# Spontaneous resolution of sinoatrial exit block and atrioventricular dissociation in a child with dengue fever

**Kaushik J S, Gupta P, Rajpal S, Bhatt S**


## ABSTRACT

**Cardiac rhythm abnormalities, including ventricular arrhythmia, atrial fibrillation and atrioventricular block, have been observed during the acute stage of dengue haemorrhagic fever. Atrioventricular or complete heart block can be fatal and may require a temporary pacemaker. We report a ten-year-old girl who presented with dengue haemorrhagic fever with sinoatrial block and atrioventricular dissociation that had a spontaneous resolution.**


**Keywords: atrioventricular block, atrioventricular dissociation, dengue haemorrhagic fever, rhythm abnormality, sinoatrial block**

*Singapore Med J 2010; 51(8): e146-e148*

## INTRODUCTION

Dengue fever is a viral infection that is transmitted by the mosquito, *Aedes aegypti* and characterised by fever, myalgia, arthralgia, rash, leucopaenia and thrombocytopaenia.[1] Cardiac rhythm abnormalities, including sinus bradycardia, ST-T changes, prolonged PR interval, bundle branch block and complete heart block, have been uncommonly observed in dengue haemorrhagic fever.[2] However, sinoatrial exit block has not been previously reported in children with dengue fever. We report dengue haemorrhagic fever in a child, with sinoatrial (SA) block and atrioventricular (AV) dissociation with a structurally normal heart.

## CASE REPORT

A ten-year-old girl presented to our centre during a dengue fever outbreak in Delhi, with complaints of fever, abdominal pain and vomiting of one week's duration. There was no history of myalgia and bone or joint pains, nor was there a history of rash or bleeding from any site. There was no past history of arrhythmia or cardiac disease. The urine output was adequate. On examination, the patient was lethargic and febrile, but not dehydrated. The vital signs were stable at admission; her heart rate was regular at 92/min, respiratory rate at 22/min and blood pressure (BP) at 110/74 mmHg. There was a petechial rash over the patient's back, trunk and upper extremities. The Hess test (tourniquet test) for capillary fragility was positive. Systemic examination did not reveal any abnormality.

Investigations revealed normal haemoglobin (12.1 g/dl), haematocrit (35.2%) and leucocyte (6500/cumm) counts, while the platelet count was observed to be low (38000/cumm). Dengue serology was reactive for immunoglobulin G (IgG) and immunoglobulin M (IgM), suggesting acute primary infection. The liver enzymes and renal profile, including serum electrolytes, were normal. The patient was managed supportively according to the World Health Organization (WHO) recommendations for management of dengue haemorrhagic fever.[3]

The following day, the patient was observed to have bradycardia (pulse rate 50/min, regular), with normal BP. The heart sounds were normal, and there were no new cardiovascular findings. Electrocardiography (ECG) revealed a rate of 44/min and varying PP intervals (an absence of normally expected P waves), which was suggestive of SA block, with no relation (dissociation) between P waves (atrial activity) and QRS complex (ventricular activity) indicative of AV dissociation. Chest radiography was normal, with a regular heart size. Echocardiography revealed normal cardiac valves, left ventricular systolic and diastolic functions, and cardiac chamber dimension. There was no evidence of pericardial effusion or vegetations. In the presence of rhythm disturbances, the patient was further investigated for underlying myocarditis. The creatinine phosphokinase (CPK) and lactate dehydrogenase (LDH) levels were 172 U/dL (normal ≤ 130 U/dL) and 776 U/dL (normal ≤ 330 U/dL), respectively. The serum potassium and calcium levels were found to be normal.

The patient's heart rate during the hospital stay remained at 46–54/min, but the BP and other haemodynamic parameters were normal. The patient was asymptomatic, and her vital signs were closely monitored for the development of any complications.

**Department of Paediatrics, University College of Medical Sciences, Dilshad Garden, Delhi 110095, India**

Kaushik JS, MD
Registrar

Gupta P, MD, FIAP
Professor

Bhatt S, MD
Registrar

**Department of Medicine, University College of Medical Science, Guru Teg Bahadur Hospital, Dilshad Garden, Delhi 110095, India**

Rajpal S, MD
Specialist

**Correspondence to:**
Dr Jaya Shankar Kaushik
Tel: (91) 98711 74069
Fax: (91) 11 2259 0495
Email:jayashankarkaushik@gmail.com

She was discharged after eight days, with a heart rate of 52/min and a normal BP, and the ECG showed a persisting complete AV block. The patient was kept on close follow-up for one week, during which her bradycardia and complete AV block persisted. A follow-up after five months showed a normal heart rate (82/min). ECG reverted to a normal sinus rhythm, and the echocardiography was also normal.

