CASE REPORT

Chest discomfort in a patient with dengue – is it an acute myocardial infarction?

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Abstract

Cardiovascular symptoms presenting in a patient with dengue fever may post a diagnostic dilemma. We describe a case of dengue myocarditis mimicking an acute myocardial infarction in a 56-year-old woman.

Introduction

Myocarditis is an inflammation of the myocardium. Viral infection is one of the most common causes of myocarditis. Although rare, dengue has been reported to cause myocarditis.[1] The exact pathophysiology of myocardial cell injury in dengue remains unknown, although it is thought to be from direct viral invasion of the cardiac muscles , a cytokine-mediated immunological response, or both.[2,3]

Diagnosing myocarditis is challenging due to its polymorphic presentations with no pathognomonic signs or symptoms, which can range from asymptomatic subclinical presentation to mild fatigue, lethargy, chest pain, or even complications such as heart failure, cardiogenic shock, cardiac arrhythmias, and death. We describe a woman who presented with typical angina chest pain following an uneventful bout of dengue.

Case Report

Madam MLC, a 56-year-old woman, presented with complaints of chest discomfort, progressive shortness of breath, leg swelling, abdominal fullness, and decreased effort tolerance for one day. There was an absence of chest pain and profuse sweating. Other than being post-menopausal, she had no other cardiovascular risk factors. Six days prior to the onset of these symptoms, she had presented with a high-grade fever at a private hospital and been diagnosed with dengue fever (positive NS1 antigen). She was hospitalized and, following an uneventful stay, allowed to return home after six days.

At presentation, she was conscious, alert, and afebrile. Her blood pressure and pulse rate were 100/60 mmHg and 120 /min, respectively. Her oxygen saturation was 99 % with room air. Her jugular venous pulse was raised, and there was bilateral pedal oedema. Precordial examination was unremarkable but crepitations were audible at the lung bases.

An electrocardiogram (ECG) revealed sinus tachycardia with a rate of approximately 100/min; saddle-shaped ST-segment elevation greater than 2mm in leads II, aVF, V4, V5, and V6 without reciprocal ST depression, and diminished QRS amplitude in several limb and chest leads (**Figure 1**). A chest radiograph confirmed the presence of minimal bilateral pleural effusion. Echocardiography revealed minimal pericardial effusion with no evidence of cardiac tamponade and an ejection fraction of 60%. The chambers sizes were normal with no hypokinetic areas. Her troponin I level was elevated (10.51 ng/ml; reference < 0.04 ng/ml) along with the other cardiac markers. A summary of relevant laboratory results is shown in **Table 1**.

A clinical diagnosis of dengue myocarditis with acute left heart failure was made. She was administered supplemental oxygen and an intravenous diuretic (furosemide) and advised to have complete bed rest. Serial ECGs done up to 12 hours post-admission showed similar ST-segment elevations with no Q wave formation. Serial aspartate transaminase (AST) showed a downward trend to normalization by day 7 of hospitalization. She was discharged after 12 days of uneventful hospitalization with the advice to avoid strenuous exercise till her next review at the outpatient clinic.

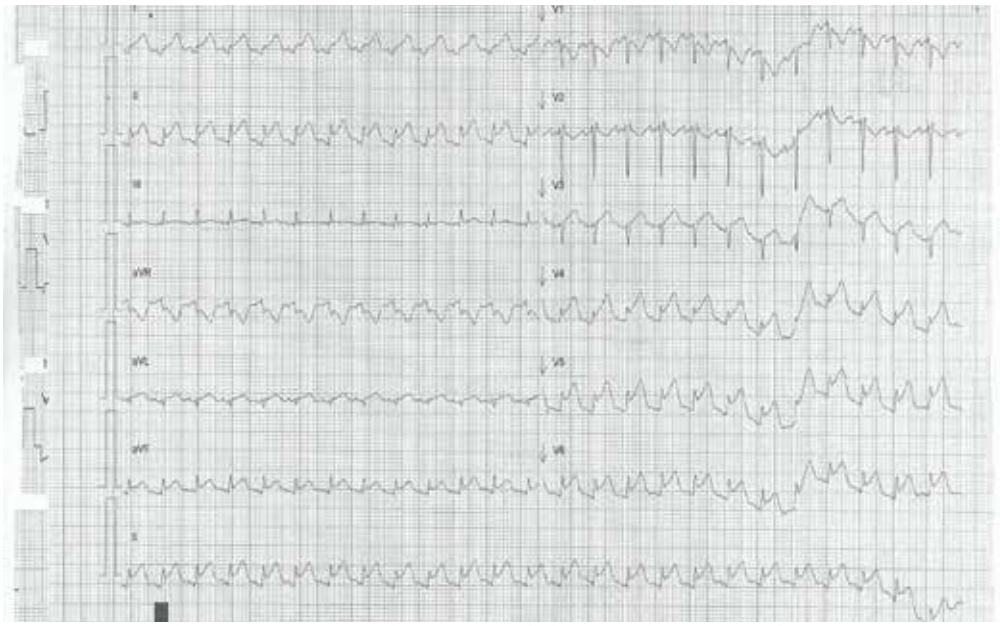

Figure 1: ECG showing ST segment elevation revealed greater than 2mm in leads II, aVF, and V4 to V6.

