

Case Report

Fulminant Myocarditis: A Very Rare Manifestation of Dengue Fever

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A B S T R A C T

Dengue is a highly prevalent arthropod-borne viral disease in tropical and subtropical areas of the globe. The majority of dengue cases are benign but rare complications and presentations are seen increasingly due to the rising burden of the disease mainly in tropical countries. In dengue, clinical manifestations vary from asymptomatic infection to severe disease. Dengue fever is characterised by fever, myalgia, arthralgia, headache and bleeding manifestations. We report a rare case of dengue fever complicated with acute myocarditis. The patient recovered gradually with supportive care.

Keywords: Dengue Fever, Fulminant Myocarditis, Systolic Dysfunction, Inotropes

Introduction

Dengue represents an important public health problem. There are four serotypes - DENV1, DENV2, DENV3, and DENV4. It is transmitted by *Aedes aegypti* and *Aedes albopictus*. A typical manifestations include Guillain Barre Syndrome, fulminant hepatitis, and cardiogenic shock. Fulminant myocarditis is a rapidly evolving form of acute myocarditis with associated cardiogenic shock requiring inotropes or mechanical circulatory support.¹ In this case report, we present a patient who developed fulminate myocarditis during the course of dengue fever.

Case Report

A 46-year-old female with no comorbid illness presented to our hospital with fever and myalgia. Blood investigations revealed a total count of 2500 cells/cm³ and a platelet

count of 49,000/cm³. She was admitted with a provisional diagnosis of tropical fever syndrome and necessary investigations were sent. NS1 and IgM Elisa for dengue turned out to be positive. She was treated with supportive measures. On the second day, she developed retrosternal chest pain and breathlessness. ECG showed ST depression and T wave inversion over precordial leads V4-V6, Lead 1, Lead 2, aVL and aVF (Figure 1). Chest X-ray showed features of pulmonary edema (Figure 2). Cardiac enzymes were elevated (serum troponin was 2830 ng/ml). Echo showed global hypokinesia of the left ventricle with regional variation and severe LV systolic dysfunction with EF of 30%. The patient had features of myocarditis. She was treated with noradrenaline infusion and low-dose loop diuretics for cardiac failure. The patient had worsening breathlessness and was started on NIV and dobutamine. The patient's

condition gradually improved and she was weaned off from inotropes. On day 10, an angiogram was done which showed normal coronary arteries. Cardiac MRI done on day 12 showed delayed post-contrast images suggestive

of myocardial necrosis or fibrosis (Figure 3). There was a faint enhancement in midmyocardium in nearly all left ventricular segments, more so, in mid-cavity level consistent with resolving myocarditis.

Table I. Summary of Clinical Manifestations of Fulminant Myocarditis in Dengue affected Patients

| S. No. | Author, Year of Publication | Gender | Age (Years) | Co morbid | Ejection Fraction (%) | CAG | Cardiac MRI | Myo-cardial Biopsy | Dengue Sero-type | Out-come | Treat-ment |
|--------|---------------------------------------|------------------------------|----------------------|-----------------|-----------------------|------------------|--|---|------------------|-----------------------|--|
| 1. | Lee IK et al., 2008 ² | M | 25 | Nil | 20 | Normal | NA | NA | NA | Died | Left ventricular assist device support |
| 2. | Patra S et al., 2012 ³ | F | 22 | Nil | 40 | NA | NA | NA | NA | Survived | Inotropes |
| 3. | Marques N et al., 2013 ⁴ | M | 41 | Nil | NA | NA | NA | Autopsy-Myocardial necrosis | DENV-2 | Died | Inotropes |
| 4. | Miranda CH et al., 2013 ⁵ | F | 37 | Alport syndrome | 30 | NA | NA | Diffuse foci of myocytolytic necrosis | NA | Died | Mechanical ventilation |
| 5. | Yu SH et al., 2014 ⁶ | F | 57 | SHTN | NA | LCX-50% stenosis | NA | NA | NA | Survived | ECMO |
| 6. | Ramathan K et al., 2015 ⁷ | F | 33 | Nil | 20 | NA | Diffuse sub-epicardial enhancement at midcavity | NA | DENV-2 | Survived | Inotropes |
| 7. | Ku YH et al., 2016 ⁸ | F | 52 | Nil | 34 | Normal | NA | NA | NA | Died | ECMO |
| 8. | Singh B et al., 2019 ⁹ | M | 22 | Nil | 25 | Normal | NA | NA | NA | Survived | Inotropes and diuretics |
| 9. | Teyseyre L et al., 2021 ¹⁰ | No of cases -4 F-2 M-2 | F-47, 63 M-29, 52 | T2DM | 20 | NA | Acute myocarditis | Myocardial biopsy showed inflammatory signs | DENV-2 | 2 survived and 2 died | ECMO |
| 10. | Our case | F | 47 | Nil | 30 | Normal | Enhancement in mid-myocardium & pericardial effusion | NA | NA | Survived | Inotropes and diuretics |

