DOI: 10.7860/JCDR/2015/13375.6514

Letter to Editor

Internal Medicine Section

Atrial Fibrillation Due to Acute Myocarditis During Dengue Haemorrhagic Fever

HANS RAJ PAHADIYA[1], VEERAM PARMAR[2], HARISH KUMAR[3], AMIT SAGAR[4]

Keywords: Electrocardiograph, Hypotension, Myocardial damage

Sir,

Electrocardiograph (ECG) changes are common after dengue viral infection. This include sinus bradycardia, increase PR interval, ST-T changes, AV blocks, junctional rhythm, ventricular premature beats, sinus node dysfunction and atrial fibrillation (AF). These are usually self limiting and resolve after recovery of the illness [1,2]. Clinical manifestations of cardiac complications after Dengue Haemorrhagic fever (DHF) varies considerably, from self-limiting tachy-brady arrhythmia, AV blocks to acute myocarditis with severe myocardial damage, leading to hypotension and pulmonary oedema [3]. AF is a very rare manifestation.

A 20-year-old male came to the Department of Medicine with history of high grade fever with chills since five days. He also had complaints of arthralgia, back pain and myalgia. At the time of admission he was febrile and blanching rashes were seen over his chest. His temperature was 38.4^{0}C by axilla, blood pressure (BP) was 120/80 mmHg, pulse rate 102 beats/minutes, regular and respiratory rate was 18 breaths/minutes. Cardiovascular and respiratory system revealed no significant abnormality. Complete blood count (CBC) revealed haemoglobin (Hb) 14.4 g/dL, hematocrit 49%, white cell count 3800/mm^3, and platelets 25,000/mm^3. Serum bilirubin was 1.54 mg/dL, SGPT 63 U/L and SGOT was 78 U/L. Renal function, electrolytes and coagulation profile were within normal limit. The Chest X-ray and ECG were normal. Peripheral blood film had no evidence of Malaria parasite. The antigen testing including parasite lactate dehydrogenase (LDH) tested negative for malaria. NS1 antigen and IgM antibodies were positive for dengue virus. He was treated with intravenous fluids in the form of normal saline, GNS and antipyretic with close monitoring. Successive CBC revealed decrease in the number of platelets. On the 3rd day of admission Hb was 14.2 g/dL and platelets were 15000/mm^3. He had complaint of dyspnoea. He was noted to have an irregularly irregular pulse. The BP was 96/68 mmHg. ECG showed AF with serum electrolytes as Na^+ 135 meq/L, K^+ 4.5 meq/L, Ca^{+2} 8.9 meq/L and Mg^{+2} 4.6 meq/L. Cardiac enzyme, CPKMB was 136 U/L (5-10) and Troponin-I was negative. Thyroid hormone profile was normal. A trans-thoracic two-dimensional echocardiography was done which showed a structurally normal heart with normal valvular morphology, normal chamber sizes and 60% left ventricular ejection fraction. Ultrasonography showed mild bilateral pleural effusion and mild ascites. The diagnosis of DHF was confirmed by evidence of fever, rashes, thrombocytopenia, leukocytopenia, haemoconcentration, mild bilateral plural effusion and mild ascites with positive serological tests. He was given three units of single donor apheresis platelets, 15 units' of random donor platelets. Patient did not show any signs of improvement, hence dexamethasone was started on 6th day, at dosage of 8mg q8h for the period of next 5 days. The patient became asymptomatic and AF reverted to normal sinus rhythm on 8th day of the admission. Platelet count improved to 55,000/mm^3 and 110,000/mm^3 on 8th and 10th day respectively. The patient was discharged on 12th day in haemodynamically stable, afebrile condition with normal haematological parameters.

The patient had AF probably secondary to myocarditis as CPKMB was elevated suggesting myocardial damage by dengue virus infection, as CPK-MB is a marker of myocardium damage. The AF in our patient reverted after symptomatic treatment and with dexamethasone. Dengue virus infection causes myocardial damage, either by direct invasion or an autoimmune reaction, which may result in myocarditis [4]. Salgado et al., [5] supported the hypothesis of myocarditis due to direct infection of cardiac muscle with dengue virus. They explained cardiac arrhythmias and altered contractility in dengue myocarditis secondary to increased resting intracellular calcium in the viral infected myocytes and skeletal muscles along with this, there is increased expression of inflammatory genes and protein IP- 10 after direct infection of muscle by dengue virus in one child with fatal DHF using immunofluorescence confocal microscopy in heart tissue. Myocardium tissue was almost normal with minimal inflamation [5]. One case report also explained the direct action of dengue virus on myocardium in a patient of fatal dengue myocarditis with cardiogenic shock. The histopathology of heart tissue showed multifocal areas of muscle necrosis and intense interstitial oedema associated with clusters of virus particles inside the cardiomyocytes and in the interstitial space [6].

