Trends in Medicine

Case Report ISSN: 1594-2848

Acute dengue fever complicated by transient sinus arrest requiring temporary cardiac pacing

Gnanenthiran SR[1,2*], Findlay M[3], Gray T[3] and Elsik M[4]

[1]Department of Cardiology, Concord Repatriation General Hospital, Sydney, NSW, Australia

[2]The George Institute for Global Health; University of New South Wales, Sydney, NSW, Australia

[3]Department of Microbiology and Infectious Diseases, Concord Repatriation General Hospital, Sydney, NSW, Australia

[4]Department of Cardiology, Sydney Adventist Hospital, Sydney, NSW, Australia

Abstract

Critical cardiac arrhythmias secondary to acute dengue fever are rare in adults and are likely to be underestimated. We present the first case of acute dengue fever complicated by recurrent sinus pauses and sinoatrial arrest requiring temporary pacing in the absence of other cardiac involvement. Early intervention to detect such arrhythmias may reduce the significant morbidity and mortality arising from this increasingly prevalent infection.

Introduction

Dengue is an arthropod borne viral illness prevalent in tropical and subtropical climates. The rising incidence of dengue infection represents an important global health issue. Critical cardiac conduction complications complicating dengue infection are infrequent in adults. We present a case of dengue fever complicated by sinus arrest requiring temporary cardiac pacing.

Case report

A previously well 35-year-old male commercial airline pilot presented with a two-day history of fevers, myalgia and macroscopic haematuria. He had returned from Bali, Indonesia five days prior to presentation, having sustained multiple mosquito bites during the trip. On examination, he was febrile with a temperature of 38.6°C, heart rate was 75 bpm and blood pressure were 114/69 mmHg. A diffuse, macular, blanching rash was evident over his torso. Initial laboratory investigations demonstrated a lymphopenia (lymphocytes 0.3 x 10^9/L) and mild thrombocytopenia (platelets 141 x 10^9/L). His creatine kinase was markedly elevated at 25,794 U/L. Liver function tests demonstrated a moderate transaminitis (ALT 140 U/L, AST 648U/L). Infectious mononucleosis screen, thick and thin films for malaria parasites and malaria antigen testing were negative. He was admitted to hospital for further investigation and management.

Further investigations including blood cultures and serology for Zika virus, HIV and Hepatitis A, B and C were negative. His dengue serology was positive for NS1 antigen and negative IgM and IgG (SD BIOLINE Dengue DUO®), with subsequent seroconversion for dengue IgG and IgM by neutralization assay confirming an acute and primary infection of dengue virus 3.

On day two of his admission, he had a syncopal episode post vomiting, with preceding dizziness. His electrocardiogram demonstrated sinus bradycardia (heart rate 48bpm) post episode. He was placed on cardiac monitoring and subsequently experienced another syncopal episode while seated at rest. Cardiac monitoring demonstrated a 10.2 second sinus pause (Figure 1a). On the same day, during a third syncopal episode while seated, cardiac monitoring demonstrated an 18.1 second episode of sinus arrest (Figure 1b). Isoprenaline infusion was commenced, and a temporary pacing wire was inserted, with pacing set to a VVI rate at 40min/min. Serial high sensitivity Troponin T and transthoracic echocardiography were normal. He remained haemodynamically stable with persistent sinus bradycardia (40-60bpm). The temporary pacing wire was removed on day five, when no further arrhythmias or pacing were seen.

Cardiac magnetic resonance imaging demonstrated normal cardiac size and function, with no evidence of myocarditis or myocardial oedema. An implantable loop recorder was inserted prior to discharge to monitor for any further cardiac events. At discharge, his creatine kinase, white cell count and platelet count normalised. Due to the episodes of sinus arrest with syncope, he was restricted from driving and flying until further review.

At one year follow up, the patient experienced no further syncopal events. Interrogation of his loop recorder did not demonstrate any further bradyarrhythmia, and his loop recorder was subsequently removed. He resumed driving and flying.