M-Male, F-Female, EF-Ejection fraction, CAG-Coronary angiography, LCx-Left circumflex artery, DEN-Dengue serotype, NA-No data available/Not applicable, ECMO-Extracorporeal membrane oxygenation

The follow-up echocardiogram after a week showed normal left ventricular function (ejection fraction of 60%) with no regional wall motion abnormalities.

Literature Review

We searched the PubMed database for articles published about dengue fever and fulminant myocarditis between 2000 and 2021. The information retrieved includes the year of publication, demographic details, dengue serotype, echocardiographic findings, angiogram, myocardial biopsy, clinical outcome, and treatment details. These are summarised in Table 1. There have been only 14 cases of fulminant myocarditis reported so far in PubMed between 2000 and 2021. In our case studies, risk factors for fulminant myocarditis include old age and the presence of co-morbid illnesses such as diabetes mellitus, hypertension, and asthma. Of the 14 patients reported, only five survived despite providing newer treatment modalities like Intra aortic balloon pump (IABP) and extracorporeal membrane oxygenation (ECMO).

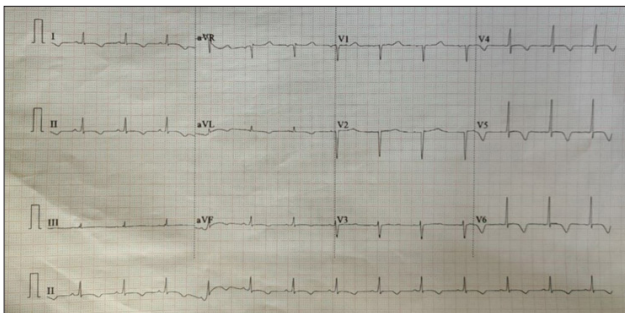


Figure 1. 12 Lead Electrocardiogram shows ST-segment Depression and T Wave Inversion over Precordial Leads V4-V6, Lead I, Lead 2, aVL and aVF



