



A Rare Case of Dengue Encephalitis

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Abstract

The clinical spectrum of dengue fever ranges from asymptomatic infection to dengue shock syndrome. Although dengue is non-neurotropic virus, there is increasing evidence for dengue viral neurotropism suggesting there may be an element of direct viral encephalitis. Encephalopathy has been thought to result from the multisystem derangement that occurs in severe dengue infection, with liver failure, shock and coagulopathy causing a cerebral insult. Dengue encephalitis is a rare disease. We report an interesting case of dengue encephalitis from Southern India. A 55 year male presented with headache, vomiting and altered sensorium. Dengue IgG/ IgM was positive. RTPCR for COVID was negative. Dengue encephalitis should be considered for differential diagnosis of fever with altered sensorium, especially in countries like India where dengue is rampant.

Keywords: Dengue fever, dengue encephalitis, neurological manifestations.

Introduction

The clinical spectrum of dengue fever ranges from asymptomatic infection to dengue shock syndrome. However neurological complications are unusual. Dengue encephalopathy is not an unknown entity. However Dengue encephalitis a direct neuronal infiltration by the dengue virus is an extremely rare disease. Although dengue is nonneurotropic virus, there is increasing evidence for dengue viral neurotropism suggesting there may be an element of direct viral encephalitis ⁽¹⁾. The spectrum of neurological manifestations seen in dengue has been classified by Murthy into 3 categories, related to neurotropic effect of the virus like encephalitis, meningitis, myositis and myelitis, due to the systemic complications of infection like encephalopathy, stroke and hypokalemic paralysis and post-infectious

complications like encephalomyelitis, optic neuritis and Guillain Barré syndrome ⁽²⁾. Dengue is endemic in our part of Karnataka. This presents rare neurologic manifestation of dengue.

Case Report

55 year male brought to emergency department with history of headache since 4 days, vomiting since 2 days (2 episodes), myalgia since 2 days, altered sensorium since 1 day. No history of fever, rash, seizure. On examination patient was irritable, pulse 80 bpm, blood pressure of 140/90 mm of Hg, SPO2 of 98% at room air, Cardiovascular, respiratory and abdominal examinations were normal. Neurological examination revealed irritability and mild neck stiffness, Pupils bilaterally equal and reactive to light. Possibility of Neuroinfection was

considered and started with Inj. Mannitol 100 ml IV, Inj. Ceftriaxone 2 gm IV, Inj. Pantaprazole 40 mg IV, Inj. Emeset 4 mg IV and relevant investigations were sent with admission in MICU. Investigations revealed Hb- 14.7 gm/ dl, Platelet- 18000/ cu.mm, TLC- 9100 cells/ cu.mm, RBS- 125 mg/ dl, B. urea- 50 mg/ dl, S. creat- 1.3 mg/ dl, T. Protein- 6.4 gm/ dl, Albumin- 3.8 gm/dl , Globulin- 2.6 gm/ dl, T. Bilirubin- 0.9 mg/dl, D. Bilirubin- 0.4 mg/dl, I. Bilirubin- 0.5 mg/ dl, AST- 83 IU/L, ALT- 79 IU/L, ALP- 168 IU/L, S. Electrolytes- Na- 134mmol/L, K- 4.5 mmol/L, Cl- 104 mmol/L, Dengue IgG/IgM – Positive, Malaria test- negative, HIV, HBsAg, HCV- Non reactive, Urine routine- blood present, ketone bodies present, ECG- normal, X ray chest - normal, NCCT brain- Normal study, USG abdomen- mild splenomegaly, RTPCR for COVID- Negative. As there was severe thrombocytopenia with positive dengue serology, 4 units of platelets were transfused and cap doxycycline 100 mg was added. As patient became unconscious and GCS was 9/15, patient was given supplemental oxygen, lumbar puncture was done ,CSF analysis showed Clear fluid, 2 cells/ cu mm, all Lymphocytes , sugar- 95 mg , protein- 106 mg, culture yield no growth, ADA- 0.045. Considering viral encephalitis patient was started with Inj. Dexona 8 mg IV, Inj. Acyclovir 500 mg IV. Patient clinically improved with regaining conscious in 24 hours. Platelet count monitoring done twice a day which showed 35000 and 60000 on first day, 80000 on second, 95000 on third, 133000 on fourth and 170000 on fifth day.

Patient was clinically stable and discharged after 10 days, with no focal neurological deficits.

Discussion

Dengue encephalopathy is a rare entity, with the incidence ranging from 0.5% to 6.2%⁽¹⁾. Encephalopathy has been thought to result from the multisystem derangement that occurs in severe dengue infection, with liver failure, shock and coagulopathy causing a cerebral insult. The criteria for dengue encephalitis are fever, acute

signs of cerebral involvement, presence of anti-dengue IgM antibodies or dengue genomic material in the serum and/or cerebrospinal fluid, exclusion of other causes of viral encephalitis and encephalopathy⁽³⁾. Varathraj also has defined criteria for dengue encephalitis⁽⁴⁾. Dengue has classically been thought to be not neurotropic. However there is increasing evidence regarding direct dengue viral neurotropism. Cases of dengue encephalitis has been reported by Solomon et al. and recently by Borawake et al. and Kutiyal AS et al from India^(5,6,7). The main symptoms of dengue encephalitis are headache, seizures and altered consciousness⁽⁴⁾. Typical symptoms of dengue fever like myalgias, rash and bleeding are seen in less than 50% of encephalitis cases⁽⁶⁾. So Solomon et al. have suggested that dengue should be considered in all encephalitic patients in endemic areas, regardless of the presence or absence of classical features. Our patient did not have classical features of dengue like rashes hypotension.

Conclusion

Dengue encephalitis should be considered in the differential diagnosis of fever with altered sensorium, especially in countries like India where dengue is rampant⁽⁸⁾. Physicians must have a high index of suspicion or else this uncommon manifestation of a common disease can be easily missed.

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