Table 1: Laboratory results

Laboratory parameter	Reference	Day of hospital stay						
		1	2	3	4	5	6	7
White blood cell (10^9/L)	4.5 – 11	12.6	14.1		10.6		7.7	7.5
Hemoglobin (g/L)	12 – 16	13.5	13.3		11.7		10.2	10.3
Hematocrit (%)	36 – 46	40.9	39.8		35.2		30.2	31.0
Platelet (10^9/L)	150 – 400	171	237		269		304	343
Aspartate transaminase (AST) (U/L)	8 – 20	206	219	150	105	79	51	41
Lactate dehydrogenase (U/L)	45 – 90				520			
Creatine kinase (U/L)	10 – 70				254			
Troponin I (ng/ml)	< 0.04	10.51						
Dengue IgM		equi-vocal						

Discussion

The clinical presentation of myocarditis is highly variable, often mimicking other non-inflammatory cardiac disorders. Our patient presented with features of acute heart failure (AHF) at the tail end of an uneventful dengue infection. The differential diagnoses would include acute myocardial infarction (AMI) and dengue myocarditis. A high level of clinical suspicion is required to diagnose the latter.

Laboratory markers of myonecrosis, such as creatine kinase (CK-MB), troponin I or T (cTI or CTT), lactate dehydrogenase (LDH), alanine transaminase (ALT), and aspartate transaminase (AST), are elevated in myocarditis. Troponin I (cTI) is often markedly elevated in the early phase of the disease and is suggestive of acute myocarditis. However, the magnitude of cTI elevation is not related to survival.[4] AST is considered the most sensitive marker of myocarditis with a sensitivity of 85%, although its specificity is relatively low.[5,6] In addition, C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) are typically elevated with leukocytosis. In our patient, cardiac markers were elevated, with raised AST and leukocytosis (**Table 1**).

ECG changes characteristic of myocarditis include saddle-shaped ST-segment elevations, which are usually diffused. This diffusion is typically absent in lead VI without the reciprocal ST depressions usually seen in ST-elevation myocardial infarction (STEMI) and diminished QRS amplitudes.[7] In addition, the ST elevation is typically < 4mm high and

does not occur simultaneously with T-wave inversions, unlike STEMI. Other ECG changes associated with myocarditis include abnormal Q waves, transient second- or third-degree heart block, prolonged QRS or QT, ventricular arrhythmias, and AV conduction defects.[7]

Although the ECG changes and raised troponin I level in our patient were in favor of AMI, the preceding history of a viral infection and absence of chest pain and profuse sweating prompted consideration of the alternative diagnosis of dengue myocarditis. The lack of Q-wave formation in serial ECGs in this patient provided another vital clue that she did not suffer an AMI.

Nevertheless, the diagnosis of dengue myocarditis is challenging due to its non-specific clinical presentation and the lack of a safe and sensitive non-invasive diagnostic test. Endomyocardial biopsy remains the 'gold standard' diagnostic test, but the procedure is invasive and not commonly done. Post-viral myocarditis should be considered when a patient presents with or without cardiac symptoms and signs, such as raised cardiac markers, ECG changes suggestive of acute myocardial injury, and arrhythmia or cardiac function abnormalities on an echocardiography, with a history of viral illness.[8]

Treatment of dengue myocarditis depends on its presentation. General measures in this patient included heart failure therapy through judicious use of diuretics; oxygen supplementation, as hypoxia may, at least in in vitro studies, aggravate myocarditis5; and prolonged rest, as in vitro studies have shown exercise to be associated with increased viral replication in the myocardium. Nonsteroidal anti-inflammatory drugs are not recommended and have been shown to exacerbate myocarditis.[9]

The ability to recognize dengue myocarditis is valuable in avoiding misdiagnosis. The abnormal ventricular function in dengue myocarditis generally resolves rapidly, and patients generally have good outcomes, although fatal complications have been reported.[3]

References

1. Obeyesekere I, Hermon Y. Myocarditis and cardiomyopathy after arbovirus infections (dengue and chikungunya fever). *Br Heart J*. 1972; 34: 82I-27.
2. Lee IK, Lee WH, Liu JW, Yang KD. Acute myocarditis in dengue hemorrhagic fever: a case report and review of cardiac complications in dengue-affected patients. *Int J Infect Dis*. 2010; 14(10): e919-22.
3. Miranda CH, Borges M de C, Schmidt A, et al. A case presentation of a fatal dengue myocarditis showing evidence for dengue virus-induced lesion. *Eur Heart J Acute Cardiovasc Care*. 2013; 2(2): 127-130.
4. Imazio M, Demichelis B, Cecchi E, et al. Cardiac troponin I in acute pericarditis. *J Am Coll Cardiol*. 2003; 42(12): 2144-48.
5. Lauer B, Niederau C, Kühl U, et al. Cardiac troponin T in patients with clinically suspected myocarditis. *J Am Coll Cardiol*. 1997; 30(5): 1354-59.
6. Soongswang J, Durongpisitkul K, Ratanarapee S, et al. Cardiac troponin T: its role in the diagnosis of clinically suspected acute myocarditis and chronic dilated cardiomyopathy in children. *Pediatr Cardiol*. 2002; 23(5): 531-35.
7. Nakashima H, Honda Y, Katayama T. Serial electrocardiographic findings in acute myocarditis. *Intern Med*. 1994; 33(11): 659-66.
8. Caforio AL, Calabrese F, Angelini A, et al. A prospective study of biopsy-proven myocarditis: prognostic relevance of clinical and aetiopathogenetic features at diagnosis. *Eur Heart J*. 2007; 28(11): 1326.
9. Caforio AL, Pankuweit S, Arbustini E, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J*. 2013; 34(33): 2636-48.