European Journal of Clinical Pharmacy
Print ISSN: 2385-409X **Online ISSN**: 3105-0409
Website: https://farmclin.com/

Original Research Article

A Comparative Study of Left Ventricular Function and Outcomes in Patients with Viral Myocarditis and Dengue-Related Myocarditis

Article History:

Name of Author:
Dr Suhasini Atharga[1], Dr Shrikanth Metri[2]

Affiliation: *[1]Assistant Professor, Department of Cardiology JN Medical College, KAHER Belagavi, India.*
[2]Associate Professor, Department of Medicine, JN Medical College KAHER Belagavi, India

Corresponding Author:
Dr. Shrikanth Metri

Received: 18-11-2025
Revised: 26-11-2025
Accepted: 02-12-2025
Published: 21-12-2025

Abstract: Myocarditis remains a significant cause of acute cardiac dysfunction, with viral and dengue-related etiologies contributing widely in tropical and subtropical regions. Both conditions present with variable clinical severity and myocardial involvement, yet direct comparative data delineating differences in left ventricular (LV) function and outcomes are limited. ***Aim:*** To compare left ventricular function and clinical outcomes in patients with viral myocarditis versus dengue-related myocarditis. ***Methods:*** This observational comparative study included 140 patients (70 viral myocarditis, 70 dengue myocarditis) admitted to a tertiary-care hospital. Clinical parameters, laboratory markers, and echocardiographic measurements including LVEF, LV dimensions, diastolic indices, and presence of global hypokinesia were recorded. Clinical outcomes such as acute heart failure, arrhythmias, cardiogenic shock, ICU stay, LV recovery at three months, and mortality were evaluated. Statistical analyses included t-tests, Chi-square tests, and logistic regression for predictors of adverse outcomes. ***Results:*** Viral myocarditis patients exhibited significantly lower LVEF (43.7 ± 8.9% vs. 48.1 ± 7.6%, p = 0.001), larger LV dimensions, higher E/e′ ratio, and greater prevalence of global hypokinesia (51.4% vs. 30.0%, p = 0.009). Dengue myocarditis was associated with significantly lower platelet counts and higher inflammatory markers. Acute heart failure was more frequent in viral myocarditis (40% vs. 24.3%, p = 0.047), and viral patients had longer ICU stays and hospitalizations. LV recovery at three months was significantly better among dengue myocarditis patients (75.7% vs. 58.6%, p = 0.027). Predictors of adverse outcomes included older age, viral etiology, LVEF <40%, elevated E/e′, increased troponin, higher CRP levels, and reduced platelet counts. ***Conclusion:*** Viral myocarditis is associated with more severe LV dysfunction, prolonged recovery, and higher complication rates compared with dengue myocarditis, which generally demonstrates a more reversible myocardial injury pattern. Early differentiation using clinical and echocardiographic markers is essential to optimize management and prognostication.

Keywords: Viral myocarditis; Dengue myocarditis; Left ventricular function; Cardiac outcomes.

.

INTRODUCTION

Myocarditis is an inflammatory disease of the myocardium characterized by myocyte necrosis, ventricular dysfunction, and a wide spectrum of clinical presentations ranging from subclinical illness to fulminant heart failure and sudden cardiac death. Viral infections remain the most common cause of myocarditis worldwide, with cardiotropic viruses such as enteroviruses, adenoviruses, parvovirus B19, and more recently SARS-CoV-2 implicated in direct myocardial injury and immune-mediated inflammatory responses. Viral myocarditis poses significant diagnostic and therapeutic challenges due to its heterogeneous clinical manifestations, variable natural course, and the absence of highly specific noninvasive diagnostic modalities. Left ventricular (LV) dysfunction is a key determinant of prognosis in viral myocarditis, influencing the risk of arrhythmias, heart failure progression, and long-term adverse outcomes. Echocardiography, including tissue

Doppler and strain imaging, has emerged as a frontline tool for assessing myocardial involvement, allowing clinicians to evaluate both systolic and diastolic abnormalities at presentation and during follow-up.[1]

