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Case Report

Dengue Myocarditis in Healthy Young Male Athlete

Tan YP *, Cheong WC, Yusoof HM, Zain MM, Lau IS, and Rosman A

Department of Medicine, Selayang Hospital, Malaysia

***Corresponding author**

Yu Peng Tan, Department of Medicine, Selayang Hospital, Lebuh Raya Selayang-kepong, 68100 Batu Caves, Selangor, Malaysia, Email: yupengtan@yahoo.com

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Abstract

We report a case of dengue myocarditis involving young male athlete with good recovery. A 14 year-old gentleman presented with a 5-day fever with chest discomfort and typical viral fever presentation. His preliminary diagnosis of dengue fever was confirmed via positive non-structural protein-1 and the ensuing polymerase chain reaction (PCR) confirmed dengue virus serotype DEN 1. He had myocarditis manifested by his electrocardiogram which revealed deepening of T inversion from V1 to V4 with notably biphasic T inversion mimicking Wellen's Syndrome. The T inversion resolved as he recovered. The cardiac enzyme troponin T was positive (0.07 µcg/L; normal < 0.03 µcg/L). The echocardiography done showed normal chambers with no regional wall abnormalities. He recovered with only supportive management and intravenous normal saline hydration.

INTRODUCTION

Dengue fever is a mosquito-borne disease or arthropod-borne viral disease caused by flavivirus with 4 serotypes namely DEN 1, DEN 2, DEN 3 and DEN 4. Clinically dengue presents as with or without warning signs and severe dengue with or without shock (compensated and decompensated). There are 3 distinct phases during dengue fever. The febrile phase is usually lasted about 3 to 7 days. This is followed by critical phase denoted by the time of defervescence (temperature < 38°C). After that the body will enter recovery phase with the normalization of blood counts and reabsorption of the body fluid leaked to third space during the illness. There are atypical presentation for dengue like bone marrow involvement (Hemophagocytic Lymphohistiocytosis HLH), myocarditis, myositis, encephalitis, fulminant hepatitis and retinitis. The myocarditis is caused by inflammation of myocardium which commonly caused by viral aetiologies like dengue virus, herpes and enteroviruses [1].

CASE PRESENTATION

A healthy 14-year-old male athlete presented to the emergency department with a 5-day history of high grade fever at home. He complained of vomiting for the first two days and developed generalized rashes at day 4. He had chest discomfort during his arrival to emergency department. The chest discomfort was described as heaviness over precordium lasting about 1 hour without perspiration or numbness over limbs.

On presentation his tympanic temperature was 39.3°C, respiratory rate was 19 breaths per min, heart rate was 98 beats per min and blood pressure at 102/60 mmHg. He was alert and oriented to time, place and person. His hydration status was good. Cyanosis or cold extremities were absent. There were maculopapular petechial rashes over his body and limbs. His heart sounds and breath sounds were normal. His abdomen was soft and non-tender. The remainder of his physical examination was unremarkable (Figure 1).

He had leucopenia (TWC 1.4 × 10^9; normal range 4.0–11.0 × 10^9) and thrombocytopenia (65× 10^9/L; normal range 170–420 × 10^9/L). Hematocrit was 40.5 (normal range 41–51 %) with a hemoglobin of 12.3 g/dL (normal range 13–17 g/dL). As for the cardiac biomarkers the creatinine kinase was 424u/L, lactate dehydrogenase LDH was 742u/L. The troponin T serum level

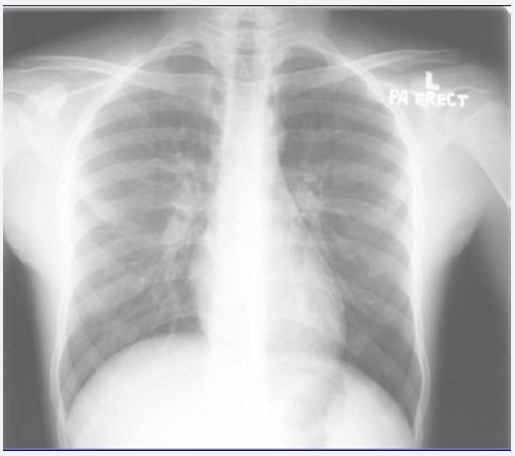

Figure 1 Chest radiograph taken during admission which is clear and without any pulmonary infiltrates.

was 0.07 µg/L (normal range < 0.03 µg/L). He had transaminitis with alkaline transaminases (ALT) of 131u/L.

