A Rare Case of Dengue Myocarditis: Challenges in Diagnosis and Management

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ABSTRACT

Dengue fever is a viral infection prevalent worldwide in tropical and subtropical regions and affects millions of people annually. It is associated with several complications, including rare yet significant cardiac complications, such as myocarditis. Myocarditis caused by dengue fever is challenging to diagnose and can be initially mistaken for acute coronary syndrome. This case report describes the diagnostic and management difficulties encountered by examining a young male patient who developed myocarditis as a complication of severe dengue fever. A 30-year-old male, a known smoker, presented to the emergency department with chest pain, diaphoresis, and hypotension. ECG indicated inferior wall ST-T changes. A physical examination revealed no abnormalities. The patient had a five-day history of fever and myalgia. The patient was initially diagnosed with acute coronary syndrome and cardiogenic shock. Primary coronary angiography revealed no abnormal findings. Echocardiography revealed a decreased LV ejection fraction of 40% and global wall hypokinesia with normal chamber dimensions, necessitating further investigation of the etiology. Subsequently, he was diagnosed with dengue virus-induced myocarditis based on laboratory investigations. This case underscores the rarity of dengue-associated myocarditis and emphasizes the pivotal role of maintaining a high clinical suspicion index for early diagnosis and management. Additionally, it emphasizes the significance of continuous case reporting to enhance our understanding and shape the evidence-based practices to address this uncommon complication.

Keywords: Dengue fever, Myocarditis, Diagnosis, Management

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INTRODUCTION

Dengue fever is a mosquito-borne viral illness caused by flavivirus and is primarily transmitted by the Aedes aegypti mosquito. It can present with widespread clinical manifestations ranging from asymptomatic cases to severe and potentially life-threatening conditions.

The dengue virus (DENV) is an RNA virus consisting of four distinct serotypes. Notably, infection with one serotype does not confer immunity to others, thereby increasing the risk of severe dengue with subsequent infection.

In 2009, the World Health Organization classified dengue into three groups: dengue viral fever without warning signs, with warning signs, and severe dengue disease. Severe dengue virus infection involves severe bleeding, plasma leakage, and end-organ impairment.1

Although cardiac symptoms related to dengue are infrequent, cardiac rhythm abnormalities, including atrioventricular nodal blocks, sinus node dysfunction, atrial fibrillation, premature ventricular beats, as well as myocarditis, pericarditis and perimyocarditis, have been recorded during severe dengue fever.2

The global prevalence of dengue is staggering,
with an estimated 390 million cases occurring annually. Of these infections, approximately 96 million manifest with varying degrees of clinical or subclinical severity. The impact of dengue extends to diverse geographic locations. One of these is Pakistan, where dengue fever exhibits a seasonal endemic pattern, often surging during the monsoon season when conducive conditions for mosquito breeding are prevalent.

This report aims to increase awareness among the medical community about dengue-associated myocarditis through a detailed case presentation and discussion of diagnostic and management challenges. Promoting timely intervention for this uncommon cardiac manifestation can improve patient outcomes.

This report presents a case of myocarditis as a rare and unconventional cardiac manifestation of dengue infection in a 30-year-old male patient.

**CASE REPORT**

A 30-year-old male, a known smoker, presented to the emergency department with a two-hour history of typical chest pain accompanied by cold sweats, diaphoresis, and associated hypotension. Upon arrival, his vitals were as follows: blood pressure, 80/60 mmHg; heart rate, 120 beats/min; respiratory rate, 29 breaths/min; and oxygen saturation (SpO₂), 92%. The ECG showed changes in the inferior wall ST-T (Figure 1).

Figure 1 displays the patient's ECG with specific abnormalities highlighted. Red arrows indicate ST-T changes observed in the inferior wall leads, while a blue arrow highlights the presence of poor “R” wave progression in the anterior wall leads.

![Figure 1: Electrocardiogram (ECG) findings in the case report](image1)

Both views of two-dimensional echocardiography showed normal chambers but reduced LV ejection fraction.

