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Case Report

Mobitz type I atrio-ventricular block in dengue myocarditis, requiring temporary pacing

S. de Mel [a,1,*], R. Mowjood [b,1], L. Kapuruge [b], C. de Mel [b], D. Path [a]

[a] Department of Haematology–Oncology, National University Health System, 5 Lower Kent Ridge Road, Singapore 119074, Singapore
[b] Nawaloka Hospitals, Colombo, Sri Lanka

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ABSTRACT

We present a case of dengue myocarditis related Mobitz type I atrio-ventricular (A-V) block. To our knowledge, this is the first report of such a patient requiring pacing. An early response to methylprednisolone suggests the possibility of a therapeutic role for steroids in these patients.

Introduction

The arbovirus dengue is an important cause of morbidity and mortality in tropical countries. The most common cardiac manifestation of dengue viral infection is myocarditis [1]. Abnormalities of cardiac rhythm are a recognized complication of myocarditis and have been reported in association with dengue cardiac involvement [1]. A-V blocks are, however, a rare complication of dengue myocarditis. We report a case of Mobitz type I A-V block in a patient with dengue myocarditis who required transvenous pacing for symptomatic bradycardia.

Case report

A 20 year old Sri Lankan male from a suburban region presented with a two day history of fever. He complained of a mild headache and epigastric discomfort but denied chest pain or palpitations. He had no past medical history of note, and there was no family history of ischemic heart disease or rhythm abnormalities. He was on no regular medication and did not smoke or drink alcohol.

On admission, he had a pulse rate of 74 beats per minute (bpm) and a blood pressure of 120/82 mmHg. Physical examination revealed no abnormality, specifically, his heart sounds were normal with no murmurs and there was no clinical evidence of cardiac failure.

Initial investigations revealed a hemoglobin of 14.3 g/dl reference range (RF) [11.0–16.5], Hematocrit 51.4% RF [36.0–48.0], White Blood Cell count 6.1×10^9/L RF [4.0–11.0] and platelets 162×10^9/L RF [150–400]. His serum potassium was 4.1 mmol/l RF [3.6–5.2], while magnesium and calcium levels were within the normal range. His renal and liver functions were normal and a 12 lead electrocardiogram (ECG) on admission (Fig. 1A) revealed normal sinus rhythm at a rate of 70 bpm. Dengue IgG and IgM antibodies were positive by immunochromatography and his platelet count showed the expected fall to 40×10^9/L on day five of fever, recovering to normal by day nine.

On the morning of his fifth day of fever (third day of admission) he was found to be bradycardic at a rate of 50 bpm and complained of chest discomfort. His 12 lead ECG (Fig. 1B) revealed Mobitz type I A-V block, there was no ST elevation or evidence of ischemia. The patient was transferred to an intensive care unit and a temporary transvenous pacing wire was inserted in anticipation of significant bradycardia with hemodynamic compromise. During the evening of the same day his pulse rate dropped to 30 bpm, his blood pressure at the time was 118/60 mmHg, he complained of mild chest discomfort but was otherwise well. A two dimensional echocardiogram revealed normal ventricular function and no wall motion abnormalities. Pacing was commenced at this stage (Fig. 1C) and he was treated with intravenous methylprednisolone at a dose of 8 mg/kg per day for 2 days. Assays for troponin T, creatine phosphokinase (CPK) and myocardial bound CPK were all normal.

* Corresponding author. Tel.: +65 81424187/91381115.
E-mail address: sanjay_widanalage@nuhs.edu.sg (S. de Mel).
[1] These authors contributed equally to this work.

