

# CASE REPORT

## DENGUE MYOCARDITIS

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**Abstract.** We report a 13-year-old boy who developed bradycardia and hypotension a day after recovery from dengue hemorrhagic fever. His electrocardiogram, during the bradycardia, showed a junctional rhythm with a rate of 50 beats/minute. This is the first reported case of sinus node dysfunction following dengue infection.

A previously healthy 13-year-old Thai boy was referred from a private hospital because of bradycardia. He was admitted to Nakhon-Christian Hospital with history of high fever for 12 hours without a local source of infection. Four days after admission, he started developing hypotension and plasma leakage, evidenced by bilateral pleural effusions and ascites, with an enlarged and tender liver. He recovered from shock on day 5, after appropriate fluid management. There was no bleeding diathesis and the highest hematocrit was 43%.

A complete blood count (CBC) on the day of admission revealed the following: white blood cell count 5,100/mm<sup>3</sup> (with 88% neutrophils, 10% lymphocytes, and 2% monocytes); hemoglobin 12.3 g/dl; hematocrit 37%; platelets 12,000/mm<sup>3</sup>. Serum sodium was 137 mmol/l, potassium 3.35 mmol/l, chloride 106 mmol/l, and bicarbonate 19.6 mmo/l. His urinalysis result was normal.

On the 6<sup>th</sup> day of illness, he developed hypotension and bradycardia. His slowest heart rate was 50 beats/minute in a junctional escape rhythm seen on 12-lead electrocardiography (ECG). He was transferred to the intensive care unit for close monitoring. At the beginning, adrenaline was infused at a rate of 0.2 µg/kg/minute. His heart rate increased to 80 beats/minute in a junctional rhythm (Fig 1) and became hemodynamically stable. Then he developed frequent episodes of premature ventricular contractions (PVCs), there-

fore lidocaine 1 mg/kg was intravenous administered, followed by a continuous infusion at 16 µg/kg/minute.

He was referred to Songklanagarind Hospital on day 7 of illness. He had cold clammy skin, fair tissue perfusion, slightly puffy eyelids, and mild hepatomegaly. Cardiac auscultation revealed an S<sub>3</sub> gallop without a significant murmur. An electrocardiogram demonstrated sinus tachycardia, non specific ST-T change, and no PVCs (Fig 2). A CBC demonstrated a hematocrit of 32%, white blood cells 8,100/mm<sup>3</sup> (neutrophils 71%, lymphocytes 18%), and platelets 39,000/mm<sup>3</sup>. The following investigations gave normal or negative results: serum urea 6.4 mmol/l, serum creatinine 0.7 mmol/l, serum sodium 133 mmol/l, serum potassium 3.9 mmol/l, serum chloride 102 mmol/l, serum bicarbonate 21 mmol/l, and blood and urine cultures were negative. A two-dimensional echocardiogram showed normal cardiac structures, no valvular regurgitation, but impaired left ventricular (LV) systolic function (ejection fraction 40%). Chest x-ray showed cardiomegaly and bilateral pleural effusions. The sedimentation rate was 30 mm/hour, total creatine phosphokinase (CK) 486 U/l, CK-MB 18.92 U/l (normal < 10 U/l), CK-MB/CK was 3.89, and troponin T was 0.12 ng/ml. (level >0.1 ng/ml is considered elevated) (Lauer *et al*, 1997) The initial dengue hemagglutination titer was >1:10,240; with dengue IgM 100 IU/ml (normal <40 IU/ml). Based on World Health Organization laboratory criteria for the diagnosis of dengue hemorrhagic fever (DHF), the patient had secondary dengue infection or DHF (WHO, 1986).

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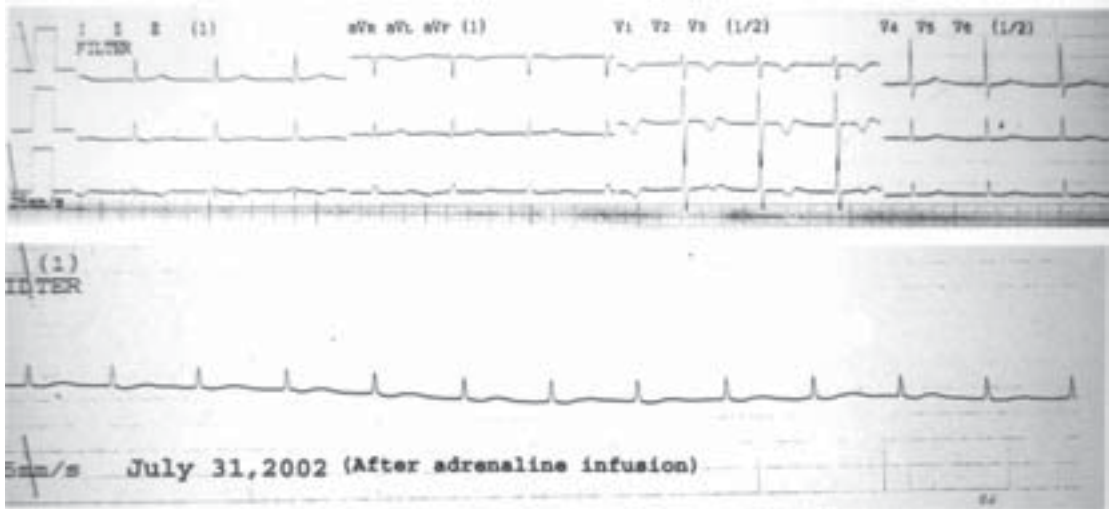


