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Management of Two Cases of Expanded Dengue Syndrome with Myocarditis, Arrhythmia and Heart Failure

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Abstract:

Dengue infection may present as asymptomatic, dengue fever, dengue hemorrhagic fever and dengue shock syndrome. However, atypical manifestations in other organs called expanded dengue syndrome. One of the cardiac complications in dengue is myocarditis. Our two patients resented here with fever followed by chest pain, sweating and collapse of Blood pressure and their lab reports were quite similar such as low platelet, ECG change, raised Troponin I, low ejection fraction on echocardiography. They were rescued by injection intravenous Methylprednisolone therapy.

Here we present this case series to improve recognition and prevent progression to fatal outcome. In this regards healthcare providers should maintain a high degree of suspicion regarding potential cardiac complications in patients with dengue fever.

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Introduction:

Dengue fever is caused by dengue virus (DENV) and the incidence of dengue has grown drastically around the world in recent decades. A vast majority of cases are asymptomatic. So, the actual numbers of dengue cases are under-reported. Many cases are also misdiagnosed as other febrile illnesses[1].

In 2023, from 1st January to 2nd October, over 4.2 million cases have been reported from 79 countries[2]. Dengue is a disease of tropical and subtropical regions but geographically, dengue has spread to numerous new locations those were safe and previously unaffected. This is probably due to the recent increase in global trade[3].In Bangladesh, between 1st January to 1st October 2023, the Ministry of Health and Family Welfare of Bangladesh has reported a total of 2,35,204 dengue cases and among them total no of 1148 cases have died[4].

Expanded dengue syndrome is a new entity added to the classification system to incorporate a wide spectrum of unusual manifestations caused by affecting various organ systems[5].

Cardiac complications in patients with dengue illness are not uncommon. The most common complication is myocarditis. However, the study of myocarditis in dengue is still very lacking (6).

Here we will discuss two cases who developed fulminant myocarditis triggered by a dengue virus infection. We want to notify that though this presentation is very rare in the community and mortality is high but life of these patients were saved due to prompt and necessary steps.

Case 1:

A 15-year-old boy presented to us with fever, vomiting and body ache. But his NS1 was negative. On 5th day of fever, pt developed restlessness and profuse sweating followed by dropping of blood pressure. He was then admitted in ICU and at that time his BP was non-recordable with absent peripheral pulses suggesting development of Dengue Shock Syndrome. He was rapidly resuscitated with IV fluid boluses and colloid on the basis of calculated dose. But he had a pulse rate of 160/min in addition to a poorly recorded mid-arm systolic blood pressure of 90/60 mmHg. At that point he was transfused with one Unit whole blood. His extremities were cold and lower limb pulses were poorly palpable. He was confused, restless, and was in respiratory distress. Inotropic support started immediately with high flow oxygen through BiPAP to maintain O2 saturation level. Meanwhile his lab report was available and showed dengue IgM was positive,

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CBC showed WBC-3.93 K/µL, Hb-11.80g/dL, HCT level was 49%, platelet count - 10k, S creatinine 1.9mg/dl, S. Albumin- 2.96, SGOT-650 IU/, FDP->150µg/ml,APTT-50sec, PT-patient 23, control-12, INR-1.95. Infusion of 20% human Albumin, Aphaeretic platelet and Fresh frozen plasma was given. Despite all these measures, his blood pressure was unstable. Few hours later he developed chest pain, palpitation and further collapse of blood pressure. Immediate ECG showed ST segment elevation in Inf. and Ant. Leads (Fig-1). Trop-I was 6.01ng/ml, NT-proBNP-1679.80pg/mI. As the patient's condition was deteriorating Methyl prednisolone (500mg) was given IV over 20 min as a rescue medication and continued for total three days.

After that his blood pressure started to rise and stable above 100/80 mmHg, and his urine output increased to 0.8–1.4 ml/kg/h. His ECG became normal. His O2 demand started to decrease gradually. One day after, he developed atrial fibrillation (Fig-2) and it was reverted with antiarrhythmic drug Amiodarone.