Figure 2. Chest X-ray Showing of Pulmonary Edema

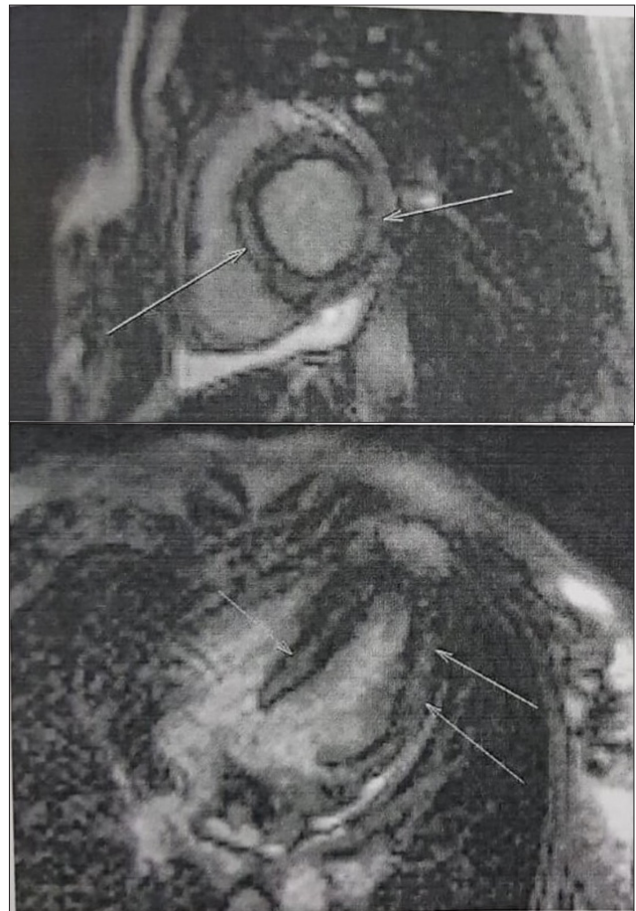


Figure 3. Mid-cavity Short-axis Late Gadolinium Image of the Heart Heart obtained 5 Minutes after Administration of Intravenous Contrast. In Delayed Post Contrast Images, There is a Faint Enhancement in Midmyocardium in Nearly all Left Ventricle Segments, More so in Mid-cavity Level and Minimal Pericardial Effusion Noted

Discussion

Acute fulminant myocarditis is a life-threatening emergency. Criteria to diagnose fulminant myocarditis include 1) Rapid onset of symptoms of heart failure within two weeks of an inciting event, 2) Prodromal symptoms of upper respiratory or gastrointestinal viral infection, 3) Rapid hemodynamic instability requiring a large dose of inotropes, 4) Cardiac magnetic resonance (CMR) or endomyocardial biopsy (EMB) proven myocarditis, and 5) Exclusion of other cardiac diseases like coronary artery disease. Fulminant myocarditis is diagnosed by symptoms, ECG and ECHO changes, CMR and EMB. The gold standard for diagnosis is an endomyocardial biopsy. CMR is an alternative choice if EMB is not feasible. In CMR, early enhancement is a sign of hyperemia and late enhancement indicates necrosis. Late gadolinium enhancement in CMR was observed more frequently in patients with fulminant myocarditis than in those with non-fulminant myocarditis (80% vs 20%). Patients

presenting with fulminant myocarditis (defined as initial left ventricular ejection fraction [LVEF] < 50%, low cardiac output syndrome requiring inotropes) are at increased risk for adverse cardiac events. Inotropic agents are the first-line drugs administered to enhance cardiac pumping function. In patients not responding to inotropes, mechanical circulatory devices like intra-aortic balloon pumps (IABP) and extracorporeal membrane oxygenation (ECMO) can be used. The role of steroids in fulminant myocarditis remains unknown. Myocarditis patients with heart failure refractory to inotropic agent/IABP/ECMO can be considered for cardiac transplantation.¹ Prompt triage and a high index of suspicion are vital to provide aggressive treatment. Recognition of patients with fulminant presentation is potentially lifesaving as more than half can survive without sequelae.

Conclusion

Though cardiac manifestations are common in dengue, myocarditis is rare. Fulminant myocarditis is one of the rare entities of myocarditis which is associated with a rapid downhill course if not recognised early and treated promptly. The upsurge in serum TNF-alpha, interleukin-6, 13 and 18 causes increased vascular permeability which leads to the development of shock in the patients. Further studies are needed to clarify whether these cytokines play a role in the development of myocardial cell injury and also the role that DEN serotype plays. With an increasing incidence of dengue fever, newer treatment strategies are needed for the management of fulminant myocarditis and improvement of the outcome.⁸ We present this case for its rare entity and fulminant presentation which improved with supportive management because of timely diagnosis.

Contributors

We were all involved in caring for the patient and editing and approving the final manuscript. Written consent for publication was obtained from the patient.

Conflict of Interest: None

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