patients also experience cardiac complications, such as inflammation of the heart muscle (myocarditis), irregular heartbeats (arrhythmias), and weakened heart function. In severe dengue, cardiovascular collapse from hypovolemia due to plasma leakage can be complicated by myocardial dysfunction, leading to refractory shock and multi-organ failure[2].These complications may begin subtly with palpitations, shortness of breath, or abnormal ECG findings[3] . In severe cases, they can progress to life-threatening heart failure or shock, ultimately transforming a manageable infection into a medical emergency[4].

Dengue infection with cardiac consequences strongly increases the risk of death[5]. Studies reveal that individuals with dengue myocarditis have much greater death rates—up to 21.4% versus 1.6% in dengue patients without cardiac involvement[6,7]. Although more studies now explore dengue's systemic effects, cardiac complications remain under diagnosed and poorly integrated into standard management protocols due to their subclinical nature in many patients[8]. This oversight contributes to delayed interventions and higher mortality and morbidity.

Mechanistically, elevated troponin-I (Trop I) in dengue arises from myocardial injury triggered by a multifaceted pathogenesis. Direct viral invasion of cardiomyocytes promotes apoptosis via viral proteins and genetic material. This is aggravated by inflammation and edema due to immune-mediated damage, driven by cytokine storms (e.g., pro-inflammatory cytokines like TNF-α and IL-6), T-cell activation, and complement pathway upregulation. This manifests as subclinical myocarditis or cardiac impairment, leading to higher vascular permeability, capillary leakage, and lower cardiac preload. Elevated troponin-I, hence a sensitive indicator of cardiomyocyte damage, is seen in dengue patients[9, 10].

Early phase NS1 antigen testing helps to confirm a dengue diagnosis, but determining which patients will get cardiac involvement is still difficult. A sensitive and easily accessible marker of myocardial damage, cardiac troponin-I (trop - I) also matches with the seriousness of dengue fever[11]. According to earlier research, NS1-positive dengue patients with high troponin-I are more likely to have Electro Cardio Graphic (ECG) abnormalities, palpitations, breathlessness, or echocardiographic indications of myocardial dysfunction[12]. The justification for this study was to show a strong link between the severity of dengue fever, as determined by raised troponin-I, and the increased risk of cardiac events. Clinicians will be better equipped to concentrate on monitoring cardiac diseases, modify fluid and hemodynamic management as necessary, and distribute intensive care resources more effectively.

MATERIALS AND METHOD

A retrospective analysis was conducted on 100 adult patients (aged 18–70 years) hospitalized with confirmed dengue infection (NS1 antigen or IgM positive) between August 2024 and September 2025.

Based on WHO 2009 criteria, patients were categorized into dengue fever (DF), dengue hemorrhagic fever (DHF), and dengue shock syndrome (DSS). Serum hs-Troponin I levels,

measured via automated immunoassay during the clinical course, were retrieved and analyzed.Records of patients with pre-existing ischemic heart disease, heart failure, chronic kidney disease, severe liver disease, pregnancy, or recent major surgery were excluded from the study. Statistical significance was determined using one-way ANOVA and Bonferroni post-hoc tests.

Continuous variables (e.g., hs-Troponin I) were shown using mean ± standard deviation (SD). Comparison across dengue severity groups (DF, DHF, DSS) was done using one-way ANOVA for total variations; then post-hoc Bonferroni test was employed for pairwise comparisons. Using chi-square test, categorical data were compared. Statistically significant was regarded to have a p-value less than 0.05

RESULTS

Table 1: Characteristics of the study subjects (n = 100)

Patient characteristics	Result
Age (years) Range	39.67 ± 15.8 12-83
Gender-Male (%) Female (%)	52% 48%

n= number of subjects

The study comprised 100 patients who had been diagnosed with dengue fever and had a positive dengue NS1 antigen. The average age of the study subjects was 39.67 ± 15.8 years. Approximately 52% of them were men and 48% were women as shown in Table 1.

Table 2: Comparison of hs-Troponin I among different stages of dengue fever (n = 100)

Dengue severity	hs-Troponin I		*p*-value
	n	Mean ± SD	
Dengue Fever	44	18.22955 ± 19.492033	<0.05
Dengue Hemorrhagic Fever	38	179.26632 ± 19.492033	
Dengue Shock Syndrome	18	674.05000 ± 124.909507	

p-value obtained from a one-way ANOVA test; n=number of subjects

The comparison of hs-Troponin I levels at various dengue stages (n = 100) is shown in Table 2. hs-Troponin I levels were significantly higher in patients with dengue shock syndrome (674.05 ± 124.91) than in patients with dengue fever (18.23 ± 19.49) and dengue hemorrhagic fever (179.27 ± 19.49). Across all stages, the increase in hs-Troponin I was statistically significant and showed a gradual rise with illness severity ($p < 0.05$).

Table 3: Comparison of hs-Troponin I among different severity grades of dengue fever (n=100)

Dengue Severity	*p*-value
Dengue Fever vs Dengue Hemorrhagic Fever	<0.001
Dengue Hemorrhagic Fevervs Dengue Shock Syndrome	<0.001
Dengue Fever vs Dengue Shock Syndrome	<0.001

n=number of subjects; *p*-value reached by applying post-hoc analysis using the Bonferroni test to observe the difference in hs-Troponin I levels in between different stages of dengue fever.