After two days, he was stable and shifted to Cabin and observed for next three days. He was discharged from hospital in stable condition. (fig-3 and fig-4)

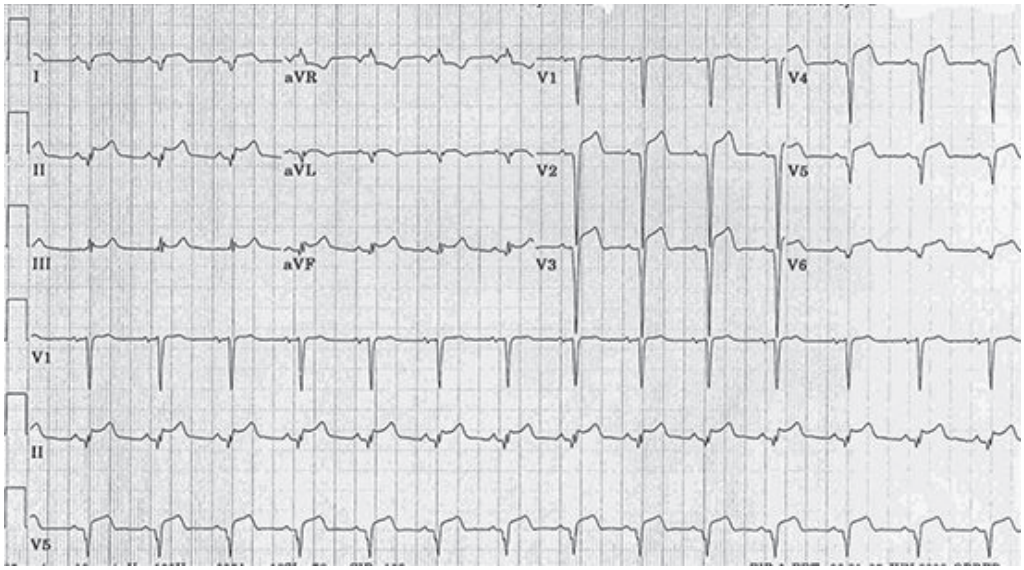

Fig.-1: *ECG showed ST elevation in II, III and AVF and V2-V6*

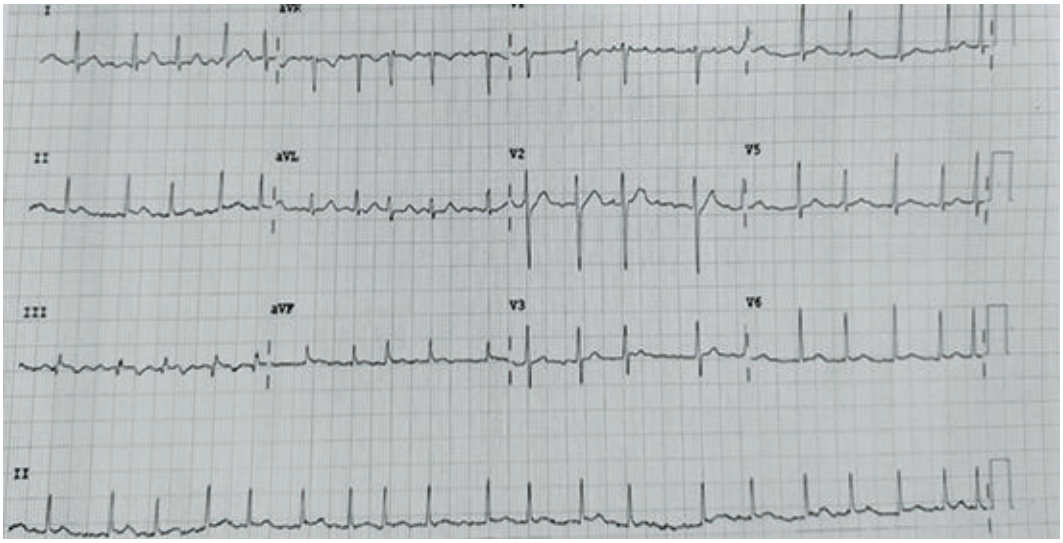

Fig.-2: *ECG shows Atrial Fibrillation*

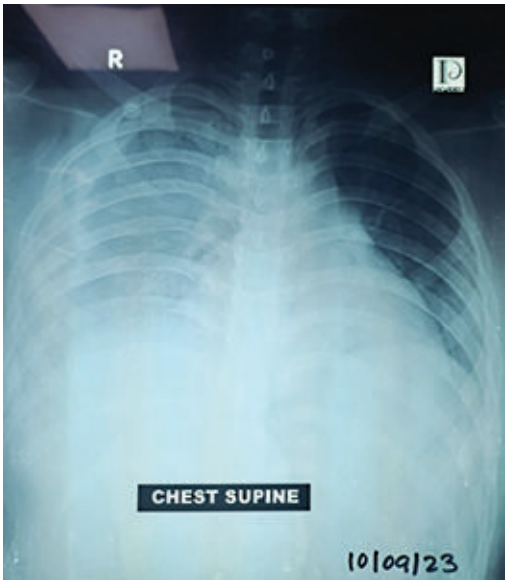

Fig-3: *X-ray chest in ICU showed right sided*

Massive pleural effusion effusion on right side.

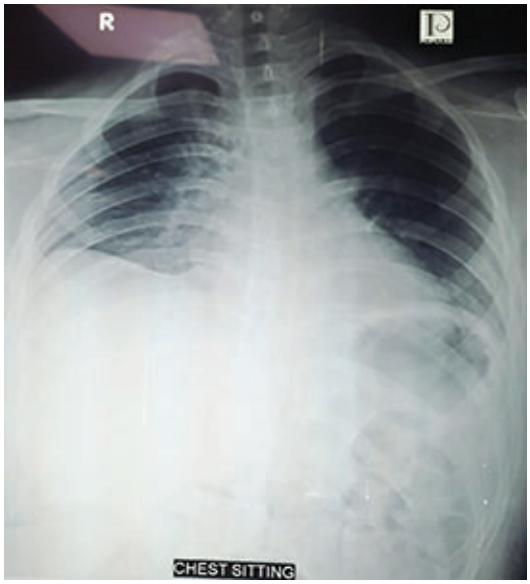

Fig.-4: *X-ray chest on discharge showed mild pleural*

Case 2:

A 26-yr-old man presented with fever for four days, vomiting, diarrhea. His NS1 was positive. He was hemodynamically stable and was on intravenous maintenance fluid therapy. On the 1st afebrile day CBC showed WBC-2.40K/µL, Hb-11.10g/dL, HCT level was 44% with low platelet-40k/µL), SGOT 127 IU/L but suddenly patient developed severe central chest pain, sweating with collapse. Blood pressure and pulse were not recordable. ECG was done immediately and showed sinus tachycardia only.(Fig:5)

He was immediately given 500ml Normal Saline bolus in 20 minutes and 300ml colloid. But his vitals were unstable. He was then shifted to ICU. In ICU his BP-90/60mm of Hg, P-140b/min, Auscultation of the lung