Dengue-related myocarditis represents a unique and increasingly recognized subset of myocardial inflammation, particularly in tropical and subtropical countries where dengue fever remains endemic. Myocardial involvement in dengue is multifactorial, attributed to direct viral invasion, cytokine storm, capillary leak, and immune-mediated myocardial damage. Unlike classical viral myocarditis, dengue myocarditis often occurs in the context of thrombocytopenia, hepatic dysfunction, and profound systemic inflammation, creating additional diagnostic and therapeutic complexities. Left ventricular dysfunction in dengue patients can manifest as global hypokinesia, reduced ejection fraction, diastolic dysfunction, arrhythmias, and conduction abnormalities. Although many patients recover ventricular function, a subset may progress to severe outcomes including cardiogenic shock, acute heart failure, and mortality.[2]

Comparative research examining conventional viral myocarditis and dengue-related myocarditis is scarce, despite the distinctly different pathophysiological mechanisms, clinical course, and prognosis of these two conditions. Identifying variations in left ventricular systolic and diastolic impairment, biomarker trends, recovery trajectories, and clinical outcomes may improve risk stratification and inform tailored management strategies. Understanding how dengue myocarditis differs from classical viral myocarditis is particularly important in regions where dengue outbreaks contribute significantly to cardiovascular morbidity. Furthermore, such comparisons may elucidate whether dengue-induced myocardial injury is transient and reversible or comparable in severity to traditional viral etiologies.[3][4]

Aim

To compare left ventricular function and clinical outcomes between patients with viral myocarditis and dengue-related myocarditis.

Objectives

1. To assess and compare left ventricular systolic and diastolic function in patients with viral myocarditis and dengue-related myocarditis.
2. To evaluate clinical outcomes, including complications, recovery of LV function, and mortality, in both patient groups.
3. To identify factors associated with adverse outcomes in viral versus dengue-related myocarditis.

MATERIAL AND METHODOLOGY

Source of Data

Data were obtained from medical records of patients diagnosed with myocarditis and admitted to the Department of Cardiology and Internal Medicine at the study center. Echocardiography logs, laboratory records, and electronic hospital case files were reviewed.

Study Design

This was a hospital-based comparative observational study conducted retrospectively and prospectively.

Study Location

The study was carried out at a tertiary care teaching hospital equipped with advanced cardiology, echocardiography, and intensive care facilities.

Study Duration

The study was conducted over a 4-year period, including retrospective data (first 2 years) and prospective enrollment (last 2 years).

Sample Size

A total of 140 patients were included in the study, with 70 diagnosed with viral myocarditis and 70 with dengue-related myocarditis.

Inclusion Criteria

1. Patients aged ≥18 years diagnosed with myocarditis based on clinical, laboratory, ECG, and echocardiographic findings.
2. Viral myocarditis patients confirmed by clinical presentation and relevant serological/virological tests.
3. Dengue myocarditis patients confirmed by NS1 antigen/IgM positivity and compatible cardiac involvement.
4. Patients who underwent echocardiography within 48 hours of admission.

Exclusion Criteria

1. Patients with pre-existing cardiomyopathy or structural heart disease.
2. Known coronary artery disease or prior myocardial infarction.
3. Chronic kidney disease stage ≥3 or hepatic failure.
4. Incomplete medical records or missing echocardiographic data.
5. Pregnant women.

MATERIAL AND METHODS

Procedure and Methodology

All eligible patients were identified from the hospital database. Detailed demographic, clinical, laboratory, electrocardiographic, and echocardiographic parameters were recorded. Viral myocarditis cases were classified based on clinical history, viral markers, ECG changes, elevated cardiac enzymes, and echocardiographic LV dysfunction. Dengue myocarditis cases were diagnosed using positive dengue serology combined with cardiac

abnormalities such as elevated troponin, arrhythmias, or LV dysfunction.

Echocardiography was performed using standardized protocols. Measurements included LVEF (Simpson's method), LVEDD, LVESD, fractional shortening, E/A ratio, E/e' ratio, global hypokinesia or segmental wall motion abnormalities, and presence of pericardial effusion. Hemodynamic status and clinical progression were documented throughout hospitalization.

Sample Processing

Blood samples were analyzed for cardiac biomarkers (troponin I/T), CK-MB, complete blood count, liver and renal function tests, and inflammatory markers. Dengue serology (NS1, IgM, IgG) and viral markers were processed using ELISA-based assays. ECG and chest X-ray findings were recorded.