Although poorly recognized in adults, cardiac manifestations of dengue virus infection are an important contributor to dengue related morbidity and mortality [1] (Table 1). These include myocarditis most commonly, but also pericarditis, heart failure [1] and conduction abnormalities [2,3]. Whilst the underlying mechanism of dengue

Correspondence to: Sonali Gnanenthiran, Department of Cardiology, Concord Repatriation General Hospital, Sydney, NSW, Australia, E-mail: sgnanenthiran@georgeinstitute.org.au

Key words: *dengue; cardiac pacing; cardiac magnetic resonance imaging*

Received: February 01, 2022; **Accepted:** February 18, 2022; **Published:** February 21, 2022

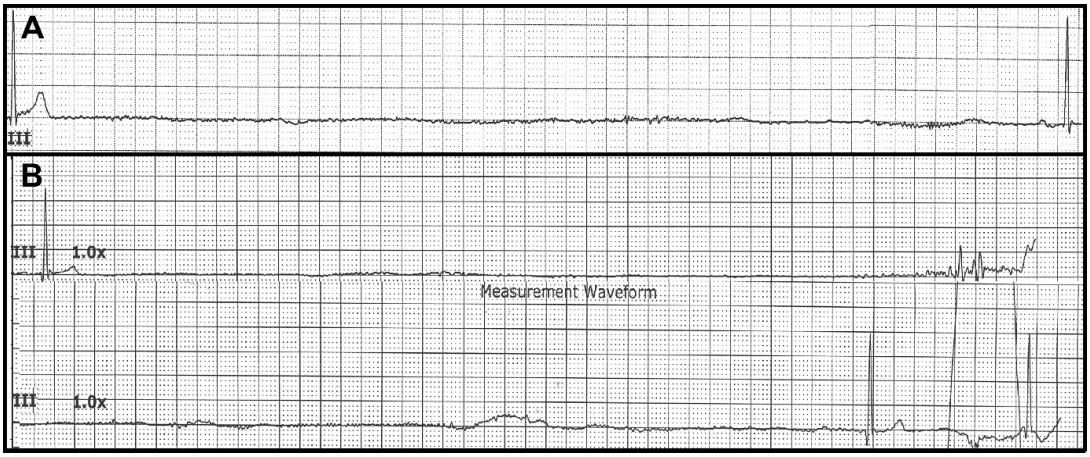

Figure 1. Electrocardiograms. (A) Cardiac monitoring demonstrated a 10.2 second sinus pause. (B) Cardiac monitoring demonstrated an 18.1 second episode of sinus arrest.

Table 1. Clinical Manifestations.

Phases		
	Febrile	• Headache, Eye pain • Myalgia, Arthralgia, Macular Rash
	Critical	• Severe complications in a small proportion of patients o Systemic vascular leak syndrome characterized by plasma leakage, bleeding, shock and organ impairment o Haemorrhagic manifestations o Moderate-to-severe thrombocytopenia
	Convalescent	• Recovery stage with vital signs stabilising
Organ involvement		
	Gastrointestinal	• Anorexia, nausea • Vomiting • Abdominal pain • Diarrhoea • Transaminitis
	Haematological	• Skin and/or mucosal bleeding • Petechiae or ecchymoses • Hepatomegaly • Lymphadenopathy • Leukopenia and thrombocytopenia
	Respiratory	• Cough • Sore throat • Nasal congestion
	Cardiovascular	• Myocarditis • Pericarditis • Heart failure • Conduction abnormalities: sinus bradycardia, first degree AV block, Mobitz type I and type II second-degree AV block, complete heart block, ventricular arrhythmia and sinus node dysfunction • T wave and ST-segment abnormalities,
	Neurological	• Encephalopathy • Seizures
	Renal	• Acute kidney injury • Rhabdomyolysis • Glomerulonephritis • Acute tubular necrosis

related cardiac rhythm abnormalities is not fully understood, it likely arises from myocardial inflammation from direct viral infection and immune-mediated damage. The inflammatory processes involving myocytes and the interstitium can alter membrane potentials and calcium homeostasis [4]. Additionally, changes in myocardial wall tension and oxygen consumption may promote arrhythmias. Similarly, dengue related myocyte fibrosis and atrophy may induce ectopic pacemaker activity [5]. Host response to viral infection and the subsequent release of cytokines are also thought to be contributory factors in arrhythmias. Elevated tumor necrosis factor-α, interleukins 6, 13 and 18, and cytotoxic factors that lead to increased vascular permeability and shock [6], may also play a role in the development of myocardial cell injury. Both cardiac and skeletal muscle have been identified as sites of dengue virus replication, and there is evidence for this in our case as our patient experienced a severe bradyarrhythmia as well as rhabdomyolysis with a markedly elevated creatine kinase. Rhabdomyolysis and cardiac abnormalities have been reported in dengue fever [7] but to our knowledge this is the first reported case with both such manifestations simultaneously. Interestingly in our case, sinus arrest occurred without other apparent cardiac involvement, as manifested by a normal troponin and cardiac MRI.

