Case Report

Dengue Fever with Thrombocytopenia with Myocarditis

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ABSTRACT

There has been a resurgence of cases of Dengue Fever with Thrombocytopenia recently. The mortality rates due to Dengue have also increased. The most common cause of mortality in Dengue fever has been reported to be due to Renal Failure. Cardiac involvement is relatively rare in Dengue Fever but is seen in the form of Cardiac Arrhythmias; Atrial fibrillation, Ventricular premature contractions, Tachy-brady syndrome, Blocks, ST-T changes, and very rarely Myocarditis and Acute pulmonary oedema. Left ventricular dysfunction has been described in Dengue Fever with thrombocytopenia and may be a contributory factor for morbidity and mortality in Dengue Shock Syndrome.

Here we report a case of Dengue Fever with thrombocytopenia in a young female who developed Supraventricular tachycardia due to myocarditis. She was treated aggressively with supportive measures and survived the potentially fatal complication. Hence it is important to have a high index of suspicion for myocarditis in Dengue Fever with Thrombocytopenia and shock.

Key words : Dengue Fever, Thrombocytopenia, Cardiac arrhythmia, Myocarditis

Introduction -

Dengue, an arboviral infection transmitted by Aedes aegypti and Aedes albopictus mosquitoes, is emerging as the most important mosquito-borne viral disease. It is a serious global public health problem, with 2.5 billion people at risk and an annual range of 50 to 390 million infections, which include dengue fever, dengue hemorrhagic fever (DHF), and dengue shock syndrome (DSS). Between 2006 and 2012, India reported an annual average of 20,474 dengue cases¹. The disease burden is much more after correcting for underreporting.

Mortality rate due to DHF in adults ranges from 1-8 per cent. An increasing moratlity upto 14 per cent is a cause for concern. Acute renal failure has been consistently identified as a risk factor of mortality in patients with DHF. The 2009 WHO guidelines on dengue has included organ dysfunction as an indicator for mortality. Cardiac involvement was underestimated but now should be considered as a major cause of morbidity and mortality.

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Case report -

A 35 old female patient was admitted with complaints of high grade continuous fever, severe backache and bodyache since 5 days and rash all over body since 1 day. Patient also complained of headache and generalized weakness. There was no history of oliguria, cough, rhinitis or any bleeding tendencies. She did not have any comorbidity. On admission she looked very ill and was febrile with temperature 103.6° F, pulse rate 110/min and respiratory rate of 16/min. Her Blood Pressure was 90/60 mm of Hg. She had a erythematous, maculopapular rash all over the body (Figure 1). It was nonitchy and palms and soles were spared. Systemic examination was normal. Her investigations on admission revealed haemoglobin of 12.8 gms%, TLC = 2100/cu mm, DLC Polymorphs - 53%, Lymphocytes 45%, Eosinophils 1% and Monocytes 1%. Her platelet count was 32,000/cu mm and platelets were also depleted on peripheral smear. Serum Bilirubin was 2.9 mg/dl, AST - 65 IU and ALT 97 IU. Her NS1, IgM, IgG antibodies for Dengue virus were strongly positive. Serotyping and quantitative estimation was not done. Serological tests for leptospirosis and H1N1 were negative. Her renal functions were normal and X-ray chest was normal. Her Electrocardiogram (ECG) was normal on admission. She was started on supportive treatment with antipyretics, Intravenous fluids and antibiotic Amoxycillin + clavulinic acid.

On next day she developed breathlessness and palpitations, her blood Pressure dropped to 70 mm of Hg, her oxygen saturation dropped to 78% and chest examination revealed bilateral basal crepts. Her ECG showed Supraventricular tachycardia (Figure 2). It was immediately reverted by Intravenous Diltiazem. Her 2 D ECHO examination revealed global hypokinesia with observed ejection fraction of 35%. With suspicion of myocarditis her CPK MB enzyme levels were estimated which were elevated to 52 IU. Her C-reactive protein levels were 8.4 mg/dl. Her platelet count had dropped to 13,000/cu mm by then. She was given 4 units of platelet transfusions, Oral L- carnitine, Oral carvedilol and was put on noninvasive ventilation. She was also given low dose diuretics. She responded to treatment and her platelet count improved to 64,000/cu mm on 4th day and her fever subsided and she improved symptomatically. She was weaned of ventilatory support and discharged after 8 days. Her 2 D ECHO on discharge showed improvement with ejection fraction of 47%.

