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**Case Report**

# Posterior reversible encephalopathy syndrome in dengue: a rare manifestation

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## ABSTRACT

Dengue is an important arthropod born disease with its cases spiking every 2-3 years and spectrum of disease ranging from mild febrile illness to severe illness with multiple systemic complications including rare neurological manifestation. We hereby presented a case report of rare presentation of posterior reversible encephalopathy syndrome (PRES) in an eight year old child suffering from dengue.

**Keywords:** Dengue, *Aedes aegypti*, *Aedes albopictus*

## INTRODUCTION

Dengue is a febrile illness caused by infection with one of four dengue viruses (DENV) transmitted by *A. aegypti* or *A. albopictus* mosquitoes during the taking of a blood meal. Given estimates of 390 million infections worldwide each year and over 2.5 billion individuals at risk for infection, the DENVs remain important arthropod-borne viruses from a medical and public health perspective.[1] PRES can occur as a neurological complication of dengue but is rare.[2]

PRES usually occurs in the setting of hypertensive emergencies, characterized by bilateral increase in T2 signal intensity in the white matter on MRI usually concentrated in the posterior part of the hemispheres. Findings are of white matter edema which normalizes over several weeks.[3]

## CASE REPORT

An eight-year-old female presented to Adesh Institute of Medical Sciences in casualty with history of fever for 5-6 days with complaints of headache and 2 episodes of vomiting and vacant stare for the last 1 hour, with 1 episode of tonic positioning.

On presentation, her vitals were heart rate of 118 bpm, blood pressure 80/40mm of Hg, temperature 98.1 ºF and saturation of 88% on room air. She had reduced pulse volume with normal capillary refilling time. Systemic examination was otherwise unremarkable. Patient was thoroughly evaluated and injection midazolam 2 ml IV stat was given and loading with injection levetiracetam was given and fluid boluses were given and patient was provided oxygen support at 8 lt/min via Hudson mask. Simultaneously routine investigations including CBC, LFT, RFT, electrolytes, CRP and PT/INR were sent and an urgent CEMRI was planned.

Full blood count analysis revealed a hemoglobin level of 16.2 g/dl, white cell count of 8.6×109 /l and platelet count of 120×109 /l. His renal profile was normal, with raised liver enzymes alanine aminotransferase 198 u/l and aspartate aminotransferase 189 u/l with CRP of 4.0 g/l. Patient was started on ionotropic support and antibiotic coverage and 2FFP were transfused in view of deranged INR. Over the course of hospital stay patient's condition improved, inotropes were tapered off and patient was discharged in stable condition.

CEMRI brain revealed T2/FLAIR hyperintensities in subcortical regions of bilateral parieto-occipital lobes (Figure 1).

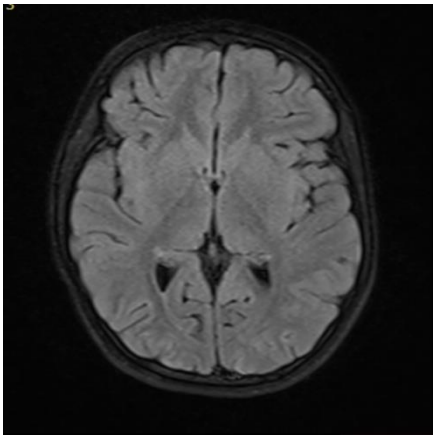

**Figure 1: CEMRI brain revealed T2/FLAIR hyperintensities in subcortical regions of bilateral parieto-occipital lobes.**

