

Dengue perimyocarditis: a case report

登革熱心包心肌炎：個案報告

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Dengue is the most important mosquito borne viral disease in the world. It is endemic in South and Southeast Asia, Central and Latin America, and Africa. One of its manifestations is dengue shock syndrome (DSS), a medical emergency with a very high mortality. Cardiac involvement is a known but infrequent feature of dengue. Cardiac dysfunction may complicate management of hypotension in DSS. We report a case of a 16-year-old male presenting with fulminant DSS as well as perimyocarditis that both confounded the clinical picture and complicated the clinical management. (*Hong Kong j.emerg.med.* 2010;17:58-60)

登革熱是全球最重要的經蚊子傳染病毒疾病。是南亞、東南亞、中美洲、拉丁美洲及非洲的地方病。其中的一個徵象是登革熱休克綜合徵，為很高死亡率的醫學急症。牽涉心臟為登革熱已知但不尋常的徵狀；心臟機能障礙可令處理登革熱休克綜合徵的低血壓複雜化。本文匯報一名16歲男子的個案，呈現暴發性的登革熱休克綜合徵及心包心肌炎，混淆臨床徵狀及令臨床處理複雜化。

Keywords: Dengue haemorrhagic fever, myocarditis, pericarditis

關鍵詞：登革出血熱、心肌炎、心包炎

Case report

A 16-year-old student with no prior medical or cardiac history was referred by his primary care physician to the emergency department of a Singapore hospital in September 2006 at about 13:00 hour for epigastric pain and shortness of breath.

He had been having fever for six days, which had not responded to courses of oral amoxicillin followed by amoxicillin/clavulanate. This was associated with nausea and vomiting. There was no travel history. He subsequently developed epigastric pain and shortness of breath one day prior to his attendance to hospital.

On examination, he was slightly lethargic and breathless. His mucous membranes looked dry and his skin had a flushed appearance. His heart rate was 138 beats per minute and his blood pressure was 100/72 mmHg. He had a respiratory rate of 20 breaths per minute but there was persistent difficulty in obtaining his oxygen saturation. His heart sounds were dual and not muffled, and there were no murmurs or rubs. His breath sounds were clear, with equal air entry bilaterally. There was mild epigastric tenderness but his abdomen was otherwise soft, with no guarding or rebound tenderness. Bowel sounds were present. His throat was injected but there were no pustules or exudates. There was no petechial rash or bruising noted.

Intravenous access was obtained and blood samples were taken and sent for full blood count, urea/electrolyte panel, cardiac enzymes, liver function test, dengue serology, coagulation profile, and group and cross match. Intravenous normal saline drip was started, and the patient was given intravenous (IV) metoclopramide 10 mg and IV hyoscine 20 mg.

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Electrocardiogram (ECG) showed widespread ST segment elevations. Bedside ultrasonography (US) showed the presence of pericardial effusion. A portable chest radiograph did not reveal cardiomegaly, pulmonary congestion, or air under the diaphragm.

In view of the history of fever and shortness of breath, with the above ECG and US findings, the provisional diagnosis then was acute pericarditis with pericardial effusion.

The duty cardiologist consulted was agreeable for the patient to be admitted to the cardiology intensive care unit (ICU). The patient was transferred to the ICU at about 15:30 hour, where his skin was noted to be flushed and mottled. His blood pressure dropped to 70-80/50 mmHg while his heart rate was 130-140/min.

His laboratory investigations were as follows: Hb 18.4 g/dL, Hct 54.6%, WBC 7.8×10^3 /uL with atypical lymphocytes 4%, platelet 42,000/uL, creatinine 156 umol/L, urea 8.7 mmol/L, sodium 130 mmol/L, potassium 5.6 mmol/L. His dengue IgM was positive, with IgG negative. The CK-MB was raised at 23 ng/mL, and troponin T was 1.72 ng/mL. His ESR was 3 mm/hr. The impression was that of dengue shock syndrome complicated by pre-renal azotemia and perimyocarditis.

Intra-arterial and central venous pressure lines were inserted. He was resuscitated with 2 litres of IV Gelafundin and 1 litre of IV normal saline over 2 hours, as well as a maintenance IV drip of 2.5 litres of normal and dextrose saline over 24 hours, and started on IV dopamine and IV noradrenaline infusions.

An urgent 2-D echocardiogram revealed generalised segmental wall motion abnormalities, severely impaired systolic function with ejection fraction (EF) of 25-30%, a small pericardial effusion with no evidence of tamponade, no mitral or aortic regurgitation.