During the general physical examination, the patient was alert and conscious. On cardiovascular assessment, he had no identifiable murmurs or other abnormalities. On chest auscultation, his chest was clear. He had no ascites on the abdominal examination. He had no pedal edema or any signs of fluid overload. Furthermore, the patient had a five-day history of high-grade fever and myalgia.

The medical team promptly initiated resuscitation by administering intravenous fluids. Based on the clinical presentation, comorbidities, and ECG findings, the medical team diagnosed the patient with acute coronary syndrome (ACS) and cardiogenic shock. According to the American Heart Association guidelines for ACS, the patient underwent drug loading.

Blood samples were simultaneously collected and sent for analysis. Subsequently, the medical
team transferred the patient to the catheterization laboratory for primary coronary angiography. Primary coronary angiography revealed no abnormalities, which warranted further investigation of the etiology of his presentation. Furthermore, echocardiography revealed a decreased LV ejection fraction of 40%, and segmental wall motion abnormalities showed normal chamber dimensions but global wall hypokinesia (Figure 2).

Laboratory data revealed elevated hemoglobin, mean corpuscular volume, hematocrit, and white blood cell count, but reduced platelet count. His metabolic panel also showed increased alanine transaminase, aspartate aminotransferase, and gamma-glutamyl transferase levels, but decreased serum albumin and total protein levels. Laboratory investigations sent for dengue included NS1-antigen and anti-dengue virus (IgM), along with cardiac troponin I, which was positive (Table 1).

The patient fully recovered from dengue fever, but experienced residual cardiac dysfunction at discharge. This table presents the numerical values of various laboratory test results conducted during the patient's evaluation.

DISCUSSION

The impact of dengue extends to diverse geographic locations; one region experiencing a complex interplay of dengue serotypes and epidemiological dynamics is Pakistan, where dengue fever is endemic. The World Health Organization (WHO) classifies dengue according to warning signs and clinical symptoms. The WHO criteria for severe dengue disease include severe plasma leakage (resulting in shock and fluid accumulation with respiratory distress), bleeding as evaluated by a clinician, and organ involvement (liver, central nervous system, heart, and other organs).

Following studies, one in Malaysia consisting of 700 patients and another comprising 1245 patients by Zhang, reported the prevalence of severe dengue at 4.9% and 5.6%, respectively. A review of the incidence of cardiovascular manifestations of dengue fever reported that 46.1% of patients exhibited cardiac symptoms ranging from frequent ECG abnormalities to rare myocarditis and pericarditis. In A prospective study on 81 patients reported cardiac involvement in 22% of the population and a 2.4% prevalence of myocarditis. Another recent retrospective observational study to record the prevalence of myocarditis in dengue fever, consisting of 1008 patients diagnosed with dengue viral infection, conducted in Pakistan by Baqi, reported a 4.2% prevalence of dengue myocarditis.

However, the infrequent presentation, diagnosis, and reporting of such cases in clinical settings make it difficult to determine a precise figure. The difference between these prevalence rates can be attributed to the under-diagnosis of cardiac involvement in dengue owing to the low