Statistical Methods

Data were entered into Microsoft Excel and analyzed using SPSS version 25. Continuous variables were expressed as mean ± SD and compared using Student's t-test or Mann–Whitney U test. Categorical variables were presented as frequencies and percentages and compared using Chi-square or Fisher's exact test. Multivariate logistic regression was used to identify predictors of adverse outcomes. A p-value <0.05 was considered statistically significant.

Data Collection

Data were collected using a structured proforma that included demographic details, clinical presentation, laboratory parameters, ECG findings, echocardiographic measurements, treatment given, hospital stay duration, complications, and outcomes at discharge.

RESULTS

Table 1: Baseline Characteristics of Patients with Viral vs Dengue-Related Myocarditis (N = 140)

Variable	Viral Myocarditis (n=70)	Dengue Myocarditis (n=70)	Test Statistic	95% CI of Difference	p-value
Age (years), Mean ± SD	41.8 ± 12.7	36.4 ± 11.9	t = 2.52	0.98 to 9.82	0.013
Male Sex, n (%)	44 (62.9%)	39 (55.7%)	$\chi^2 = 0.78$		0.377
Heart Rate (bpm), Mean ± SD	101.6 ± 18.4	108.7 ± 21.2	t = 2.12	0.53 to 13.64	0.036
Systolic BP (mmHg), Mean ± SD	102.3 ± 15.8	96.1 ± 14.2	t = 2.31	1.02 to 11.38	0.022
Troponin-I Positive, n (%)	57 (81.4%)	48 (68.6%)	$\chi^2 = 3.36$		0.067
Platelet Count ($\times 10^9$/L), Mean ± SD	178.6 ± 62.4	92.8 ± 38.7	t = 9.05	67.09 to 105.51	<0.001
CRP (mg/L), Mean ± SD	23.9 ± 11.7	31.8 ± 14.6	t = 3.42	3.20 to 12.50	0.001
Creatinine (mg/dL), Mean ± SD	1.12 ± 0.42	0.94 ± 0.28	t = 2.71	0.05 to 0.31	0.008

Table 1 compares the baseline characteristics of patients with viral myocarditis and dengue-related myocarditis. Patients with viral myocarditis were significantly older than those with dengue myocarditis (41.8 ± 12.7 vs. 36.4 ± 11.9 years, p = 0.013), indicating a modest but meaningful age difference favoring younger dengue patients. The distribution of male sex was comparable between the groups (62.9% vs. 55.7%, p = 0.377), suggesting no significant sex-based predisposition. Dengue myocarditis patients demonstrated significantly higher heart rates (108.7 ± 21.2 bpm vs. 101.6 ± 18.4 bpm, p = 0.036), likely reflecting the systemic inflammatory response and autonomic imbalance typical of dengue infection. Conversely, systolic blood pressure was significantly lower in dengue myocarditis (96.1 ± 14.2 mmHg vs. 102.3 ± 15.8 mmHg, p = 0.022), consistent with plasma leakage and capillary permeability changes observed in dengue.

Although troponin-I positivity was more common in viral myocarditis (81.4% vs. 68.6%), the difference did not reach statistical significance (p = 0.067). As expected, platelet counts were markedly lower in dengue myocarditis (92.8 ± 38.7 $\times 10^9$/L vs. 178.6 ± 62.4 $\times 10^9$/L, p < 0.001), reflecting typical dengue-related thrombocytopenia. CRP levels were significantly higher in dengue patients (31.8 ± 14.6 vs. 23.9 ± 11.7 mg/L, p = 0.001), indicating greater systemic inflammation. Serum creatinine was slightly higher in viral myocarditis (1.12 ± 0.42 mg/dL vs. 0.94 ± 0.28 mg/dL, p = 0.008), possibly due to reduced renal perfusion or associated viral sepsis.

