



## Clinical case report based study

## Acute myocardial infarction being the presentation of dengue myocarditis

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## ABSTRACT

Dengue fever is one of the most common vector-borne viral infections in tropical countries. Myocarditis is an uncommon complication of severe dengue fever. Sometimes, dengue myocarditis masquerades as acute myocardial infarction. We report here a case of a 22-year-old female, who presented with acute chest pain five days after the onset of a febrile episode. Initially she was diagnosed as acute myocardial infarction depending on the biochemical and echocardiography evidence. However, later investigation confirmed she was suffering from myocarditis due to dengue fever, which mimicked acute myocardial infarction. A Medline search revealed only few other reported cases of dengue myocarditis that mimicked of acute myocardial infarction.

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## 1. Introduction

Dengue virus (DENV) infection is caused by one out of four antigenically distinct, but related single-stranded, positive-sense Ribonucleic acid (RNA) viruses in the family of *Flaviviridae*. Dengue is a worldwide condition, spread throughout the tropical and subtropical zones, between 30°N and 40°S. This virus is transmitted by mosquito vectors, primarily *Aedes aegypti*.<sup>1</sup> DENV infections cause a broad spectrum of illnesses from self-limited fever to severe hemorrhagic manifestations and increased vascular permeability.<sup>2</sup> Cardiac manifestations of dengue are rarely associated with severe dengue fever. Cardiac conduction disorders such as atrioventricular blocks, sinus node dysfunction, and ectopic ventricular beats have been reported during episodes of Dengue Hemorrhagic Fever (DHF).<sup>3</sup> Myocarditis is a rare complication of DENV and it rarely mimics acute myocardial infarction (MI). We are reporting a case in which the patient presented as acute myocardial infarction and later was diagnosed as a confirmed case of dengue myocarditis.

## 2. Case report

A 22-year-old female presented with acute onset of chest and abdominal pain a few hours before hospital admission. She had a

history of fever, with body ache and joint pain, about seven days back. It lasted for three days. She was apparently all right in between, until she had started this chest and abdominal pain. The pain was associated with vomiting and respiratory distress. She also had generalized swelling of the body with nonpruritic body rash, which started two days before admission. She did not have any history of bleeding, cough, or altered sensorium. She did not have any significant past history of a cardiac problem. A general physical examination revealed low-grade fever (38 °C), erythematous confluent maculopapular body rash, facial puffiness, bipedal edema, tachycardia (sinus, heart rate 120 beats/min), and hypotension (90/70 mmHg). Her oxygen saturation on room air was 88%. The cardiac examination revealed presence of a third heart sound, without any murmur or added sound. She had bilateral basal crackles on respiratory system examination. On abdominal examination, she was found to have tender hepatomegaly. Other system examinations did not reveal any significant findings. A twelve-lead electrocardiogram (ECG) showed ST-segment elevation in leads V2–V4 with T-wave inversion in leads II, III, and a VF [Fig. 1]. The bedside troponin *t*-test was positive. The chest X-ray showed pulmonary congestion with bilateral minimal pleural effusion. Echocardiography was done, and it showed anteroseptal area hypokinesis with global left ventricular (LV) dysfunction and an ejection fraction of 40%. From the above clinical and investigational findings, she was presumptively diagnosed as having acute myocardial infarction, heart failure, and cardiogenic shock. Her biochemistry report revealed that the serum levels of creatine

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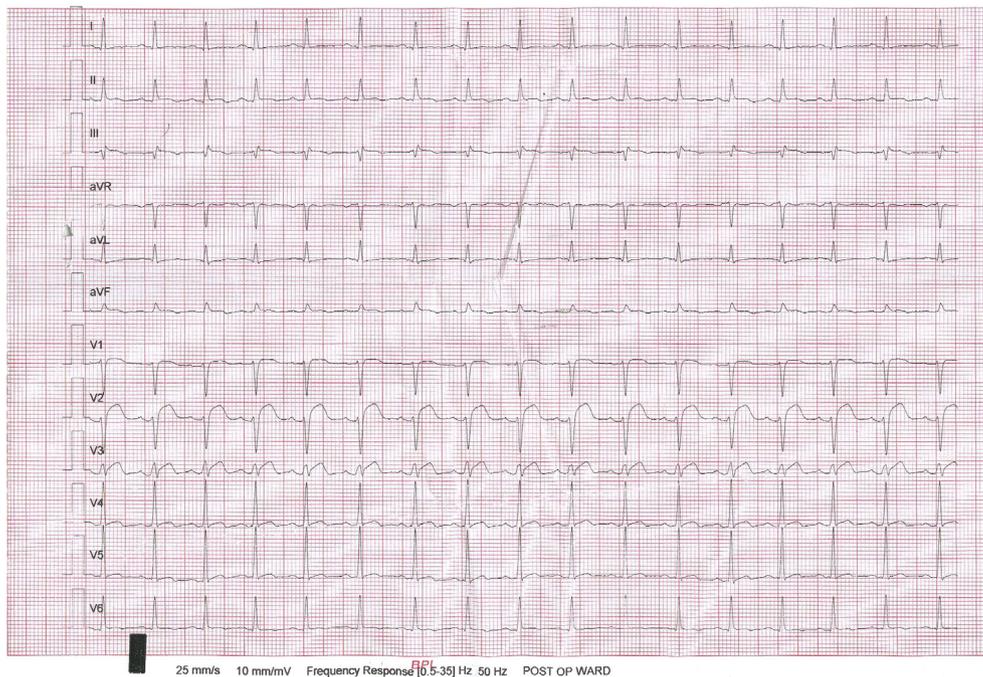