## DISCUSSION

Dengue fever is a major cause of morbidity and mortality in the South East Asia region, including in India.[4] Massive bleeding and shock are the most common causes of death. Cardiac manifestations of dengue fever may play a role in the pathogenesis of shock and could influence the outcome of the disease. However, the cardiac manifestations in dengue are invariably benign, transient and self-limited, and are attributed to subclinical viral myocarditis.[5] Approximately 90% of patients with viral myocarditis recover completely, and only few patients develop long term sequelae. During the latent period, the virus can trigger an autoimmune reaction that may progressively destroy myocardial tissue, leading to dilated cardiomyopathy.

Cardiac rhythm disturbances, such as bradycardia and ventricular ectopics, are known to occur during the convalescence period.[1] ECG changes in dengue haemorrhagic fever include sinus bradycardia, ST segment elevation and non-specific ST-T changes.[5,6] Most of these changes are transient and revert to normal at follow-up. SA, AV and bundle branch blocks, AV dissociation, complete heart block and premature ventricular contraction have been described in a series of 218 cases of biopsy-proven viral myocarditis.[7] Rhythm abnormalities reported in dengue fever include complete AV block,[6] first-degree AV block,[8] atrial fibrillation,[9] Mobitz type I second-degree AV block,[10] ventricular arrhythmia[11] and sinus node dysfunction.[12] Abnormal right and left ventricular functions, left ventricular hypokinesia, mitral regurgitation and pericardial effusion have also been documented in dengue fever using echocardiography.[5,13]

An SA exit block refers to a block of impulses from the SA node to the atria. The block can be physiological if the initiating impulse is premature and arrives at the cardiac site during the normal refractoriness, or it can be pathological in cases of abnormally long refractory periods or abnormally slow conduction velocity.[14] An SA node exit block is characterised by progressive lengthening of the PR interval, varying PP interval or absent P wave on ECG. To the best of our knowledge, SA exit block has not been reported in cases of dengue fever to date.

The presence of bradycardia and elevated cardiac enzyme levels (LDH and CPK) was consistent with a diagnosis of myocarditis,[15] although there were no signs of cardiogenic shock or congestive heart failure. The co-existence of SA block with AV dissociation on ECG could possibly explain the rhythm abnormalities associated with myocarditis in our patient. The presence of fever, a positive tourniquet test, thrombocytopaenia, and positive IgG and IgM titres for dengue virus prompted us to suspect dengue virus as the probable causative agent. AV conduction abnormalities secondary to myocarditis are also observed in infections with *Diphtheriae*, *Yersinia*, *Borrelia* (Lyme disease) and *Trypanosoma* (Chagas disease).[16]

Arboviral infection can cause myocardial damage, either by direct invasion or an autoimmune reaction, which results in myocarditis.[17] Myocarditis can manifest with complete AV block or ventricular arrhythmias, which might manifest as syncope or palpitations.[17] However, our patient did not develop any cardiac symptoms. Dengue virus-induced myocarditis could apparently explain the SA block and AV dissociation observed in our case.

Approximately 40% patients recover from the rhythm abnormality in myocarditis.[17] It is postulated that patients with fewer symptoms have a better prognosis and that complete resolution can be expected, as observed in our case.[15] Acute high grade AV blocks occasionally respond to atropine and isoproterenol.[16] These drugs were not used in our patient due to the dangerous slow escape rhythms associated with them. In view of the inadequate escape rhythms and persistent bradycardia, temporary cardiac pacing was advised for our patient. However, owing to cost constraints, the patient's parents did not opt for pacemaker implantation and preferred conservative medical management instead. This is acknowledged as a known limitation of patient care in developing nations. The patient was kept under close follow-up, and spontaneous resolution was observed after five months. The rhythm abnormalities in dengue fever tend to be benign and self-limited, and resolve in the majority of patients at discharge or on follow-up. Patients with advanced AV blocks and who are symptomatic rarely require a pacemaker implantation.