Table 2: Comparison of LV Systolic and Diastolic Function (N = 140)

LV Function Parameter	Viral Myocarditis (n=70)	Dengue Myocarditis (n=70)	Test Statistic	95% CI of Difference	p-value
LVEF (%), Mean ± SD	43.7 ± 8.9	48.1 ± 7.6	t = 3.23	1.77 to 7.17	0.001
LVEDD (mm), Mean ± SD	53.6 ± 5.8	49.3 ± 5.1	t = 4.37	2.40 to 6.07	<0.001
LVESD (mm), Mean ± SD	38.9 ± 5.9	35.4 ± 5.0	t = 3.52	1.53 to 5.25	<0.001
Fractional Shortening (%), Mean ± SD	19.8 ± 4.2	23.1 ± 4.8	t = 4.33	1.76 to 4.82	<0.001
E/A Ratio, Mean ± SD	1.24 ± 0.38	1.07 ± 0.30	t = 3.00	0.06 to 0.29	0.003
E/e′ Ratio, Mean ± SD	14.6 ± 3.1	12.8 ± 2.7	t = 3.46	0.77 to 2.53	<0.001
Global Hypokinesia, n (%)	36 (51.4%)	21 (30.0%)	$\chi^2 = 6.85$		0.009
Pericardial Effusion, n (%)	19 (27.1%)	11 (15.7%)	$\chi^2 = 2.86$		0.091

Table 2 evaluates and compares echocardiographic parameters of left ventricular systolic and diastolic function between viral and dengue myocarditis patients. LVEF was significantly lower in viral myocarditis (43.7 ± 8.9%) compared with dengue myocarditis (48.1 ± 7.6%, p = 0.001), suggesting more severe systolic dysfunction in viral etiologies. Supporting this, LV dimensions were significantly larger in viral myocarditis, with higher LVEDD (53.6 ± 5.8 mm vs. 49.3 ± 5.1 mm, p < 0.001) and LVESD (38.9 ± 5.9 mm vs. 35.4 ± 5.0 mm, p < 0.001). Fractional shortening was also lower in viral myocarditis (19.8 ± 4.2% vs. 23.1 ± 4.8%, p < 0.001), reinforcing the presence of greater systolic impairment.

Diastolic function indices showed that viral myocarditis patients had significantly higher E/A ratios (1.24 ± 0.38 vs. 1.07 ± 0.30, p = 0.003) and significantly elevated E/e′ ratios (14.6 ± 3.1 vs. 12.8 ± 2.7, p < 0.001), indicating increased LV filling pressures and diastolic dysfunction. Global hypokinesia was more common in the viral group (51.4% vs. 30.0%, p = 0.009), consistent with diffuse myocardial involvement. Pericardial effusion occurred more frequently in viral myocarditis (27.1% vs. 15.7%), though the difference was not statistically significant (p = 0.091).

Table 3: Clinical Outcomes in Viral vs Dengue Myocarditis (N = 140)

Outcome Variable	Viral Myocarditis (n=70)	Dengue Myocarditis (n=70)	Test Statistic	95% CI	p-value
Acute Heart Failure, n (%)	28 (40.0%)	17 (24.3%)	$\chi^2 = 3.96$		0.047
Arrhythmias (AF/VT), n (%)	19 (27.1%)	11 (15.7%)	$\chi^2 = 2.86$		0.091
Cardiogenic Shock, n (%)	12 (17.1%)	5 (7.1%)	$\chi^2 = 3.07$		0.080
ICU Stay (days), Mean ± SD	4.8 ± 2.3	3.2 ± 1.7	t = 4.53	0.86 to 2.35	<0.001
Full LV Recovery at 3 months, n (%)	41 (58.6%)	53 (75.7%)	$\chi^2 = 4.87$		0.027
Mortality, n (%)	8 (11.4%)	3 (4.3%)	$\chi^2 = 2.29$		0.130
Length of Hospital Stay (days), Mean ± SD	7.3 ± 2.9	6.1 ± 2.4	t = 2.57	0.29 to 2.17	0.011

Table 3 compares clinical outcomes between the two groups and reveals several important differences. Acute heart failure occurred significantly more frequently in viral myocarditis (40.0% vs. 24.3%, p = 0.047), reflecting the more profound systolic impairment observed in this group. Although arrhythmias (27.1% vs. 15.7%) and cardiogenic shock (17.1% vs. 7.1%) were numerically higher in viral myocarditis, the differences did not reach statistical significance. ICU stay was significantly longer among viral myocarditis patients (4.8 ± 2.3 vs. 3.2 ± 1.7 days, p < 0.001), indicating greater severity of illness.

Interestingly, dengue myocarditis patients demonstrated higher rates of full LV functional recovery at 3 months (75.7% vs. 58.6%, p = 0.027), suggesting better myocardial reversibility in dengue-associated injury compared with classical viral myocarditis. Mortality was higher in viral myocarditis (11.4% vs. 4.3%), although the difference was statistically nonsignificant (p = 0.130). Length of hospital stay was also significantly greater in the viral group (7.3 ± 2.9 vs. 6.1 ± 2.4 days, p = 0.011).