Fig. 1. Electrocardiogram showed ST-segment elevation in leads V2–V4 with T-wave inversion in the inferior leads.

kinase (422 U/L, normal: 30–350), creatine kinase MB fraction (45.0  $\mu\text{g/L}$ , normal: 1.0–6.0), and troponin T (1.48  $\mu\text{g/L}$ , normal:  $<0.01$ ) were elevated. The blood count showed leukopenia (4000/cmm), thrombocytopenia (56,000/cmm), and anemia (Hb-10.3 grams/dL). The liver function tests showed that hypoalbuminemia (albumin-3 grams/dL) and renal function tests were within normal limits, and the blood clotting profile was also normal. As she had a history of fever few days prior, along with thrombocytopenia, body rash, generalized edema, and hypoalbuminemia, a dengue serology was sent. Blood test was positive for Dengue IgG and IgM antibodies (by enzyme-linked immunosorbent assay (ELISA)), confirming a diagnosis of dengue fever. She was treated with vasopressor (inj. Dopamine and inj. Dobutamine). During the course of her hospital stay, her platelet count improved and repeats echocardiography three days after admission showed only LV global hypokinesia, without any regional wall motion abnormalities. Her blood pressure improved, respiratory distress diminished, and finally she was off vasopressor after the fifth day of her hospital stay. Although she was planned for a coronary angiogram after normalization of her platelet count, she refused and was discharged on the seventh day after admission.

Thus, from the above-cited clinical and investigational evidence, she was finally diagnosed as dengue myocarditis rather than acute MI. In this case, dengue myocarditis was masquerading as acute MI.

### 3. Discussion

Depression of myocardial function has been reported to be associated with the hemorrhagic form of DENV infection. Myocardial involvement in dengue may result either from direct DEN invasion of the cardiac muscles or a cytokine-mediated immunological response, or both.<sup>4</sup> Myocardial dysfunction has been reported to be more severe in patients with dengue shock syndrome (DSS) when compared to those with dengue fever (DF) or non-shock Dengue Hemorrhagic Fever (DHF).<sup>5</sup> Cardiac arrhythmia such as atrioventricular block and sinus node dysfunction, as well as, reversible myocarditis has been reported in patients with DF; most of these are self-limiting.<sup>3</sup> Ventricular dysfunction is also

associated with the acute phase of DHF. Approximately, 16% of the patients with DF have ejection fractions of less than 50%, and 70% of the patients with DHF or DSS have diffuse ventricular hypokinesia.<sup>5</sup> Fatal dengue myocarditis is a very rare complication of dengue fever.<sup>6</sup> However, global LV hypokinesia is usually seen in dengue myocarditis. Although, ST-segment and T-wave changes in the electrocardiogram (ECG) along with regional wall motion abnormality, mimicking acute MI, as a presentation, is very rare.<sup>7,8</sup> In the Medline search, like in our case, only two similar cases of dengue myocarditis mimicking acute MI have been reported so far.<sup>7,8</sup> The first one reported by Lee et al,<sup>7</sup> was a 25-year-old male, who presented with acute pulmonary edema and cardiogenic shock. He had ECG features suggestive of anterior wall MI, and died. Another case reported by Lee IK et al,<sup>8</sup> was a 65-year-old female who presented with DHF. She had significant ST-segment depression in the chest leads V3–V6, but survived. However, only in our case, the patient presented with acute chest pain following a febrile illness and other features suggestive of acute MI, like elevation of cardiac biomarker and ECG and echocardiographic changes. However, as she had a history of fever, joint pain followed by rash, and blood investigations suggestive of some viral infection, like thrombocytopenia, leucopenia, we investigated for dengue fever, which came out as positive.

In summary, fulminant myocarditis can complicate dengue fever, and the associated electrocardiographic changes can mimic acute myocardial infarction. Even though the patient presents with acute chest pain and features of acute MI, if the patient has had other history and clinical features suggestive of dengue fever; myocarditis must be kept in mind before starting treatment for acute MI. Although it was self-limiting in our patient, under supportive treatment, acute myocarditis in dengue can be clinically severe to such an extent that it can have a fatal outcome.

### Author contribution

Soumya Patra, Gaurav Bhardwaj, J. S. Manohar & K.H. Srinivasa were involved in the diagnosis and management of the patient. Soumya Patra reviewed the literature and drafted the manuscript.

J.S. Manohar, K.H. Srinivasa & C.N. Manjunath corrected the manuscript. All authors approved the final version of the manuscript.

### Conflicts of interest

All authors have none to declare.

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