## REFERENCES

1. Halstead SB. Dengue fever and dengue hemorrhagic fever. In: Kliegman RM, Behrman RE, Jenson HB, Stanton BF, eds.

Nelson Textbook of Pediatrics. 18th ed. Philadelphia: Saunders Elsevier, 2007: 1412-5.
2. Gulati S, Maheshwari A. Atypical manifestations of dengue. Trop Med Int Health 2007; 12:1087-95.
3. World Health Organization (WHO). Guidelines for management of dengue fever/dengue haemorrhagic fever in small hospitals. In: WHO regional office for south-east Asia [online]. Available at: www.searo.who.int/LinkFiles/Dengue_Guideline-dengue.pdf. Accessed 29 June, 2009.
4. Ministry of Health and Family Welfare (India). Status note on dengue fever and dengue hemorrhagic fever. In: National vector borne disease control programme [online]. Available at: www.nvbdcp.gov.in/Doc/DenStatusNote.pdf. Accessed June 9, 2009.
5. Yusoff K, Roslawati J, Sinniah M, Khalid B. Electrocardiographic and echocardiographic changes during the acute phase of dengue infection in adults. J HK Coll Cardiol 1993; 1:93-6.
6. Kularatne SA, Pathirage MM, Kumarasiri PV, Gunasena S, Mahindawanse SI. Cardiac complications of a dengue fever outbreak in Sri Lanka, 2005. Trans R Soc Trop Med Hyg 2007; 101:804-8.
7. Kawamura K, Kitaura Y, Morita H, Deguchi H, Kotaka M. Viral and idiopathic myocarditis in Japan: a questionnaire survey. Heart Vessels Suppl 1985; 1:18-22.
8. Naresh G, Kulkarni AV, Sinha N, Jhamb N, Gulati S. Dengue hemorrhagic fever complicated with encephalopathy and myocarditis: a case report. J Commun Dis 2008; 40:223-4.
9. Horta Veloso H, Ferreira Júnior JA, Braga de Paiva JM, et al. Acute atrial fibrillation during dengue hemorrhagic fever. Braz J Infect Dis 2003; 7:418-22.
10. Khongphatthallayothin A, Chotivitayatarakorn P, Somchit S, et al. Morbitz type I second degree AV block during recovery from dengue hemorrhagic fever. Southeast Asian J Trop Med Public Health 2000; 31:642-5.
11. Chuah SK. Transient ventricular arrhythmia as a cardiac manifestation in dengue haemorrhagic fever – a case report. Singapore Med J 1987; 28:569-72.
12. Promphan W, Sopontammarak S, Pruekprasert P, Kajornwattanakul W, Kongpattanayothin A. Dengue myocarditis. Southeast Asian J Trop Med Public Health 2004; 35:611-3.
13. Bhatia V, Parida AK, Arora P, et al. Electrocardiographic and echocardiographic findings during the recent outbreak of viral fever in National Capital Region. Indian Heart J 2007; 59:360-2.
14. Walsh WP, Berul CI, Triedman JK. Cardiac arrhythmias. In: Keane JF, Lock JE, Fyler DC, eds. Nadas' Pediatric Cardiology. 2nd ed. Philadelphia: WB Saunders, 2006, 477-524.
15. Bernstein D. Diseases of the myocardium. In: Kliegman RM, Behrman RE, Jenson HB, Stanton BF, eds. Nelson Textbook of Pediatrics. 18th ed. Philadelphia: Saunders Elsevier, 2007: 1963-72.
16. Kannankeril PJ, Fish FA. Disorders of cardiac rhythm and conduction. In: Allen HD, Driscoll DJ, Shaddy RE, Feltes TF, eds. Moss and Adams' Heart Diseases in Infants, Children, and Adolescents Including Fetus and Young Adults. 7th ed. Philadelphia: Wolter-Kluwer/Lippincott Williams & Wilkins, 2008: 293-341.
17. Pinney SP, Manchi DM. Myocarditis and specific cardiomyopathy. In: Fuster V, Walsh RA, O'Rourke RA, Poole-Wilson P, eds. Hurst's the Heart. 12th ed. New York: McGraw Hill, 2008: 863-84.