Table 4: Factors Associated with Adverse Outcomes (N = 140)

Predictor Variable	Adverse Outcome (n=45)	No Adverse Outcome (n=95)	Test Statistic	95% CI	p-value
Age (years), Mean ± SD	44.9 ± 13.1	38.2 ± 11.7	t = 3.04	2.39 to 11.01	0.003
Viral Myocarditis Diagnosis, n (%)	29 (64.4%)	41 (43.1%)	χ^2 = 5.73		0.017
LVEF <40%, n (%)	22 (48.9%)	17 (17.8%)	χ^2 = 13.26		<0.001
E/e′ > 15, n (%)	24 (53.3%)	20 (21.0%)	χ^2 = 13.42		<0.001
Troponin-I Level (ng/mL), Mean ± SD	1.82 ± 0.71	1.21 ± 0.54	t = 5.22	0.39 to 0.82	<0.001
Platelet Count (×10⁹/L), Mean ± SD	112.4 ± 55.9	144.6 ± 68.2	t = 2.73	8.08 to 56.41	0.007
CRP (mg/L), Mean ± SD	34.5 ± 13.4	25.6 ± 11.2	t = 3.98	4.38 to 13.21	<0.001

Table 4 analyzes factors associated with adverse outcomes and demonstrates clear differences between patients who experienced complications and those who did not. Patients with adverse outcomes were significantly older (44.9 ± 13.1 vs. 38.2 ± 11.7 years, p = 0.003), indicating age as a key vulnerability factor. A significantly higher proportion of these patients had viral myocarditis (64.4% vs. 43.1%, p = 0.017), underscoring the more aggressive nature of viral etiologies. Markers of systolic and diastolic dysfunction were strongly associated with poor outcomes: LVEF <40% was present in 48.9% of the adverse-outcome group compared to only 17.8% of those without complications (p < 0.001), and E/e′ > 15 was significantly more common among adverse outcomes (53.3% vs. 21.0%, p < 0.001).

Biomarkers also played a predictive role. Troponin-I levels were significantly higher among patients with adverse events (1.82 ± 0.71 vs. 1.21 ± 0.54 ng/mL, p < 0.001), indicating more extensive myocardial injury. Platelet counts were lower in the adverse-outcome group (112.4 ± 55.9 vs. 144.6 ± 68.2 ×10⁹/L, p = 0.007), suggesting coexisting inflammatory or hematological derangements. CRP levels were markedly elevated among adverse outcomes (34.5 ± 13.4 vs. 25.6 ± 11.2 mg/L, p < 0.001).

DISCUSSION

Table 1 The baseline characteristics in the present study demonstrate clear epidemiological and clinical differences between viral and dengue-related myocarditis. The significantly younger age in dengue myocarditis (36.4 vs. 41.8 years) aligns with findings by Nerella S et al. (2022)[5], who reported that dengue myocarditis disproportionately affects young adults during epidemic outbreaks. The comparable distribution of males between groups mirrors the observations of Nicacio JM et al. (2022)[3], who noted no strong sex predilection in viral myocarditis.
Higher heart rates and lower systolic blood pressures among dengue myocarditis patients in our study reflect the hemodynamic consequences of dengue plasma leakage and cytokine surge, consistent with Baqi A et al. (2022)[1], who described tachycardia and hypotension as hallmarks of severe dengue. Thrombocytopenia was profoundly lower in dengue patients, which aligns with et al. (20)[4], reaffirming platelet suppression as a key diagnostic indicator. Additionally, higher CRP values in dengue myocarditis agree with Mansanguan C et al. (2021)[2], who demonstrated augmented systemic inflammation in dengue-related cardiac involvement. Elevated creatinine levels in viral myocarditis support findings by Ingole K et al. (2022)[6], who identified renal dysfunction as a common complication in systemic viral infections.
Table 2 The present study demonstrates more pronounced systolic dysfunction in viral myocarditis, with significantly lower LVEF, higher LV dilation indices (LVEDD and LVESD), and reduced fractional shortening. These observations are strongly supported by Huits R et al. (2023)[7], who described viral myocarditis as a diffuse inflammatory process causing reversible or irreversible LV remodeling.
Conversely, dengue myocarditis showed relatively preserved LVEF and smaller LV dimensions, which is consistent with the work of Politi MT et al. (2025)[8], who found that dengue myocarditis frequently presents with transient myocardial depression rather than structural chamber abnormalities.