Urgent referral was made to the infectious disease physician who suggested supportive management, as well as to the surgeon (in view of the abdominal pain) who suggested intravenous antibiotics for possible acalculous cholecystitis.

At 18:00 hour, the patient was noted to be increasingly tachypnoeic with a respiratory rate of 50/min. Arterial blood gas showed pH 7.32, standard bicarbonate 15.2 mmol/L, pO_2 378.4 mmHg, and pCO_2 23.8 mmHg.

While being prepared for intubation, the patient deteriorated and his blood pressure became unrecordable. Cardiopulmonary resuscitation was commenced and he was endotracheally intubated. His dopamine and noradrenaline infusions were increased to the maximal doses, and IV dobutamine, vasopressin and bicarbonate infusions were also started. He was given a total of 38 doses of IV adrenaline 1:10,000, 2.4 mg IV atropine, and 250 ml IV 8.4% sodium bicarbonate. He was resuscitated for 75 minutes, during which he had 5 episodes of unsustained return of spontaneous circulation, before efforts were terminated. The cause of death was deemed to be dengue haemorrhagic shock syndrome.

Discussion

The prevalence of dengue worldwide has increased dramatically in recent decades. In the past 50 years, the incidence of dengue has increased 30-folds. The World Health Organization (WHO) estimates that about 2500 million people (or 40% of the world's population), mainly living in tropical and sub-tropical urban regions, are at risk from dengue. A rapid rise in urban populations is bringing ever greater numbers of people into contact with the mosquito vector that transmits the dengue virus, *Aedes aegypti*.¹ It is estimated that there are 50-100 million cases of dengue infection annually, with an estimated 500,000 cases of dengue haemorrhagic fever requiring hospitalisation each year, of whom a very large proportion are children. At least 2.5% of the cases die, although case fatality could be twice as high.²

Dengue infections may be asymptomatic, or manifest as a non-specific febrile illness, dengue fever (DF), dengue haemorrhagic fever (DHF), or dengue shock syndrome (DSS). According to the WHO guidelines, a case of DHF should meet the following 4 criteria: acute onset of high fever, haemorrhagic manifestation, thrombocytopenia $<100,000/\mu\text{L}$, and evidence of

increased capillary permeability or plasma leakage. DSS, defined as DHF plus signs of circulatory failure, is associated with very high mortality.³

Cardiac manifestations of dengue are uncommon.⁴ Wali et al, in a study of 17 patients with DHF/DSS, noted that acute reversible cardiac insult may be noticed in DHF/DSS and could be responsible for hypotension/shock seen in some of these patients.⁵ Méndez et al, in a study of 913 children with DHF, mentioned cardiac involvement in 8%, predominantly in male school-age children with DSS.⁶ Kamath and Ranjit, in a study of 109 children with severe DHF requiring ICU admission, described 37.5% with persistent shock, but only 4.6% with echocardiographic myocardial dysfunction, and 2.7% with pericardial effusion.⁷

In patients with DSS, transient myocardial depression is not uncommon. In a study of 91 children with DHF, Khongphatthanayothin et al found the EF was less than 50% in 13.8% of patients with DHF but 36% in patients with DSS.⁸ Interestingly, none of the children in this series had elevated troponin T levels.

Dengue myocarditis/pericarditis is exceedingly rare, with only a handful of cases reported in world literature. Nagaratnam et al described a series of 3 cases with myocarditis and pericarditis caused by the dengue virus.⁹ Wiwanitkit reported that although there were thousands of DHF cases in Thailand, there had been only two reports of dengue myocarditis, one of which was a 13-year-old boy who developed bradycardia and hypotension while recovering from DHF.¹⁰ Amongst four reports covering 51 fatalities from a total of 6154 DHF cases in Thailand, there were no cardiac manifestations documented during autopsy.¹⁰ Indeed, there were no fatalities amongst isolated case reports.

Our patient presented in circulatory shock which, in view of the ECG and US findings, was initially attributed to pericarditis with pericardial effusion. Only when the thrombocytopenia, haemoconcentration and serology results were noted was DSS appreciated. His cardiac function was markedly deranged with low EF, and

this might have contributed to the severity of his shock state and non-response to fluid resuscitation and inotropic support. In contrast to the findings of Khongphatthanayothin and Wiwanitkit,^{8,10} our patient had increased levels of both CK-MB and troponin T, and his clinical course was fulminant and ultimately fatal.

In conclusion, our patient presented with features suggestive of DSS as well as significant cardiac dysfunction that both confounded the clinical picture and complicated the clinical management of DSS. Though uncommon, clinicians caring for patients with DSS should be watchful for potentially severe cardiac manifestations such as perimyocarditis.

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