Discussion -

In the majority of infected people, dengue is a self limited disease that resolves in 57 days. However, approximately 500,000 people develop a severe form, leading to about 20,000 deaths annually. Consequently, approximately 0.5% of dengue patients develop a severe form and require a specialized treatment².

Neerja M. et al in their study documented Unusual and rare manifestations of dengue in 66% patients. Of these Hepatitis was observed in 70 % of the cases. Pleural effusion was seen in 11%, acute renal failure in 10 %, neurological complications such as encephalitis in 7.4 %, myocarditis in 9 %, and bleeding gastric ulcers in 3.4 % of the cases. DENV serotype 2 was more prevalent in patients with unusual manifestations of dengue in their study. The WHO classification system does not include unusual and rare manifestations; hence, it is essential to be aware of these manifestations and closely monitor them for better clinical management and outcome of patients³.

Exact pathophysiology of cardiac involvement in Dengue is not well understood. Weerakoon et al. performed autopsies on five patients who died due to dengue complications and showed histopathological evidence of myocarditis. The mechanism of myocardial damage in dengue could be the release of inflammatory mediators and/or the direct action of the virus on cardiomyocytes, as seen in acute myocarditis caused by other viruses. Carlos et al. Reported the histological findings in a fatal case of Dengue myocarditis. They observed that on necropsy the heart was dialted and flabby. Histopathological study showed marked interstitial oedema with a diffuse inflammatory infiltrate mainly composed of lymphomononuclear cells and fibroblasts. Diffuse foci of myocytolytic necrosis were seen where neutrophils could be identified in association with mononuclear cells. Electron microscopic study of the myocardium disclosed clusters of virus particles in diffuse foci of cardiomyocytes, presenting dissolution of myofilaments, and in the interstitial space².

Ing-Kit Lee et al. reported a case of fatal Dengue myocarditis and reviewed the cardiac manifestations in Dengue. The clinical manifestations of cardiac complications in dengue illness vary considerably. At one end of the clinical spectrum, patients are asymptomatic or have mild cardiac symptoms despite relative bradycardia, transient atrioventricular block, and/or ventricular arrhythmia. At the other severe end, patients may experience acute pulmonary edema and/or cardiogenic shock due to severe myocardial cell damage with left ventricular failure . Myocarditis can masquerade as acute myocardial infarction. The diagnosis of acute myocardial infarction should be made based on a rise in biochemical markers of myocardial necrosis (serum creatine kinase-MB and/or cardiac troponin I), coupled with ischemic symptoms and/or electrocardiographically developed Q waves or ST segment elevation/ depression⁴. Our patient was young and did not have chest pain or ECG changes suggestive of myocardial infarction. Hence myocarditis was considered and investigations supported it.

Cardiac rhythm disorders, such as atrioventricular blocks, ventricular ectopics including Ventricular trigeminy and atrial fibrillation have been reported during episodes of DHF. Most of them are asymptomatic and have been self limiting with spontaneous resolution. The cause for these rhythm disorders is thought to be myocarditis. Our patient had Supraventricular tachycardia. Electrocardiographic abnormalities as a result of dengue infection are said to be in the range of $3475\%^{5}$.

The Echocardiographic abnormalities in Dengue were studied by Wali et al. who showed that seven patients had ejection fraction less than 40%, 12 had global hipokinesia, and, after 3 weeks of follow up, all alterations had returned to normal⁸. S. P. Sengupta etal assessed the myocardial performance by 2 dimensional speckle tracking echocardiography and observed that Left ventricular global ejection fraction was reduced in patients with DhF at presentation. Peak longitudinal strain in patients with DhF was significantly attenuated in the subendocardial region and a significant increase in circumferential strain in the subepicardial region and a higher radial strain⁶. Our patient also showed Echocardiographic abnormality in the form of global hypokinesia and reduced ejection fraction which recovered after treatment.

Thus it is evident that cardiac involvement occurs in Dengue fever and can occur from self-limiting tachybrady arrhythmia to severe myocardial damage, leading to hypotension and pulmonary edema. In such a situation myocarditis should be suspected and confirmed by ECG changes and biochemical markers. Echo can be a very useful investigation for diagnosis and follow up. Myocarditis can be fatal also and physicians should have a high

index of suspicion for cardiac complications in patients with dengue illness especially in the presence of Thrombocytopenia. It should be managed aggressively in a critical care unit.

Figure 1 : Erythematous maculopapular rash in Dengue patient





Figure 2 : ECG showing supraventricular tachycardia in the patient

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VJIM Volume 18 January 2015 72