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# Acute Hemorrhagic Adem - An Unusual Presentation of Dengue Treated with Plasmapheresis

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## ABSTRACT

Neurological manifestations after dengue are not very common& acute disseminated encephalomyelitis (ADEM) following dengue infections is still more infrequent. We report a male, 10 year old who developed Hemorhhagic ADEM after 7-8 days of dengue illness. The disease was confirmed by detection of dengue IgM antibodies in serum & absence of dengue PCR & IgM in CSF. MRI brain showed hemorrhagic encephalitis involving bilateral thalami, posterior limbs of internal capsule, adjacent lenticular nuclei, left hippocampus, right temporoparietal lobes, right periventricular white matter, midbrain, pons, medulla & bilateral cerebellar peduncles. Csf analysis showed 03cells/ protein 73 mg/dl. Patient was treated with cerebral decongestants, antiepileptics & iv methylprednisolone (MPS) for 5 days. In view of slow clinical recovery after 3 doses of iv MPS, 2nd line immunotherapy – plasmapheresis was initiated & patient was treated with 4 cycles of plasmapheresis. Patient responded on plasmapheresis, was extubated, GCS improved to 9-10/15, residual sequale of mutism, tendoachilles contracture & poor cognition were seen. Therapeutic intervention with plasmapheresis resulted in rapid recovery.

Keywords: Acute disseminated encephalomyelitis, dengue, magnetic resonance imaging, plasmapheresis.

### **CASE REPORT**

A 10 year old boy brought from outside hospital with diagnosis of severe dengue, respiratory failure & encepahlopathy. Patient had altered liver enzymes, positive dengue serology (NS-1+) & CTnoncontrast showed hypodense areas in the brainstem. Clinically patient had bradycardia, normal hemodynamics, petechie over body, GCS 7/15, Posturing, hypertonia, hyperreflexia & extensor plantar. MRI brain showed hemorrhagic encephalitis involving bilateral thalami, posterior limbs of internal capsule, adjacent lenticular nuclei, left hippocampus, right temporoparietal lobes, right periventricular white matter, midbrain, pons, medulla & bilateral cerebellar peduncles. Repeat Dengue serology showed dengue IgG &M as positive. Csf analysis showed 03cells/ protein 73 & dengue CSFPCR & IgM positive. Patient was treated with cerebral decongestant, antiepileptics & iv methylprednisolone (MPS) for 5 days. In view of no clinical recovery after 3 doses of iv MPS, 2<sup>nd</sup> line immunotherapy – plasmapheresis was initiated & patient treated with 4 cycles of plasmapheresis. Patient responded on plasmapheresis, was extubated, GCS improved to 9-10/15, residual sequale of mutism, tendoachilles contracture & poor cognition were seen.

## DISCUSSSION

ADEM is an immune-mediated disease of the brain. The molecular mimicry is responsible for immunogenic injury. ADEM has an abrupt onset and a monophasic course. Symptoms usually begin 1-3 weeks after the precipitating eventinfection or vaccination. The index patient developed ADEM on the 7<sup>th</sup>- 8<sup>th</sup> day after development of clinical manifestations of dengue. Therapy consists of use of high-dose steroids, plasma exchange, and intravenous immunoglobulin with usually favorable results and leaving little functional deficit. However, recovery may be delayed and incomplete even after timely institution of treatment. The index patient was treated with high-dose methylprednisolone and plasmapheresis recovered well with impaired cognition, mutism & tendoachilles contracture.

#### CONCLUSION

In countries like India where dengue is common, development of ADEM as its complication must be considered in appropriate clinical setting. With prompt recognition and timely institution of treatment, ADEM can resolve with little residual sequalae; but any delay in management usually leads to severe neurological neurodeficit and can even be life-threatening

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