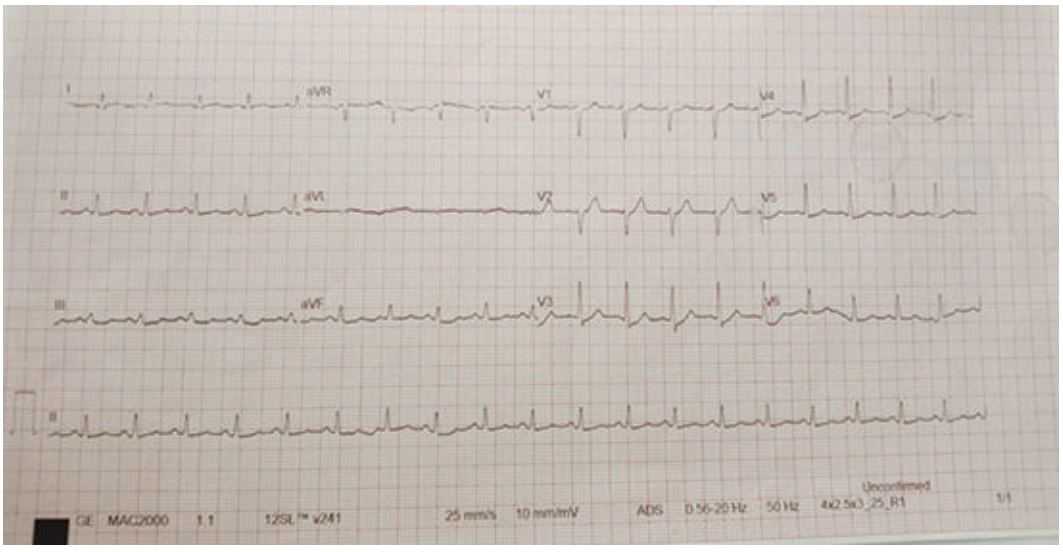

Fig.-5: *ECG: Sinus tachycardia*

showed Bilateral crepitation up to mid zone of lungs. Meanwhile following investigations were available SG0T-1170 IU/L, SGPT-312 IU/L, PT-1.23, APTT-50 sec, Trop- i was 30.01 ng/mL Creatinine- 1.68 mg/dl. Intravenous methylprednisolone 500 mg was given immediately and his condition started to improve.

