A 33-year-old woman presented to the emergency department with giddiness. She had a history of fever and vomiting 4 days before, which had completely resolved. Her medical history was otherwise unremarkable. She was leukopenic (leukocyte count 4.6×10⁹/L), lymphopenic (0.47×10⁹/L), and thrombocytopenic (89×10⁹/L), and she remained hypotensive (mean arterial pressure, 45 mm Hg) despite 3 L of fluid resuscitation. A 12-lead ECG showed hyperacute ST changes in the inferolateral leads (Figure 1). She was initially commenced on intravenous norepinephrine and epinephrine that was later changed to dobutamine. A transthoracic echocardiogram (Figure 2) revealed severe global left ventricular dysfunction (ejection fraction, ≤20%) with moderate pericardial effusion and no cardiac tamponade (Movies I and II in the online-only Data Supplement). The serum troponin I level was 9.3 μg/L (reference range <0.039 μg/L). This rose to 11.3 μg/L within 12 hours. Given the clinical, biochemical, and echocardiographic findings, she was evaluated for viral myocarditis and was shifted to the intensive care unit for observation and consideration of early mechanical cardiac support. The dengue nonstructural protein (NS1) serology was positive, and subsequent serotyping showed she had dengue virus serotype-2 (DEN-2). Cardiac MRI showed severely impaired left ventricle systolic function (ejection fraction, 19%; cardiac index, 1.12 L·min⁻¹·m⁻²). There was diffuse subepicardial enhancement at the midcavity and apical levels in the late gadolinium sequence with a moderate-sized pericardial effusion (Figures 3 and 4).

The patient developed ischemic hepatitis and bilateral lower limb petechiae with falling platelet count (to 49×10⁹/L). However, serial hematocrit measurements remained stable, suggesting no signs of ongoing hemorrhage. She required intermittent noninvasive ventilation for acute heart failure. However, her inotropes were successfully weaned after 5 days, and she was subsequently transferred to the general ward. A follow-up transthoracic echocardiogram (Figure 5) showed improved left ventricular function (ejection fraction, 65%) with left ventricular wall thickening and no pericardial effusion (Movies III, IV, and V in the online-only Data Supplement).

Dengue is an emerging global infectious disease and atypical manifestations are increasingly being reported. Dengue is an arboviral disease caused by a flavivirus transmitted by the *Aedes aegypti* mosquito. Dengue virus has 4 antigenically distinct serotypes (DEN 1, DEN 2, DEN 3, and DEN 4), and the disease is endemic in South and Southeast Asia, Central and Latin America, and Africa. The World Health Organization estimates that 40% of the world’s population living in tropical and subtropical urban regions are at high risk of getting dengue infection. The onset of dengue illness is abrupt and patients with moderate to severe dengue classically go through 3 phases: an initial phase with fever and dehydration; a critical phase manifesting as shock from plasma leakage, hemorrhage, or organ damage; and a phase of recovery marked by reabsorption of extravascular water. An estimated 1% to 5% of patients presenting to hospital with dengue develop complications including end-organ failure, coagulopathy, and capillary leak.

Transient myocardial depression can occur in dengue shock syndrome, a severe form of dengue, which manifests as circulatory shock with hemorrhagic complications (dengue hemorrhagic fever). Pericardial involvement in dengue is rare, but case reports of pericarditis have been reported. Pericardial effusions are extremely uncommon but have been documented in severe dengue. The 2009 World Health Organization revised classification included myocarditis as a form of severe dengue, but very few patients have a formal cardiac assessment, especially in endemic areas. Consequently, the true incidence of dengue myocarditis remains uncertain. Dengue myocarditis can mimic acute myocardial infarction as evidenced by ECG changes and the elevation of cardiac biomarkers. Myocyte damage is likely related to the cardiotropic nature of the virus, with serotypes DEN 1, DEN 2, and DEN 3 being frequently implicated in patients with fulminant myocarditis. Echocardiography could help guide early management of refractory shock with optimal fluid therapy and appropriate inotropes. Cardiac MRI is a useful research tool that helps confirmation of myocardial involvement seen as hyperintense signals on T2-weighted images with early and late gadolinium enhancement.
There is no specific treatment for severe dengue, and care is entirely supportive, including judicious fluid resuscitation. Fluid overload has been found to be associated with increased morbidity during the recovery phase of dengue myocarditis. Approximately 4% of patients with shock require inotropes despite fluid resuscitation. Inotropes or vasopressors are used to treat life-threatening hypotension or cardiogenic shock. There is no evidence to support the use of specific antiviral therapy, steroids, or immunoglobulin in dengue myocarditis. Early diagnosis of myocardial involvement, fluid resuscitation while avoiding overload, and inotropic support with continuous monitoring remain the cornerstones of management in dengue-affected patients with severe myocarditis.

Disclosures
None.

References

Figure 1. Twelve-lead ECG showing hyperacute ST-T changes in leads II, III, aVF, v5, and v6.
Figure 2. Transthoracic echocardiogram taken in parasternal long-axis view (A) and M mode across base of left ventricle (LV) in short-axis view (B) showing dilated LV cavity, severely reduced LV systolic dysfunction, and preserved myocardial thickness.

Figure 3. Midcavity short-axis late gadolinium image of the heart obtained 5 minutes after administration of intravenous contrast. There is rapid washout of contrast from the blood pool of the left ventricular (LV) cavity (*) and diffuse transmural enhancement of the LV myocardium (arrows). This is in keeping with an increase in the extracellular space in the LV myocardium secondary to extensive myocarditis resulting in rapid uptake of gadolinium contrast. There is also a moderate pericardial effusion (∆).

Figure 4. Still image from midcavity short-axis cine gradient echocardiographic images of the heart that shows borderline thickening of the left ventricular (LV) myocardium (arrow). The apparent irregularity of the visceral pericardium overlying the right ventricular (RV) anterior free wall (arrowheads) corresponds to the chemical shift artifact given the interface between pericardial fluid (∆) and epicardial fat.
Figure 5. Transthoracic echocardiographic images. A, M mode across base of left ventricle (LV) taken in short axis view showing improved LV function. B, Normal mitral filling Doppler signals for age.
Dengue Myopericarditis Mimicking Acute Myocardial Infarction
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