The significantly higher E/e′ ratios in viral myocarditis indicate impaired relaxation and increased filling pressures, paralleling findings by Ingole K et al. (2022)[6], who associated viral myocarditis with diastolic dysfunction due to inflammatory infiltration. Global hypokinesia was more prevalent in the viral group, matching the pattern described in cardiac MRI studies by Parveen S et al. (2023)[9].

Table 3 The clinical outcomes show more severe

complications acute heart failure, longer ICU stay, and delayed recovery in viral myocarditis. Similar findings were reported by Shah TA et al. (2023)[11], who documented higher heart failure and mortality rates in viral myocarditis due to more extensive myocardial necrosis.

The present study found significantly higher LV recovery rates in dengue myocarditis (75.7% vs. 58.6%), consistent with Nicacio JM et al. (2022)[3], who described dengue myocarditis as typically reversible once systemic inflammation resolves. Mortality, although not significantly different, was numerically higher in viral myocarditis (11.4%), supporting the meta-analysis by Caetano CC et al. (2024)[12], which associated viral myocarditis with poorer long-term prognosis compared to non-viral inflammatory cardiomyopathies.

Length of hospital stay was longer among viral myocarditis patients, consistent with the findings Bhattacharjee S et al. (2023)[13], indicating prolonged hemodynamic instability in viral etiologies.

Table 4 highlights several predictors of adverse outcomes, including older age, viral etiology, reduced LVEF, elevated E/e′, increased troponin levels, and high CRP. These factors have been repeatedly described as prognostic markers in prior studies.

Age as a risk factor parallels evidence by Shah TA et al. (2023)[11], who demonstrated worse outcomes in older myocarditis patients. Viral myocarditis being strongly associated with complications agrees with Caetano CC et al. (2024)[12], who reported stronger myocardial cytotoxic damage and arrhythmogenicity in viral etiologies. LVEF <40% correlates with poor outcomes as previously established by Mansanguan C et al. (2021)[2], whereas elevated E/e′ is consistent with the prognostic framework outlined by Parveen S et al. (2023)[9], signifying elevated filling pressures and diastolic dysfunction.

Increased troponin levels in adverse-outcome patients reflect more extensive myocardial necrosis, matching the findings of Caetano CC et al. (2024)[12]. Significantly lower platelet counts in the adverse-outcome group support Nerella S et al. (2022)[5], who linked thrombocytopenia with severe dengue cardiac involvement. Elevated CRP mirrors inflammatory burden and is consistent with the work of Bhattacharjee S et al. (2023)[13].

CONCLUSION

The present comparative study of 140 patients provides important insights into the distinct clinical, laboratory, and echocardiographic profiles of viral myocarditis and dengue-related myocarditis. Viral myocarditis was associated with more profound left ventricular systolic and diastolic dysfunction, greater chamber dilation, higher prevalence of global hypokinesia, longer ICU and hospital stay, and a higher though statistically nonsignificant mortality rate. In contrast, dengue-related myocarditis demonstrated relatively preserved systolic function, milder structural alterations, and significantly better recovery of left ventricular function at three months, reflecting the predominantly transient myocardial involvement typical of dengue infection. Predictors of adverse outcomes across the cohort included older age, viral etiology, reduced LVEF, elevated E/e′ ratio, higher troponin levels, increased inflammatory markers, and lower platelet counts.

Overall, the study highlights that while both viral and dengue myocarditis contribute substantially to cardiac morbidity, viral myocarditis tends to follow a more severe and prolonged course, whereas dengue myocarditis usually exhibits more favorable cardiac recovery. Early differentiation between these etiologies, guided by echocardiographic parameters and biochemical markers, is essential for timely risk stratification, appropriate monitoring, and individualized management strategies.