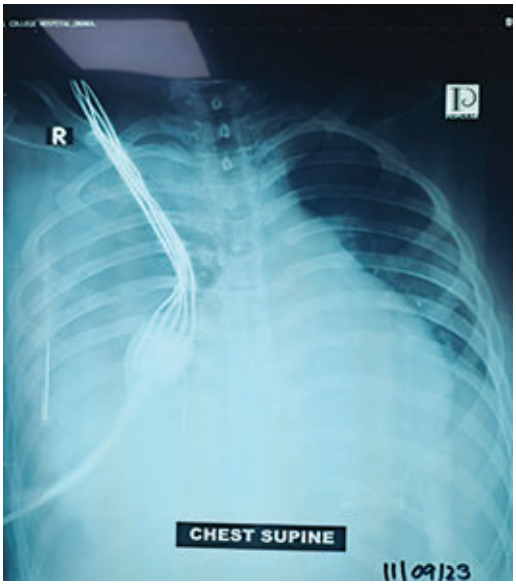

Fig.-6: *ICU*

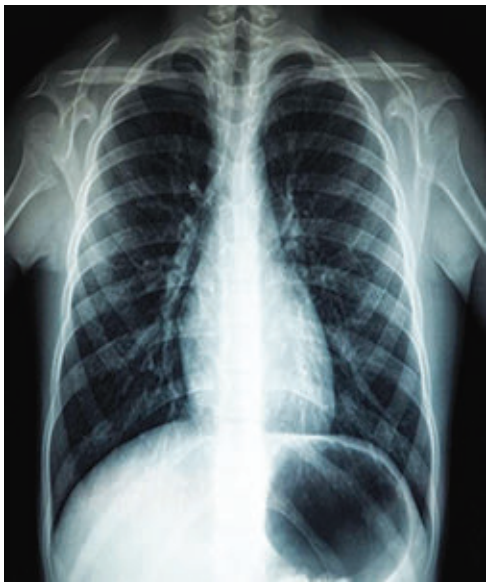

Fig.-7: *Before discharge*

But next day he developed respiratory distress, his saturation started to fall. Lab investigations showed pro-BNP 11668.60 pg/mL, Chest X-ray showed huge right sided massive pleural effusion. High flow nasal cannula with BiPAP was started but could not decrease the level of Co2 retention and he was intubated. No inotropes were required to manage the patient. As his blood pressure was stable, diuretics was given along with other medication. Echo-cardiography showed dilated LA and LV with EF 40%. Second sample of Troponin-I came down to 11.40 ng/ml. On day 3 echocardiography showed EF improved to 60%. He was extubated 48 hrs. later and after stabilization shifted to cabin from ICU. In the cabin follow up all parameters of organ damage improved which includes Troponin-I, SGOT, Creatinine. The patient was discharged with stable and good condition

Discussion:

In this case series both patients with dengue developed severe hemodynamic compromise. However, our both patients had evidence of myocarditis. So, we had to be cautious not to overload the patient with fluid to prevent myocardial strain and also possible pulmonary edema during the recovery phase. We administered intravenous methyl prednisolone as a rescue measure. This led to a dramatic recovery with rapid defervescence, hemodynamic stability, reversal of ECG changes, and improvement in urine output. The clinical predicament of these patient was most likely due to DSS with myocarditis.

There are very few data regarding dengue induced myocarditis but in one study which was conducted in India suggested that one in 206 dengue patients had cardiac symptoms attributed to myocarditis[7].

Mortality among the patients with dengue fever is due to severe DSS with or without severe organ involvement (myocarditis, hepatitis, and encephalitis). An improving fluid resuscitation alone may not be sufficient to reduce mortality[8]. In our observation, the apparent beneficial effects of methyl prednisolone in these 2 cases was game changer.

In case of dengue myocarditis, inflammatory process may affect the myocytes, the vascular structures, the conduction system, the autonomic nerves, and the interstitium, and they can also affect pericardium by contiguity[9]

Dengue myocarditis can manifest from silent forms to symptoms of chest pain, dyspnea, heart failure,

pulmonary edema, or cardiogenic shock. It can even imitate acute myocardial infarction as in our case. They can also present in the form of arrhythmias including sinus tachycardia, ventricular arrhythmias, supraventricular arrhythmias such as atrial fibrillation, and/or varying degrees of atrioventricular block can be detected [9].

Acute fulminant myocarditis is a life-threatening emergency. Criteria to diagnose fulminant myocarditis include a) Rapid onset of symptoms of heart failure within two weeks of an inciting event, b) Prodromal symptoms of upper respiratory or gastrointestinal viral infection, c) Rapid hemodynamic instability requiring a large dose of inotropes, d) Cardiac magnetic resonance (CMR) or endomyocardial biopsy (EMB) proven myocarditis, and e) Exclusion of other cardiac diseases like coronary artery disease [10].

Patients with fulminant myocarditis are at increased risk for adverse cardiac events. Inotropic agents are the first-line of treatment to enhance cardiac pumping function. In patients not responding to inotropic agent mechanical circulatory devices like intra-aortic balloon pumps (IABP) and extracorporeal membrane oxygenation (ECMO) can be used. Methylprednisolone is also a lifesaving drug. Patients with heart failure due to myocarditis who are refractory to inotropic agent/IABP/ECMO, cardiac transplantation can be considered.[11]

Prompt triage and a high index of suspicion are vital to provide aggressive treatment. Recognition of patients with fulminant presentation is potentially lifesaving as more than half can survive without sequelae.

Conclusion:

We present these cases for its rare entity and fulminant presentation which improved due to timely diagnosis and prompt management.

Fulminant myocarditis is one of the rare entities of myocarditis which is associated with a rapid downhill course if not recognized early. With an increasing incidence of dengue fever, newer treatment strategies are needed for the management of fulminant myocarditis and improvement of the outcome.

Conflict of interest:

None of the authors have any conflicts of interest to declare.

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