Table 1: Laboratory test results of the patient in the case report

<table>
<thead>
<tr>
<th>Laboratory Test</th>
<th>Patient Value</th>
<th>Units</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>18.6</td>
<td>gm/dl</td>
<td>14.00 – 18.00</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>58.8%</td>
<td>%</td>
<td>42.00 – 47.00</td>
</tr>
<tr>
<td>Mean Corpuscular Volume</td>
<td>95.3 fL</td>
<td>fL</td>
<td>77.00 – 93.00</td>
</tr>
<tr>
<td>Platelet count</td>
<td>101 ×10. e 3/uL</td>
<td>U/L</td>
<td>150.00 – 450</td>
</tr>
<tr>
<td>White Blood Cell count</td>
<td>18.94</td>
<td>×10. e 3/uL</td>
<td>4.00 – 11.00</td>
</tr>
<tr>
<td>Alanine Transaminase</td>
<td>136 U/L</td>
<td>U/L</td>
<td>5.00 – 40.00</td>
</tr>
<tr>
<td>Aspartate Aminotransferase</td>
<td>185 U/L</td>
<td>U/L</td>
<td>5.00 – 40.00</td>
</tr>
<tr>
<td>Gamma G. T.</td>
<td>136 U/L</td>
<td>U/L</td>
<td>5.00 – 50.00</td>
</tr>
<tr>
<td>Serum Albumin</td>
<td>3.1 g/dl</td>
<td>g/dl</td>
<td>3.50 – 5.00</td>
</tr>
<tr>
<td>Total Serum Protein</td>
<td>5.7 g/dl</td>
<td>g/dl</td>
<td>6.30 – 8.30</td>
</tr>
<tr>
<td>NS1 Antigen (Dengue)</td>
<td>3.99</td>
<td>&lt;0.9</td>
<td></td>
</tr>
<tr>
<td>Anti Dengue Virus (IgM)</td>
<td>2.03</td>
<td>&lt;0.9</td>
<td></td>
</tr>
<tr>
<td>Troponin I</td>
<td>3.053 ng/ml</td>
<td>ng/ml</td>
<td>&lt;0.08</td>
</tr>
</tbody>
</table>
index of clinical suspicion and the similarity of its clinical manifestations.

Against this backdrop, our focus shifted to dengue-associated myocarditis, highlighting the importance of such cases. Although this complication is well known to the medical community, delayed diagnosis and untimely management can have grave consequences. In this case, the initial presentation mimicked acute myocardial infarction, leading to primary coronary angiography that ruled out coronary artery disease. Echocardiography revealed global wall hypokinesia with a reduced left ventricular ejection fraction. These findings led to further investigation of the causes of the cardiomyopathy. Further investigations, including fever and myalgia history, positive NS1 antigen, anti-dengue virus (IgM), and elevated cardiac troponin I, supported the diagnosis of clinically suspected viral myocarditis according to the European Society of Cardiology criteria (two positive clinical criteria: acute chest pain and shock, and three positive diagnostic criteria: ECG, echocardiography, and raised cardiac troponin). ESC requires at least one clinical and diagnostic criterion for diagnosis.9

A significant challenge in dengue-associated myocarditis is the accuracy of diagnosing this elusive cardiac complication. Myocarditis can clinically present as acute coronary syndrome, acute heart failure, or cardiogenic shock. A few reported cases revealed typical presentations of acute myocardial infarction in younger patients who were later diagnosed with dengue-associated myocarditis or myopericarditis.10,11 Imaging modalities, including echocardiography and cardiac MR, helped diagnose these patients. The precise pathophysiological mechanism of dengue virus-induced myocarditis remains unclear, and limited data are available. Two studies have suggested a direct viral invasion of cardiac myocytes and interstitial and endothelial cells but could not establish a definitive relationship.12,13

There is no specific treatment or management for dengue-associated myocarditis apart from continuous monitoring and supportive care with adequate intravenous fluid resuscitation while considering hematocrit and avoiding fluid overload. Inotropes are the mainstay of treatment if there is inadequate response to the fluids in cases of severe shock.14 The cardiology team managed the patient conservatively, using fluids and inotropic support until he achieved complete recovery.

Shifting the focus to diagnostic insights, in a cohort study of 217 patients with dengue fever, a 24% incidence of myocarditis was reported on echocardiographic findings without clinical signs and symptoms of cardiac involvement but with relative bradycardia. Echocardiographic findings included left and right chamber dilatation, tricuspid regurgitation, and adequate ejection fractions. The cardiac abnormalities improved in the weeks following recovery from the illness. The results suggest that dengue-induced cardiac complications run a benign course and can be detected earlier.15

CONCLUSION

Dengue fever is prevalent in tropical and subtropical regions and exhibits various clinical manifestations, the myocarditis as a cardiac complication is a relatively unusual. Timely recognition and management of dengue myocarditis are imperative to avert severe complications. To achieve this goal, keeping a high index of clinical suspicion and conducting an early screening for cardiac abnormalities in dengue patients is essential.
REFERENCES


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RH: Design of the work, reviewing it critically for important intellectual content, approved the final version be published
MJA: Interpretation of data for the work, proofreading, approval of the final draft, approved the final version be published

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