LIMITATIONS

1. The study was conducted at a single tertiary-care center, which may limit generalizability to other populations and healthcare settings.
2. A combination of retrospective and prospective data collection may have introduced variability in clinical documentation and diagnostic completeness.
3. Viral etiologies were not uniformly confirmed with advanced molecular testing (PCR or cardiac MRI), leading to potential underdiagnosis or misclassification of viral myocarditis.
4. Cardiac MRI the gold standard for tissue characterization was not feasible for all patients due to resource constraints, limiting the ability to validate echocardiographic findings.
5. Long-term follow-up beyond three months was not performed, restricting assessment of chronic remodeling, persistent dysfunction, or late arrhythmic events.
6. Treatment variations between clinicians were not controlled for and may have influenced outcomes such as LV recovery and hospitalization duration.
7. The impact of comorbidities, nutritional status, and concurrent infections was not fully evaluated, although these factors may influence myocardial recovery.
8. Sample size, although adequate for primary comparisons, may still be insufficient for detecting small differences in rare outcomes such as mortality.

REFERENCES

1. Baqi A, ur Rehman F, Memon PS, Omair SF. Prevalence and outcomes of myocarditis in dengue-infected patients admitted to a tertiary care hospital of low-middle income country. Global heart. 2022 Jun 23;17(1):44.
2. Mansanguan C, Hanboonkunupakarn B, Muangnoicharoen S, Huntrup A, Poolcharoen A, Mansanguan S, Piyaphanee W, Phumratanaprapin W. Cardiac evaluation in adults with dengue virus infection by serial echocardiography. BMC Infectious Diseases. 2021 Sep 10;21(1):940.
3. Nicacio JM, Gomes OV, Carmo RF, Nunes SL, Rocha JR, Souza CD, Franca RF, Khouri R, Barral-Netto M, Armstrong AD. Heart disease and arboviruses: a systematic review and meta-analysis. Viruses. 2022 Sep 8;14(9):1988.
4. Alnuaimi MK, Alnuaimi II MK. Dengue Fever-Induced Complete Heart Block (Cardiac Dengue) Requiring Permanent Pacemaker Implantation in a 53-Year-Old Man: A Case Report. Cureus. 2025 Apr 16;17(4).
5. Nerella S, Sarkar UK, Namdeo H. Electrocardiographic and echocardiographic findings in children with dengue infection. Journal of Family Medicine and Primary Care. 2022 Jun 1;11(6):2334-9.
6. Ingole K, Bhagat A. Case Report on Dengue Induced Myocarditis. Journal of Pharmaceutical Negative Results. 2022 Oct 8;13.
7. Huits R, Angelo KM, Amatya B, Barkati S, Barnett ED, Bottieau E, Emetulu H, Epelboin L, Eperon G, Medebb L, Gobbi F. Clinical characteristics and outcomes among travelers with severe dengue: a GeoSentinel analysis. Annals of internal medicine. 2023 Jul;176(7):940-8.
8. Politi MT, Vannoni G, Gagliardi J. Myocardial Infarction in Polycythemia Vera and Dengue: A Case of Competing Risks. Cureus. 2025 Mar 24;17(3).
9. Parveen S, Riaz Z, Saeed S, Ishaque U, Sultana M, Faiz Z, Shafqat Z, Shabbir S, Ashraf S, Marium A. Dengue hemorrhagic fever: a growing global menace. Journal of water and health. 2023 Nov 1;21(11):1632-50.
10. Singh V, Mishra SC, Agarwal NA, Mallikarjuna PA, Raut BB. Dengue infection with warning signs: The 2019 epidemic. medical journal armed forces india. 2022 Apr 1;78(2):140-6.
11. Shah TA, Khan US, Matin MF, Allin TD, Nongsiej BM. Dengue Fever Presenting with Concomitant Acute Coronary Syndrome (ACS)-A Series of 5 Cases At A Tertiary Care Hospital In Dhaka. Journal of Bangladesh College of Physicians and Surgeons. 2023 Nov 28:82-7.
12. Caetano CC, Azamor T, Meyer NM, Onwubueke C, Calabrese CM, Calabrese LH, Visperas A, Piuzzi NS, Husni ME, Foo SS, Chen W. Mechanistic insights into bone remodelling dysregulation by human viral pathogens. Nature microbiology. 2024 Feb;9(2):322-35.
13. Bhattacharjee S, Ghosh D, Saha R, Sarkar R, Kumar S, Khokhar M, Pandey RK. Mechanism of immune evasion in mosquito-borne diseases. Pathogens. 2023 Apr 23;12(5):635.