An electrocardiogram (ECG) on presentation revealed sinus rhythm, normal voltage QRS complex but T wave inversion involved chest leads V1 to V4 with biphasic T inversion at V2 and V3. On the second day of admission the subsequent ECG revealed the biphasic T inversion progressed to involve chest lead V3 and V4. Both of the electrocardiograms were performed when he experienced chest discomfort. The chest radiography was clear of pulmonary infiltrates. There was no evidence to suggest chest infection induced pleurisy.

He was given intravenous hydration according to his progress of dengue with the diagnosis of dengue with warning signs complicated by myocarditis. While in the high dependency unit, he complained of central chest discomfort with 2 times vomiting but there was no diaphoresis. The chest discomfort lasted about 2 hours. He was subsequently nursed in coronary care unit. Transthoracic echocardiography revealed normal structured heart with no regional wall abnormalities and ejection fraction of 60%.

He received treatment of intravenous normal saline and closed monitoring. He recovered uneventfully throughout the phase of febrile and afebrile for 48 hours. While his clinical status improved his biphasic T inversion normalized. He was discharged

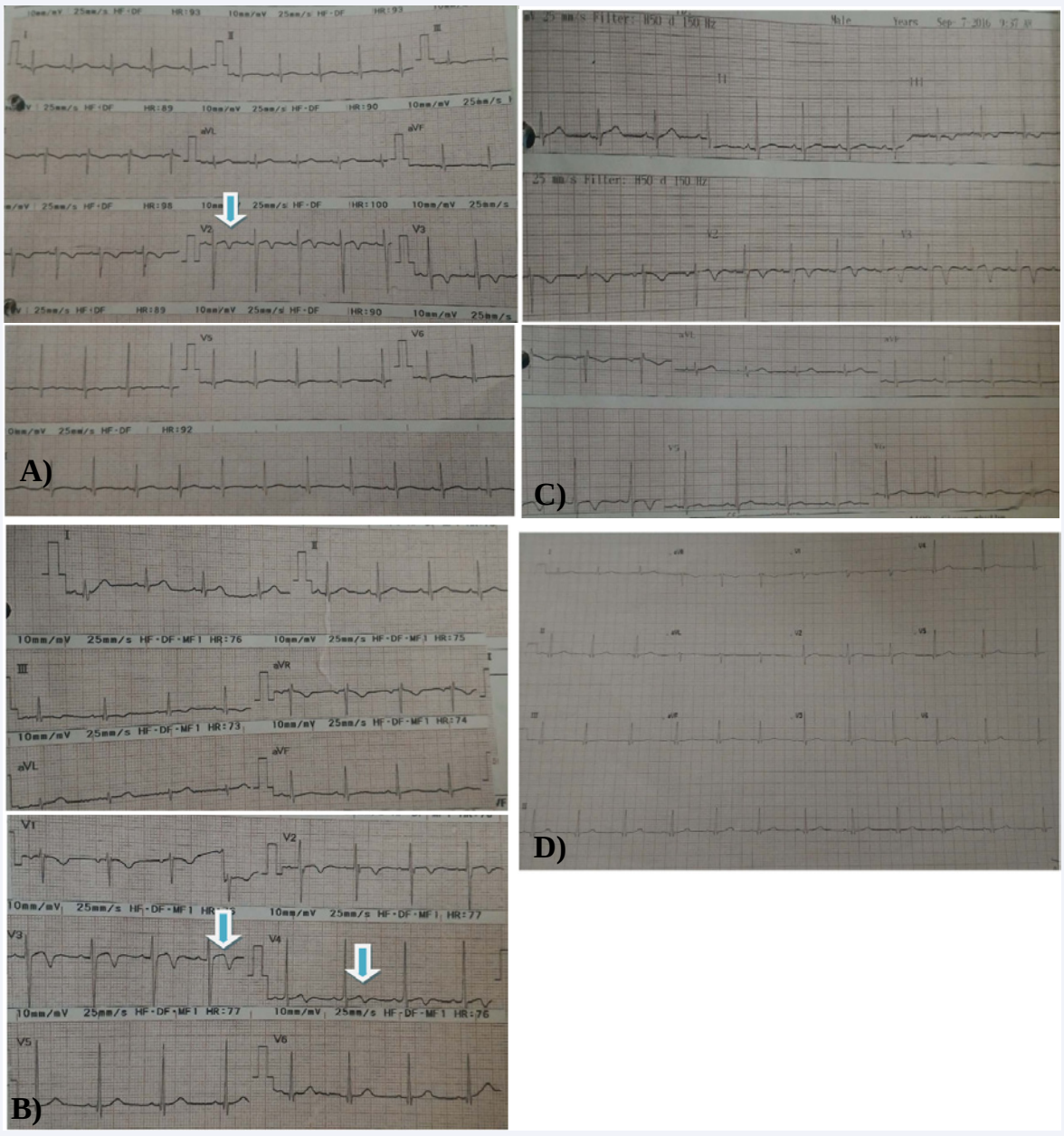

Figure 2 A electrocardiogram (Panel A) showed biphasic T inversion (Arrow) involving chest lead V2 with T inversion V1 to V4 on the day of admission. Panel B showed the progression of biphasic T inversion involving adjacent chest lead (Arrow) on the next day. Panel C revealed the resolution of biphasic T inversion on the 3rd day. Panel D showed the resolution of T inversion on day 4.

after full resolution of symptoms, platlet count of 80 × 109/L and haematocrit of 40.0%. He was well at clinic review a month later (Figure 2 A-D).

DISCUSSION