In Table 4 hs-Troponin I levels showed a progressive rise from dengue fever to dengue hemorrhagic fever and further to dengue shock syndrome, with all pairwise differences being highly significant ($p< 0.001$).

DISCUSSION

The study demonstrated a significant and progressive rise in high-sensitivity troponin I (hs-Troponin I) levels with increasing severity of dengue infection, and was detected in the highest concentrations in patients with DSS. This finding reinforces emerging evidence that myocardial injury is an important but often under recognized component of severe dengue infection and supports the utility of hs-Troponin I as a potential biomarker to suspect cardiac involvement in dengue fever. Cardiac manifestations of dengue, though historically considered atypical, are increasingly recognized as contributors to disease severity and adverse outcomes[2-4] .

In our study, individuals with DHF and DSS had significantly higher Hs-Troponin I levels than those with dengue fever. These differences were statistically significant across all severity categories. This gradual increase in hs-Troponin I indicates that myocardial damage deteriorates concurrently with the advancement of the illness. Dudveet al. published similar results, showing that myocardial dysfunction in severe dengue infections is highly predicted by increased cardiac troponin I[6]. Baki et al. found that dengue patients with myocarditis had considerably greater troponin I levels, underscoring its prognostic relevance[7]. These results align with previous findings by Wali et al. regarding cardiac involvement in dengue hemorrhagic fever[2].

The mechanisms underlying myocardial injury in dengue infection are multifactorial. Direct viral invasion of cardiomyocytes may induce cellular apoptosis and necrosis, while immune-mediated myocardial damage further contributes to cardiac dysfunction. Pro-inflammatory cytokines, including interleukin-6 (IL-6), interleukin-1β, and tumour necrosis factor-α (TNF-α), have been shown to be elevated in dengue infection and are thought to be a major cause of myocardial edema and inflammation [8,9] . Increased vascular permeability brought on by this inflammatory reaction results in plasma leakage, decreased preload, and impaired coronary perfusion. These hemodynamic alterations may exacerbate myocardial ischemia, thereby contributing to the observed elevation of Hs-Troponin I, particularly in DHF and DSS[4,10] .

The markedly elevated hs-Troponin I levels observed in DSS patients in the present study emphasize the clinical relevance of myocardial involvement during the critical phase of dengue infection. Cardiovascular collapse in severe dengue is often attributed solely to hypovolemia; however, concomitant myocardial dysfunction may worsen shock and reduce responsiveness to fluid resuscitation. Yacoub et al. highlighted that myocardial depression in dengue can complicate hemodynamic management and contribute to refractory shock and multi-organ failure[4] . Recognition of myocardial injury is therefore needed for improving fluid therapy and avoiding iatrogenic complications.

Dengue often causes subclinical cardiac involvement, which manifests as mild electrocardiographic abnormalities or nonspecific symptoms such palpitations or dyspnoea. Because of this, it is frequently underdiagnosed in standard clinical practice[8]. Koh and Hong also noted elevation of Troponin I (10.51 ng/ml)in a patient who reported chest discomfort six days after being diagnosed with dengue fever and was diagnosed with suffering from dengue myocarditis[11] . Similarly, Satarasinghe et al. documented biochemical evidence of heart damage in adult dengue patients, highlighting the necessity of heightened clinical attention[12] .

From a clinical perspective, Hs-Troponin I measurement could be a useful adjunct for risk stratification in dengue fever. Early diagnosis of patients at risk of cardiac involvement could facilitate closer monitoring, early cardiology consultation, and individualized fluid and hemodynamic management. Considering the significant prevalence of dengue worldwide, especially in tropical areas with low resources[1] , the availability and relative affordability of Hs-Troponin I assays make their integration into dengue severity assessment protocols a feasible and practical strategy[6,7] .

LIMITATIONS

The cross-sectional design limits the ability to establish a causal relationship between dengue severity and myocardial injury. Serial hs-TroponinI measurements and echocardiographic assessments, which may have offered further information about the functional impact and temporal progression of myocardial involvement, were not carried out. Additionally, alternative imaging modalities and cardiac biomarkers were not evaluated.

The study demonstrates a clean affiliation among growing severity of dengue contamination and accelerated hs-Troponin I levels, indicating progressive myocardial injury, particularly in DHF and DSS. These results emphasize how crucial it is to identify cardiac involvement as a major contributor to dengue-related morbidity and mortality. Incorporation of hs-Troponin I measurement into routine evaluation of moderate to severe dengue may aid early detection of myocardial injury, manual medical management, and enhance affected person outcomes. To further understand the function of cardiac biomarkers in dengue therapy, more prospective trials with longitudinal cardiac assessment are necessary.

CONCLUSION

These results highlight the fact that cardiac involvement frequently makes dengue shock and mortality worse. Therefore, routine hs-Troponin I testing is advised as a readily available and reasonably priced technique for early risk assessment, prompt cardiac monitoring, and optimal fluid management in hospitalized patients. To improve its clinical integration, more prospective trials with repeated measures and echocardiography are required.

CONFLICT OF INTEREST

There is no conflict of interest.

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