Fig 1–The ECG showed junctional rhythm on day 6 of illness (after adrenaline infusion).



Fig 2–The ECG on day 7 of illness showed sinus tachycardia.

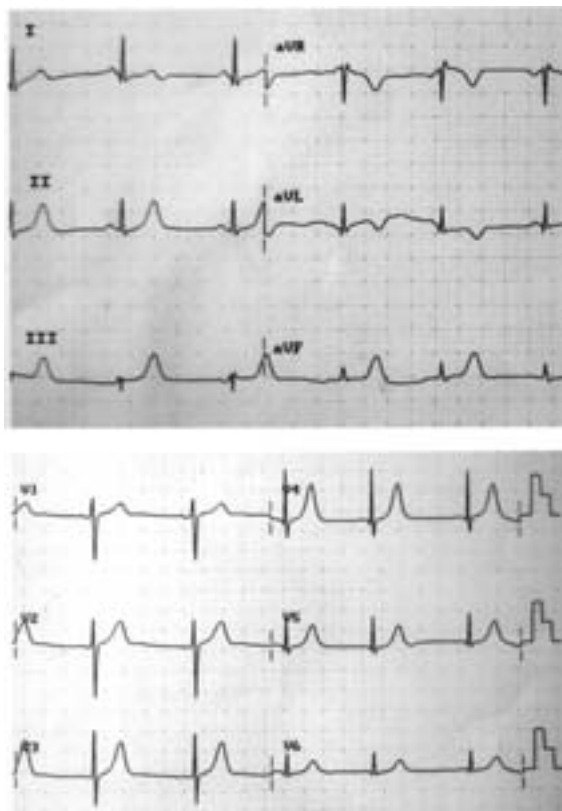


Fig 3—The ECG returned to normal sinus rhythm after recovery of dengue infection.

He was transferred to the pediatric intensive care unit for close monitoring. Neither arrhythmias nor hypotension occurred, and the adrenaline and lidocaine were successfully discontinued on the next day. A follow-up echocardiographic study on day 14 of the illness showed LV ejection fraction of 68%. Prednisolone 2 mg/kg/day was started on day 16 of the illness. He was discharged after 7 days of admission with digoxin, furosemide, and prednisolone 2 mg/kg/day. He was at a New York Heart Association (NYHA) functional class I two weeks after hospital discharge. A dengue hemagglutination titer and cardiac troponin T level were subsequently obtained with the results of 1: 5,120 and <0.01 ng/ml, respectively. His ECG at the time of follow-up showed normal sinus rhythm (Fig 3). The prednisolone was eventually tapered off 10 days after his first dose.

Myocardial dysfunction can be seen in patients with DHF. Approximately 20% of those who developed DHF have a LV ejection fraction of less

than 50%, and are likely to return to normal within a few weeks (Wali *et al*, 1998). The pathogenic mechanisms of cardiac dysfunction are not well established; alternation of autonomic tone and prolonged hypotension may play a role. Myocarditis in patients with DHF is still a matter of debate.

Our patient developed dysfunction of the sinus node and impairment of ventricular systolic performance a day after recovered from the dengue shock stage, which is an unusual pattern of cardiac involvement, for those with dengue infection. It would seem more likely that there was some degree of myocardial damage causing impairment of the LV systolic function and elevation of the serum troponin T level. Electrocardiographic abnormalities have been reported in 44-75% of patients with viral hemorrhagic fever, and prolongation of the PR interval or sinus bradycardia commonly occurs (Smyth and Powell, 1954; Boon, 1967). Some have reported atrioventricular block in variable degrees (Lim *et al*, 1970; Kongpattanyothin *et al*, 2000). The sino-atrial (SA) node dysfunction and impairment of systolic function in this patient transiently occurred during the recovery period. This may represent a transient functional impairment which may result from abnormalities in autonomic tone, or localized pathology, such as minute bleeding in the area of the SA node.

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