Dengue myocarditis has spectrum from mild case of only ECG changes to severe case of myocardial infarction induced acute heart failure (cardiogenic shock) [2,3]. Wali et al., reported mean ejection fraction of 47.06% in dengue hemorrhagic fever and 39% in dengue shock syndrome [4]. The myocardial dysfunction is usually transient except minority cases which lead to fulminant myocarditis causing fatalities [3]. This young athlete has acute myocarditis as he presented with unexplained chest pain with cardiac conduction disturbance mimicking Wellen's Syndrome and raised troponin T level. There was no drug reaction. In lieu of the endomyocardial biopsy in a rather well patient we proceeded with detection of viral genome using polymerase chain reaction to identify dengue virus.

There is no conclusive pathogenic mechanism for dengue myocarditis. Direct viral invasion of cardiomyocytes, myocardial edema from local capillary leakage, overwhelming cytokine effect, coronary hypoperfusion and intracellular calcium homeostasis disruption have just partly explained the pathophysiology [3].

The electrocardiogram changes in dengue myocarditis can happen in any phase of dengue which may include bradyarrythmia such as first and second degree heart block or tachyarrthmia for example atrial fibrillation [5]. Sharma et al., reported Mobitz type I and AV dissociation in 18 year old male whom recovered spontaneously [6]. Majority of the ECG changes are benign and self-limiting. Most of the myocarditis patients do not have elevated cardiac markers level [7].

The T inversion and biphasic T inversion noted in the febrile phase of patient suggested the presence of myocardial ischemia. With appropriate intravenous hydration therapy the biphasic T inversion as signs of myocardial ischemia resolved uneventfully.

CONCLUSION

Cardiac arrhythmia and biphasic T inversion can happen in dengue fever. The electrocardiogram changes in dengue fever are not uncommon. Once myocardial involvement in dengue fever is recognized careful monitoring with prudent fluid administration is the key for management.

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