The majority of cardiac rhythm abnormalities in dengue fever have been reported in children, with few case reports in adults. Rhythm abnormalities occur in up to 30% of hospitalized patients, with sinus bradycardia the most frequent abnormality [8]. Other ECG changes include T wave and ST-segment abnormalities, first degree AV block, Mobitz type I and type II second-degree AV block, complete heart block, ventricular arrhythmia, and sinus node dysfunction. The majority of these are transient in nature; one case recovered after 5 months, and one case required a permanent pacemaker. In most cases these abnormalities have been described in association with severe dengue and typically occur during the recovery phase but can occur at any time during the illness. It is rare for patients to require specific treatment for arrhythmias experienced because of dengue fever and almost all completely recover as their illness resolves. Sinus pauses have been described in dengue fever [9], but the prolonged duration of pauses as well as the severe symptomatic nature of the pause are unique to our case. It is also extremely rare for patients to require temporary transvenous temporary cardiac pacing for the management of arrhythmias during dengue infection [10]. Our patient experienced a good long-term outcome with no post-infection arrhythmias, avoiding the need for permanent pacemaker implantation or change in career.

Conclusion

Critical cardiac arrhythmias secondary to acute dengue fever are rare in adults and are likely to be underestimated. To our knowledge, we report the first case of acute dengue fever complicated by recurrent sinus pauses and sinoatrial arrest requiring temporary pacing in the absence of other cardiac involvement. Early intervention to detect such arrhythmias may reduce the significant morbidity and mortality arising from this increasingly prevalent infection.

References

1. Miranda CH, de Carvalho Borges M, Matsuno AK, Vilar FC, Gali LG, et al. (2013) Evaluation of Cardiac Involvement During Dengue Viral Infection. *Clin Infect Dis* 57: 812–819. [Crossref]
2. Lateef A, Fisher DA, Tambyah PA (2007) Dengue and Relative Bradycardia. *Emerg Infect Dis* 13: 650–651. [Crossref]
3. Navinan MR, Yudhishdran J, Herath S, Liyanage I, Kugadas T, et al. (2015) Complete heart block in dengue complicating management of shock due to both bleeding and leakage: a case report. *BMC Res Notes* 8: 68–74. [Crossref]
4. Salgado DM, Eltit JM, Mansfield K, Panqueba C, Castro D, et al. (2010) Heart and Skeletal Muscle are Targets of Dengue Virus Infection. *Pediatr Infect Dis J* 29: 238–242. [Crossref]
5. Klein RM, Vester EG, Brehm MU, Dees H, Picard F, Niederacher D, et al. (2000) Inflammation of the myocardium as an arrhythmia trigger. *Z Kardiol* 89: 24–35. [Crossref]
6. Chen RF, Liu JW, Yeh WT, Wang L, Chang JC, et al. (2005) Altered T helper 1 reaction but not increase of virus load in patients with dengue hemorrhagic fever. *FEMS Immunol Med Microbiol* 44: 43–50. [Crossref]
7. Davis JS, Bourke P (2004) Rhabdomyolysis Associated with Dengue Virus Infection. *Clin Infect Dis* 38: e109–e111. [Crossref]
8. S Sheetal, Jacob E (2016) A study on the Cardiac Manifestations of Dengue. *J Assoc Physicians India* 64: 30–34. [Crossref]
9. La-Orkhun V, Supachokchaiwattana P, Lertsapcharoen P, Khongphatthanayothin A (2011) Spectrum of cardiac rhythm abnormalities and heart rate variability during the convalescent stage of dengue virus infection: a Holter study. *Ann Trop Paediatr* 31: 123–128. [Crossref]
10. de Mel S, Mowjood R, Kapuruge L, de Mel C, Path D (2015) Mobitz type I atrio-ventricular block in dengue myocarditis, requiring temporary pacing. *IDCases* 2: 99–101. [Crossref]