The myocardial injury in dengue may be due to release of cytokine mediators and/or cellular components of the immune response. The patients with DHS and DSS have higher level of TNF-α, IL-6, IL-13 and IL-18, and cytotoxic factor which increase vascular permeability and shock [7]. The chances of occurrence of DHF and DSS is increased in the subjects who experience secondary dengue virus infections because of formation of both serotype-specific and serotype-cross-reactive memory T cells following primary infection which augments damage by causing exaggerated release of various above mentioned inflammatory substances and cytotoxic factors [8]. The mechanism of thrombocytopenia in dengue viral infection may be due to peripheral destruction of antibody coated platelets [9]. Steroids are used in immune thrombocytopenic purpura to increase platelets. Hence we assume that our patient responded to dexamethasone therapy which also has an anti-inflammatory effect. Recovery may also be due to clearing off of dengue viral antigens gradually. The role of steroid is still not clear [10]. A case has been reported of repeated dengue shock syndrome and dengue myocarditis responding dramatically to a single dose of methyl prednisolone [11].

AF due to febrile illness is usually transient and it resolves spontaneously after the recovery of the illness in most of the cases [1,2]. In the case of persistence and unstable AF, pure myocardial injury may be there and requires anti arrhythmic treatment in most of the situation [9,10].

AF should be considered as a complication of DHF and it should be mandatory to monitor ECG of such patients and potential role of steroid in DHF with myocarditis should be looked into in future.

REFERENCES

[1] Bhatia V, Parida AK, Arora P, Mittal A, Pandey AK, Singh G, et al. Electrocardiographic and echocardiographic findings during the recent outbreak of viral fever in National Capital Region. *Indian Heart J*. 2007;59:360-62.

[2] 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.

[3] Lee IK, Lee WH, Liu JW, Yang KD. Acute myocarditis in dengue Haemorrhagic fever: a case report and review of cardiac complications in dengue-affected patients. *Int J Infect Dis*. 2010;14:919-22.

[4] 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.

[5] Salgado DM, Eltit JM, Mansfield K, et al. Heart and skeletal muscle are targets of dengue virus infection. *Pediatr Infect Dis J*. 2010;29:238-42.

[6] Miranda CH, Borges Mde C, Schmidt A, Pazin-Filho A, Rossi MA, Ramos SG, et al. A case presentation of a fatal dengue myocarditis showing evidence for dengue virus-induced lesion. *Eur Heart J Acute Cardiovasc Care*. 2013;2(2):127-30.

[7] Mustafa AS, Elbishbishi EA, Agarwal R, Chaturvedi UC. Elevated levels of interleukin-13 and IL-18 in patients with dengue Haemorrhagic fever. *FEMS Immunol Med Microbiol*. 2001;30(3):229-33.

[8] Kurane I, Innis BL, Nimmannitya S, et al. Human immune responses to dengue viruses. *Southeast Asian J Trop Med Public Health*. 1990;21:658-62.

[9] Shashidhara KC, Murthy K AS, Gowdappa HB, Bhograj A. Effect of high dose steroid on platelet count in acute stage of dengue fever with thrombocytopenia. *J Clin Diagn Res*. 2013;7(7):1397-400.

[10] Horta Veloso H, Ferreira Junior JA, Braga de Paiva JM, Faria Honorio J, Junqueira Bellei NC, Vecenzo de Paola AA. Acute atrial fibrillation during dengue Haemorrhagic fever. *Braz J Infect Dis*. 2003;7:418-22.

[11] Mahmod M, Darul ND, Mokhtar I, Nor NM, Anshar FM, Maskon O. Atrial fibrillation as a complication of dengue Haemorrhagic fever: non-self-limiting manifestation. *Int J Infect Dis*. 2009;13:316-18.

PARTICULARS OF CONTRIBUTORS:

1. Senior Resident, Department of Medicine, Dr. SNMC, Jodhpur, Rajasthan, India.
2. Senior Resident, Department of Medicine, Dr. SNMC, Jodhpur, Rajasthan, India.
3. Senior Resident, Department of Medicine, Dr. SPMC, Bikaner Rajasthan, India.
4. Assistant Professor, Department of Medicine, Dr. SNMC, Jodhpur Rajasthan, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Hans Raj Pahadiya,
Senior Resident, Department of Medicine, MG Hospital, Jodhpur, Rajasthan-342001, India.
E-mail : drhans05sms@gmail.com

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Feb 08, 2015**
Date of Peer Review: **May 12, 2015**
Date of Acceptance: **May 28, 2015**
Date of Publishing: **Sep 01, 2015**