A transient bradycardic presyncope in a man with dengue fever: an unusual manifestation of dengue myocarditis

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SUMMARY

Myocarditis is an inflammatory condition of the myocardium leading to various degrees of myocardial dysfunction. It has a wide range of clinical manifestations, from subtle asymptomatic tachyarrhythmia or bradyarrhythmia to life-threatening heart failure with cardiogenic shock. The aetiologies of myocarditis include infections, connective tissue or autoimmune disorders, and idiopathic, of which viral infections such as dengue fever and influenza are the leading causes of myocarditis. Due to the lack of specific clinical manifestations of myocarditis, high clinical suspicion with the aid of laboratory investigations such as cardiac enzymes and imaging with echocardiogram are crucial to ensure a timely diagnosis and appropriate management. Nevertheless, the management of myocarditis is usually supportive. We report a case of dengue fever complicated with viral myocarditis and cardiogenic shock, which recovered well with supportive management.

INTRODUCTION

Dengue fever is a vector-borne acute viral infection, spread primarily by the *Aedes aegypti* and *A. albopictus* mosquitoes. The global incidence of dengue infections is rising, affecting mainly the tropical and subtropical regions, including countries like Malaysia. The geographical variation of dengue incidence can be explained by the meteorological factors such as high rainfall and temperature and low wind speed to encourage the growth of vectors in tropical and subtropical regions.[1] Thus, the main approach to reducing the incidence of dengue infections is to reduce the population and growth of vectors.

Dengue fever has a broad spectrum of clinical manifestations, ranging from a mild febrile illness to severe dengue shock syndrome with failure of multiple organs. Mild dengue fever can be treated as an outpatient with supportive management. However, severe dengue infections require close monitoring in the intensive care unit (ICU) and have high mortality rates. Organs affected in severe dengue infections include kidneys, liver and heart. Dengue myocarditis is not uncommon and similarly has a wide spectrum of clinical manifestations, ranging from a subtle asymptomatic cardiac arrhythmia to life-threatening heart failure with cardiogenic shock.[2]

CASE PRESENTATION

A 20-year-old man presented with a day of fever and lethargy associated with four episodes of vomiting and two episodes of loose stools. The vomitus contained food particles and clear fluids without blood or bilious content, and the stools were brownish without blood or mucus. Otherwise, he had no abdominal pain and no history of travelling and sick contact. He had no known medical illness and no previous hospitalisation.

He was orientated with full Glasgow Coma Scale upon presentation. His physical examination revealed a febrile man in systemic shock with a body temperature, blood pressure and heart rate of 38.9°C, 89/40 mmHg and 119 beats per minute, respectively. His systemic examinations were otherwise unremarkable with good peripheral perfusion. His initial blood investigations confirmed the diagnosis of a severe dengue fever with a positive dengue virus non-structural protein 1, haemoconcentration with an elevated haematocrit of 50.2% and acute kidney injury with a serum creatinine level of 129 µmol/L, and estimated glomerular filtration rate of 70.2 ml/min/1.73m^2. Other laboratory investigations particularly the venous blood gas, serum lactate, white cells and platelets counts and liver function test were normal.

He was given intravenous fluid resuscitation with boluses of normal saline at a rate of 20 ml/kg/h up to a cumulative 5 L fluid; however, he remained hypotensive and required vasopressor with intravenous infusion of noradrenaline at a rate of 0.1 µg/kg/minute to maintain a blood pressure of 142/80 mmHg. However, he developed acute pulmonary oedema after the resuscitation, with a respiratory rate of 38 breaths per minute and an oxygen saturation of 64% under room air. His physical examination revealed end-inspiratory fine crackles at bilateral lower zones of lungs, with pulmonary oedema and upper lobe diversion in his chest radiograph (Figure 1). He was immediately given intravenous frusemide of 40 mg and oxygen supplementation with a non-rebreathing mask and was admitted to ICU.

He was given supportive management and non-invasive ventilatory support with continuous positive airway pressure in the ICU. His clinical conditions improved after a day in the ICU with a stable haemodynamic and oxygenation without vasopressor and ventilatory support, and he became afebrile.

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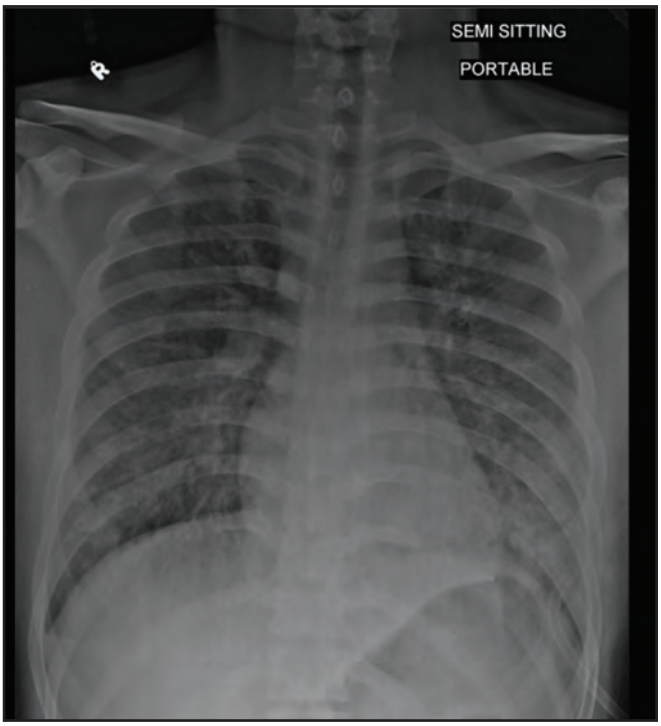

Fig. 1: Chest radiograph showed pulmonary oedema and upper lobe diversion.