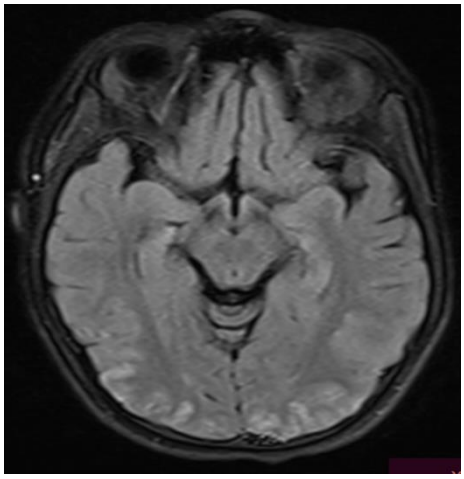

**Figure 2: No enhancement was seen on post contrast TIWI.**

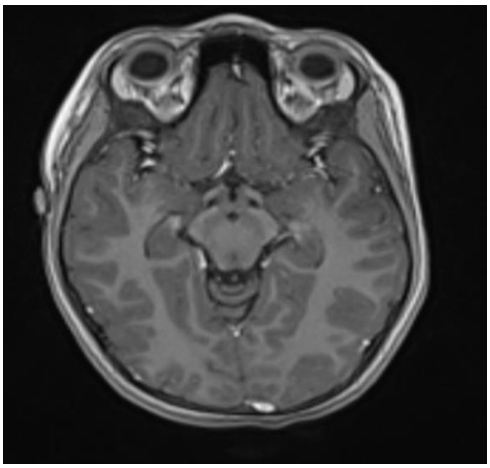

**Figure 3: Mild enhancement was seen on post contrast FLAIR.**

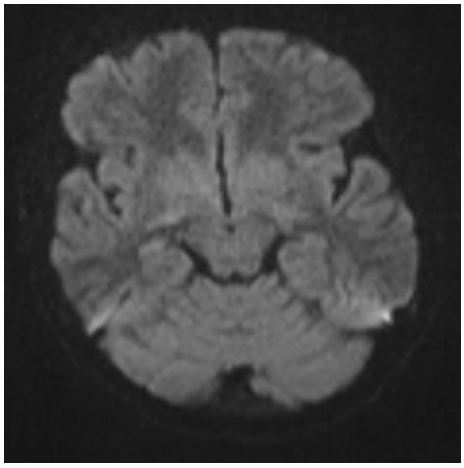

**Figure 4: No diffusion restriction was seen in these regions.**

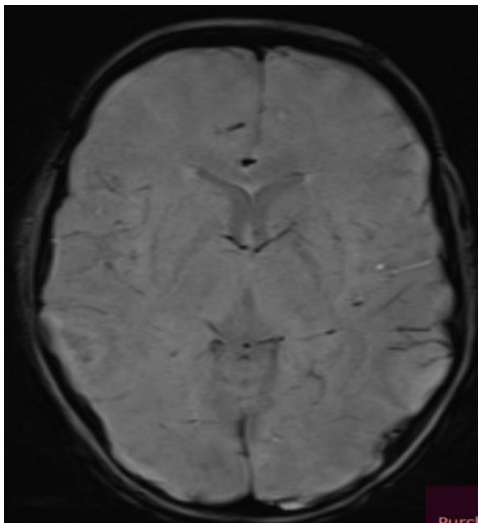

**Figure 5: No susceptibility was seen on SWI.**

No enhancement was seen on post contrast TIWI (Figure 2). However, mild enhancement was seen on post contrast FLAIR (Figure 3). No diffusion restriction was seen in these regions (Figure 4). No susceptibility was seen on SWI (Figure 5). Diagnosis was given as PRES.

## DISCUSSION

Clinical features of dengue may range from those of a mild febrile illness to severe dengue with systemic complications. Systemic complications included shock, bleeding, plasma leakage, myocarditis and in some patients, neurological complications. The latter included dengue encephalitis, dengue encephalopathy, meningitis, acute disseminated encephalomyelitis and Guillain-Barre syndrome.[2]

Pathogenesis of PRES was not fully understood but endothelial dysfunction and failure of cerebral auto regulation played a key role.[4,5] The brain edema was the result of active exocytosis of water rather than simply a

passive leak from vessels subjected to high pressures. CSF pressure and protein may be elevated to more than 100 mg/dl without any cellular reaction. Strongly associated conditions like hypertensive emergency, systemic lupus erythematosus, chronic kidney disease, immunosuppressive therapy, use of chemotherapeutic agents like tacrolimus, cyclosporine, vincristine and interferon alpha were all excluded in our patient.[6,7]

Characteristic radiographic findings of PRES included bilateral white matter changes in areas supplied by the posterior circulation but can be diffuse as described in this case and resolved over weeks. High CSF protein levels correlated with cerebral edema and disease severity.[8]

Differentiating PRES from dengue encephalitis and ADEM from PRES in a children with dengue was very important as treatment of ADEM required potentially toxic therapy such as steroids whereas PRES recovered with supportive management only.[9]

## CONCLUSION

In children with severe dengue associated encephalopathy, PRES should be considered as a differential diagnosis, especially in those with hypertension. Neuroimaging is warranted in such cases as PRES can be identified by its characteristic findings on MRI as well as for reassurance of better prognosis, in contrast to those with dengue encephalitis. Early diagnosis with CEMRI can help in early treatment and better prognosis.

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