



A RARE CASE OF ISOLATED DENGUE ENCEPHALITIS

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ABSTRACT

Dengue is one of the leading causes of viral infections in tropical countries like India. Encephalitis associated with dengue infection is usually part of multi-system involvement. The isolated dengue encephalitis without multi-system involvement is a rare complication and it is associated with high morbidity and mortality. This case is about 25 years old female admitted with fever, generalized myalgia, and decrease level of consciousness since 3 days. Dengue NS1 Antigen and Ig M antibody were positive. MRI Brain was suggestive of hyperintensities in bilateral thalamus with tonsillar herniation. In the medical ICU, even with continued mechanical ventilation, anti cerebral edema measures, antiepileptics and other supportive care patients, neurological status progressively deteriorated and died after 24 hours of admission. This case report highlights about a rare case of isolated dengue infection.

Key words:Dengue, Encephalitis, Flavivirus, Mortality, Hemorrhage.

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INTRODUCTION

Dengue infection is caused by flavi virus of flaviviridae family. It is a single stranded enveloped RNA virus. Dengue infection is endemic to over 100 countries; four out of every 10 people in the world are at risk of infection [1]. It is caused by bite of AedesAegypti mosquito which is found in abundance at 35 degree north to 35 degree south latitude. Dengue encephalitis has been associated with high morbidity and mortality [2]. Neurological features are secondary to encephalopathy (cerebral edema, hemorrhage, hyponatremia, hepatic and renal failure, anoxia, microcapillary hemorrhage and release of toxic products) or neurotropism [3].

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CASE REPORT

A 25 year old female, with no prior comorbidities presented to our hospital with fever, generalized myalgia and headache since 3 days and decrease in conscious level with one episode of seizure since 1 day. There was no history of cough, abdominal pain, shortness of breath, nausea, vomiting rashes and bleeding tendencies. The patient was initially evaluated at an outside hospital where she received antibiotics, analgesics and other supportive care. But as her symptoms worsened she was referred to our hospital for further care.

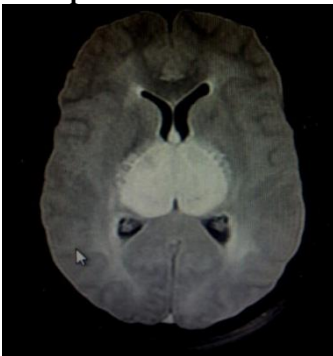
On examination patient was febrile with a temperature of 100* degree, heart rate 126/min, blood pressure 90/60 mm Hg, SPO2 92% on room air and respiratory rate 32/min. On examination patient was drowsy with mild neck stiffness, Glasgow coma scale (GCS) E1V1M2, Pupils dilated with sluggish reaction to light reflex. Other systemic examination was unremarkable.

Laboratory investigations showed total leucocyte count (TLC) 7000, platelet count of 98000, hemoglobin 14.4 gm/dl, serum creatinine 1.8, blood Urea 46, sodium 128 meq/l, potassium 4.0 meq/l. total bilirubin 1.8 (direct 1.1, indirect 0.7) Albumin 2.1, Globulin 2.4, AST 62, ALT 78, GGT 67, Random blood Sugar 97mg/dl. ABG: Ph 7.42, PCO2 15, PO2 142, HCO3 13.5, BE -8. Blood for malaria parasite negative, Leptospira Ig M negative, Dengue NS1 Antigen and Ig M antibody were positive.

She was started on intravenous fluids in view of hypotension, intubated due to low GCS and mechanically ventilated. A provisional diagnosis of meningo-encephalitis /encephalopathy was considered and the patient was started on empiric antibiotics, Acyclovir and

other supportive care. MRI Brain was done which was suggestive of hyperintensities in bilateral thalamus, lower pons, cerebellar parenchyma and periventricular fronto-parietal white matter with tonsillar herniation (Figure-1). In the medical ICU she was mechanically ventilated continued on anti cerebral edema measures, antiepileptics and other supportive care. Two hours later her neurological status deteriorated where she was deeply comatose with no motor response to painful stimuli, absent pupillary light reflex, fixed and dilated pupils, absent doll's eye response, absent gag reflex and cough reflex. Apnoea test was positive. Later patient had bradycardia and cardiac arrest where she could not be resuscitated and declared dead.

Fig 1. Periventricular fronto-parietal white matter with tonsillar herniation.



DISCUSSION

The dengue infection clinical spectrum can vary from simple febrile illness to hemorrhagic fever and dengue shock syndrome [4]. Four serotypes of dengue namely DEN 1 to 4 has been identified. Infection with any particular serotype provides lifelong immunity; and subsequent infection with other serotype causes severe dengue hemorrhagic fever due to antibody dependent enhancement phenomenon. In 1976, Sanguanserm Sri et al first described about neurological association with dengue infection. Neurological manifestation can occur between two and 30 days after fever onset. Age group may range from few months to 79 years. Neurological complication commonly reported with DEN-2 and DEN-3 serotypes.

The manifestation which are reported include encephalopathy, encephalitis, GuillainBarre Syndrome [5], transverse myelitis, acute disseminated encephalomyelitis [6], myositis [7], neuromyelitisoptica and optic neuritis. The central nervous system (CNS) manifestations are secondary to systemic manifestation (metabolic disturbances, hemorrhagic disturbances), Neurotropism (direct viral infection of CNS) and post infection sequelae (autoimmune reaction) [8,9]. Symptoms include headache, irritability, insomnia, seizures, altered level of consciousness, focal neurological deficit, and papilledema.

Diagnostic criteria has been mentioned [10] which includes fever, signs of cerebral involvement (altered

conscious level, seizures), presence of NS₁ antigen / Ig M antibodies / PCR in serum or CSF and exclusion of other causes of viral encephalitis. Diagnosis is by virus isolation in cell culture (Gold standard). NS₁ antigen detection has low sensitivity (52-66%) but high specificity (90-100%) [11]. The sensitivity can be increased up to 93-100 using reverse transcription polymerase chain reaction (RT-PCR). Serum IgM antibody (ELISA) detection has 92% sensitivity and 99% specificity. Up to 50% of the patients can have normal CSF.

The absence of antibodies in CSF will not rule out encephalitis [12,13]. Other flavi viral infections such as St. Louis encephalitis, Japanese encephalitis, West Nile fever and yellow fever can interfere with serological diagnosis of dengue fever. Hyperintense lesions are found in global pallidus, temporal lobe, thalamus, hippocampus, pons and spinal cord. Treatment is usually symptomatic and supportive. Glucocorticoids can be used for treatment of dengue encephalitis [14].

This case report mainly highlights about rare case of isolated dengue encephalitis. Our patient presented with mainly encephalitis features without any other organ involvement.

STATEMENT OF HUMAN AND ANIMAL RIGHTS

All procedures performed in human participants were in accordance with the ethical standards of the

institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors

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DECLARATION OF INTEREST

None declared.

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