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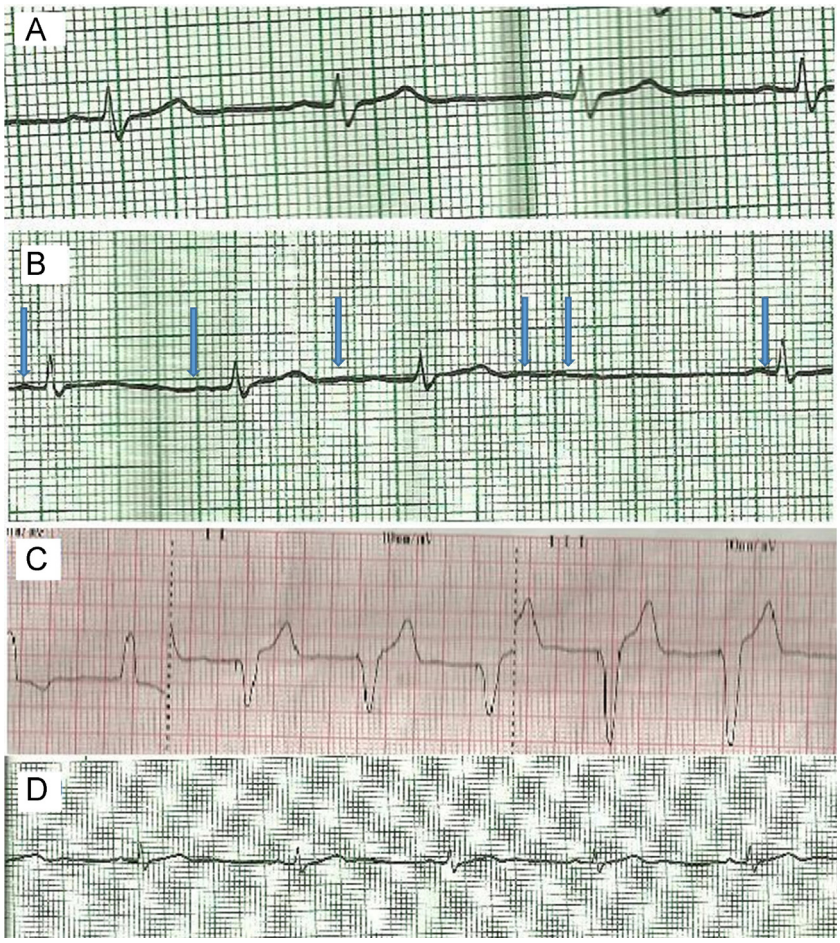

Fig. 1. (A) ECG on admission showing normal sinus rhythm. (B) ECG on day three showing Mobitz type I atrio-ventricular block. Blue arrows indicate the P waves. (C) ECG showing a paced rhythm on day three. (D) ECG following discontinuation of pacing showing normal sinus rhythm.

The A-V block resolved within one day of commencing steroids and we were able to remove his pacing wire after two days (fifth day of admission). He was transferred out of the intensive care unit on day seven and made an uneventful recovery. A 12 lead ECG (Fig. 1D) done after discontinuation of pacing revealed normal sinus rhythm.

Discussion

The most common cardiac manifestation of dengue viral infection is myocarditis, which is likely to occur with all four serovars of dengue but may be more prevalent in DEN 3 infection [1,2].

Myocardial inflammation is known to predispose to arrhythmias and three mechanisms are thought to contribute. Firstly, the inflammatory processes involving myocytes and the interstitium can lead to alterations in membrane potential. Secondly, changes in the parameters of ventricular dynamics such as increased wall tension and myocardial oxygen consumption also increase the potential for arrhythmias. Finally, fibrosis and secondary atrophy of myocytes can favor ectopic pacemaker development [3]. It is likely that the former two mechanisms play the predominant role in arrhythmias associated with dengue myocarditis which is an acute, reversible condition.

A variety of arrhythmias have been reported in association with dengue viral infection. Sinus bradycardia was shown to be particularly prevalent during one outbreak in Singapore although its use as a predictive factor for more malignant arrhythmias was not assessed [4]. In a series of 250 patients from Kerala (India), 16.8% had bradycardia. The majority of these were sinus bradycardia while two patients were reported to have Mobitz type 1 and 2 A-V block respectively [5]. Transient ventricular ectopy and atrial fibrillation have also been reported although neither case was associated with hemodynamic compromise [6,7].

Mobitz type 1 A-V block in dengue viral infection has been reported in children although the frequency of this complication in the pediatric setting is uncertain [8]. The two children in question were both in the recovery phase, their arrhythmias resolved spontaneously and they did not require pacing. Mobitz type 1 A-V block in adults has been reported in the acute and deferverscent phases of dengue infection, spontaneous resolution of the A-V block occurred in each case [5,9,10]. To the best of our knowledge, ours is the first report of such a patient developing symptomatic bradycardia which required temporary pacing.

There is a paucity of randomized controlled trial evidence for the use of intravenous corticosteroids in viral myocarditis. They have, however, been shown to be effective in acute myocarditis of other etiologies, in conjunction with intravenous immunoglobulin, or cyclosporine with anti CD3 monoclonal antibody therapy (muromonab) [11,12]. We therefore suggest that the use of steroids as a therapeutic option in dengue myocarditis be further evaluated by randomized controlled trials.

Our case also illustrates that Mobitz type 1 atrio-ventricular block associated with dengue viral infection is a potentially life threatening condition and close monitoring with consideration of early pacing is warranted in these patients.

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