

Case Study

Dengue fever complicated with Non-STEMI

Maheswaran Umakanth

Lecturer in medicine, Faculty of Health Care Sciences, Eastern University, Sri Lanka

***Corresponding Author:**

Maheswaran Umakanth

Email: mumakanth1972@gmail.com

Abstract: Dengue is a vector born viral infection that belongs to family Flaviviridae. It is a single stranded RNA virus and endangers 2.5 billion people world wide. Dengue viral (DENV) infections cause a broad spectrum of illnesses from self-limited fever to severe hemorrhagic manifestations and increased vascular permeability. Cardiac manifestations of dengue are rarely associated with severe dengue fever. There are ample of dengue with myocarditis reported all over the World. But dengue with acute myocardial infarction (AMI) is rare, we reported dengue with AMI during the last outbreak and also we reported our challenges faced during the management period.

Keywords: Dengue, acute myocardial infarction, Aedes mosquitoes

INTRODUCTION

Dengue is a viral infection transmitted by Aedes mosquitoes[1]. Dengue is a flavivirus with four different antigenically diverse serotypes. It is a rapidly growing health problem with an anticipated 2.5 billion people at risk, mainly in countries of south and south-east Asia, the Caribbean, Central and South America, and more recently in Africa. The spread of dengue is thought to be due to a combination of factors such as increased urbanization, population expansion, migration and international travel and the difficulties of effective vector control. It is estimated that there are between 50 and 100 million cases of dengue each year, of which 500 000 are severe life-threatening infections[2]. There have been numerous urban outbreaks of dengue with significant health and economic impacts.

Cardiac conduction disorders such as atrio-ventricular blocks, sinus node dysfunction, and ectopic ventricular beats have been reported during episodes of Dengue Hemorrhagic Fever (DHF)[3]. Myocarditis is a rare complication of dengue fever and it rarely mimics acute myocardial infarction (MI)[4].

Dengue virus (DENV) is a small single-stranded RNA virus comprising four distinct serotypes (DEN-1 to 4). These strongly related serotypes of the dengue virus belongs to the genus flavivirus, family Flaviviridae[2]. Overall, the drivers behind the global expansion in diseases are thought to include certain vector and host factors, including the urban-adapted Aedes mosquito vector becoming newly established in many areas of the world through distribution on cargo ships, globalization and increase in breeding sites

through rapid and often poorly planned urbanization of cities[5].

According to the SriLankan epidemiology unit report that first three month of 2017, around 30,000 cases were reported, of that 41.32% were reported from western province[6]. Secondary dengue viral infection is more common in our clinical practice. When compared with primary dengue infection, secondary dengue infection is associated with more severe clinical presentation[7]. Dengue virus and the dengue virus-encoded NS₁ are present in blood during the acute phase, and elevated early viremia and NS1 antigenemia have been associated with more severe clinical presentations[8].

CASE REPORT

A 35- year-old gentle man complained of fever, myalgia, lethargy cough and joints pain for 2 days duration. Third day of fever he complained left sided chest pain. Despite his lethargy, he was still able to maintain good oral intake and urine output. Physical examination revealed a temperature of 39°C pulse rate was 80/min with blood pressure of 110/90mmHg. The patient did not have a rash and his systemic examination was unremarkable. A cardiovascular and precordium assessment revealed no audible murmurs or other cardiac abnormalities. Total blood count was $3 \times 10^9/L$, with low platelets of $30 \times 10^9/L$, prothrombin time 10.2 s (control, 10.6 s), activated partial thromboplastin time (APTT) 30.4 s (control, 30.2 s), liver function tests showed elevated alanine aminotransferase and aspartate aminotransferase levels of 49 U/L and 153 U/L,

respectively. A chest radiograph disclosed normal lungs.

The tourniquet test was negative. He did not have any history of bleeding, cough, or altered sensorium. He did not have any significant past history of cardiac problem. But his haematocrit started to rise by more than 20% from the base line.

Routine ECG was done which showed sinus rhythm and occasional premature ventricular contractions (PVC) with ST and T wave ischemic changes in the antero-lateral leads. Cardiac specific Troponin-I was highly positive, and then diagnosis was made as Non-STEMI. Urgent transthoracic echocardiography was done which showed lateral ischemia. Sixth day of fever blood sent for dengue antibody test which was positive in both IgM and IgG antibodies. He was started intravenous fluid as his haematocrit started to rise. We did not start anticoagulation and anti platelets as his platelets below $50 \times 10^9/l$.

DISCUSSION

Dengue viral infections cause a broad spectrum of illnesses from self-limited fever to severe hemorrhagic manifestations and fatal complications. Dengue fever rarely complicated with cardiac involvement. Available literature in the pubmed revealed that mild cardiac involvement such as bradycardia, heart block, ventricular arrhythmia, severe form of myocarditis, left ventricular impairments and rarely acute coronary syndrome.