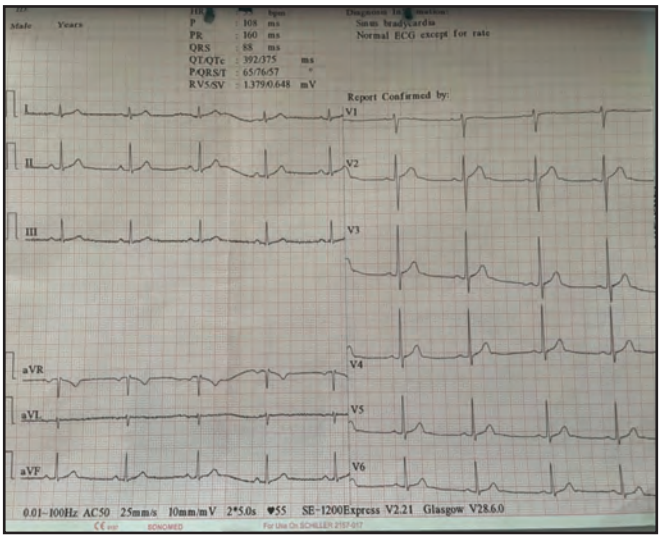

Fig. 2: Electrocardiogram showed sinus bradycardia.

However, he developed a transient episode of presyncope with dizziness and diaphoresis the following day, which coincided with hypotension and bradycardia with a blood pressure of 67/47 mmHg and a heart rate of 50 beats per minute. The incident happened when he was lying on the bed, with a sudden onset of dizziness and diaphoresis, which lasted for 10 minutes. His blood pressure and heart rate prior to the event were 140/80 mmHg and 88 beats per minute, respectively. His electrocardiogram during the event showed sinus bradycardia (Figure 2). His repeated blood investigations revealed raised cardiac enzymes with troponin I of 86 ng/ml, aspartate transaminase of 55 U/L, and lactate dehydrogenase of 523 U/L. An echocardiogram after the event showed a mild left ventricular systolic dysfunction with an ejection fraction of 45%, hypokinesia at basal to mid-inferoseptal of left ventricle, and a dilated right ventricle. He was discharged well after a seven-day hospital stay with a normal follow-up echocardiogram after a month.

DISCUSSION

The clinical manifestations of myocarditis are non-specific with great heterogeneity, thus requiring individualised diagnostic approach and management. Recent onset heart failure, cardiac arrhythmias, and chest pain are three main patterns of presentation of myocarditis, which may represent any structural heart and coronary artery diseases. The findings of most investigations, such as cardiac enzymes, electrocardiogram, and echocardiography, are not specific to differentiate myocarditis from other heart diseases. A cardiac magnetic resonance imaging offers a good accuracy in diagnosing myocarditis while endomyocardial biopsy with histopathologic analysis is the only way to confirm a diagnosis of myocarditis. Nevertheless, endomyocardial biopsy is an invasive procedure, thus only recommended to be done in severe cases with severe left ventricular dysfunction and life-threatening arrhythmias.[3] Therefore, any unexplainable haemodynamic changes should raise the suspicion of myocarditis.

The exact pathophysiology of myocardial injury in dengue fever is not fully understood yet, and it is postulated to be a result of direct dengue virus invasion or cytokine-mediated immunological response.[2] Cardiac involvement in dengue fever is usually underdiagnosed as any haemodynamic instabilities such as hypotension, tachycardia, and pulmonary oedema are commonly presumed to be attributed to dehydration and capillary leakage in dengue fever.[4] Our current case demonstrated a misdiagnosed dengue myocarditis during presentation with cardiogenic shock unresponsive to fluid resuscitation, only able to raise the suspicion of myocarditis after a transient bradycardic presyncope. Early recognition of myocardial involvement in this case could have prevented overzealous fluid resuscitation, leading to pulmonary oedema.

Bradyarrhythmia, mainly sinus bradycardia, is the most common cardiac conduction abnormality seen in dengue infections. It is usually benign and self-limiting, as demonstrated in our case. Other cardiac conduction disturbances that might require intervention with anti-arrhythmic medications and cardiac pacing include various degrees of atrioventricular block, supraventricular and ventricular tachyarrhythmias, and non-specific electrocardiographic changes. The mainstay therapeutic approach in a suspected or confirmed case of myocarditis is supportive management to ensure haemodynamic stability and optimal fluid status.[2-4] Steroidal therapy has no strong evidence in myocarditis and is only recommended in severe cases for certain types of myocarditis confirmed by endomyocardial biopsy.[3]

CONCLUSION

Myocarditis has no specific clinical manifestation. The diagnosis of myocarditis greatly depends on the clinical acumen of the attending doctor. Although the treatment of myocarditis is mainly supportive, early recognition of myocarditis is crucial to ensure an appropriate fluid regime and vasopressor support to restore the haemodynamic stability and to avoid complications such as pulmonary oedema.

DECLARATION

There is no competing interests by the authors with the manuscript. There is no funding. A written consent was taken from the patient for publication.

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