Myocardial involvement mainly due to direct infection of dengue virus on myocardium and involvement of immunological mediators or both[9]. Sometime acute dengue myocarditis mimics myocardial infarction. In both condition non specific ECG changes are common. The endomyocardial biopsy is the gold standard investigation of choice for diagnosing myocarditis. Most of the time cardiac involvements are self limiting and any ECG changes in case of dengue fever, we always think about myocarditis rather than acute myocardial infarction[4]. Cardiac markers of myocardial injury, especially increased troponin I, were helpful to confirm diagnosis. However, most myocarditis patients do not have high levels of cardiac biomarkers[11].

Kularatne et al reported in Sri Lanka showed that 62.5% of 120 adults with dengue fever (DF) had an abnormal electrocardiogram[12]. Burhanuddin et al conducted a study in Indonesia which revealed that troponin level seen slightly higher in DSS than DHF but both in cases troponin level are within normal range³. This indicates that high level of troponin in case of dengue fever is significant it could be due to

myocarditis or MI. There are some reported cases revealed that possible elevated troponin other than MI or myocarditis, could be due arrhythmia[3]. This patient did not show any arrhythmic events.

Eventhough we have diagnosed Non-STEMI we were unable to start antiplatelets and anticoagulation. Management of a patient with MI who is also at a risk of bleeding is a difficult situation and balancing between two life threatening conditions can be challenging. According to the available evidence better to withhold both antiplatelets and anticoagulation when the platelets count dropped below $50 \times 10^9/l$ [13]. Furthermore, there are no strategy in literature on management of such patients and therefore it becomes imperative that clinicians contribute to their experiences in managing such tricky patients. As there are no guidelines on management of anticoagulation in the face of dengue-induced thrombocytopenia, initially we didn't start any anticoagulation or antiplatelets because trend of platelets was dropping. Once the platelets started to raised beyond $50 \times 10^9/l$ then we started one antiplatelets.

Regarding anticoagulation, in these high-risk patients one usually starts with unfractionated heparin (UFH) as a substitute of low-molecular weight heparins (LMWH). UFH has a shorter half-life and can be blocked, monitored, and potentially better reversed immediately when severe bleeding occurs. When patients remain without bleeding after 48 hours, one may switch to LMWH[14]. These antiplatelets and anticoagulation concepts purely based on immune thrombocytopenia (ITP) and cancer related venous thrombo-embolism. Can we apply this management to a patient who had dengue with low platelets? We didn't start anticoagulation to this patient. However, while he was in the ward we started aspirin as a monotherapy.

CONCLUSION

The dengue virus is competent to involve multiple organ systems concurrently or in isolation. Atypical manifestations, such as myocarditis, dysrhythmias and MI are usually mild or even asymptomatic but can have serious implications on patient outcomes. This case highlights the necessity of awareness. A better understanding of cardiac complications will potentially improve the treatment of dengue illness by avoiding otherwise preventable morbidity and mortality in the affected patients. Future studies should be undertaken to develop clinical guidelines for patients on mandatory anticoagulation who have dengue with warning signs or are at risk of bleeding. A co-ordinated multidisciplinary approach is necessary.

Conflict of interest

There is no conflict of interest

REFERENCES

1. Halstead, S. B. (2007). Dengue. *The Lancet*, 370(9599), 1644-1652.
2. World Health Organization. (2009). Dengue guidelines for diagnosis, treatment, prevention and control: new edition.
3. Iskandar B, Juherinah DD, Febriani AD. The Levels of Troponin T in Patients with Dengue Hemorrhagic Fever. *American Journal of Clinical and Experimental Medicine*. 2015;3(4):149-53.
4. Patra, S., Bhardwaj, G., Manohar, J. S., Srinivasa, K. H., Kharge, J., & Manjunath, C. N. (2013). Acute myocardial infarction being the presentation of dengue myocarditis. *Journal of cardiovascular disease research*, 4(2), 159-161.
5. Guzmán, M. G., & Kourí, G. (2004). Dengue diagnosis, advances and challenges. *International journal of infectious diseases*, 8(2), 69-80.
6. Senanayake, N., Gurunathan, G., Hart, T. B., Amerasinghe, P., Babapulle, M., Ellapola, S. B., ... & Basanayake, V. (1993). An epidemiological study of the health of Sri Lankan tea plantation workers associated with long term exposure to paraquat. *Occupational and Environmental Medicine*, 50(3), 257-263.
7. Vaughn, D. W., Green, S., Kalayanarooj, S., Innis, B. L., Nimmannitya, S., Suntayakorn, S., ... & Nisalak, A. (2000). Dengue viremia titer, antibody response pattern, and virus serotype correlate with disease severity. *The Journal of infectious diseases*, 181(1), 2-9.
8. Anam, A. M., & Rabbani, R. (2013). Ogilvie's syndrome in severe dengue. *The Lancet*, 381(9867), 698.
9. Wali, J. P., Biswas, A., Chandra, S., Malhotra, A., Aggarwal, P., Handa, R., ... & Bahl, V. K. (1998). Cardiac involvement in dengue haemorrhagic fever. *International journal of cardiology*, 64(1), 31-36.
10. Lee, K., Lee, W. H., Liu, J. W., & Yang, K. D. (2010). Acute myocarditis in dengue hemorrhagic fever: a case report and review of cardiac complications in dengue-affected patients. *International Journal of Infectious Diseases*, 14(10), e919-e922.