Journal of Bangladesh College of Physicians and Surgeons
Vol. 41, Dengue Fever Supplement Issue, October 2023

Dengue-Associated Posterior Reversible Encephalopathy Syndrome Occurring in a Woman with 32 Weeks Pregnancy: A Case Report from Bangladesh

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Abstract:

Posterior reversible encephalopathy syndrome (PRES) is an increasingly well recognised reversible clinicoradiological condition. Dengue can cause various neurologic complications like encephalitis, myelitis, Guillain-Barré syndrome (GBS), and myositis, which are increasingly being recognized in addition to the more common manifestations of plasma leakage and coagulopathy. Posterior reversible encephalopathy syndrome (PRES) is rare in dengue, although the pathophysiology of endothelial dysfunction likely underlies both. Here we report a case of dengue-associated posterior reversible encephalopathy syndrome in a pregnant woman.

(J Bangladesh Coll Phys Surg 2023; 41: 99-100)
DOI: https://doi.org/10.3329/jbcps.v41i40.69731

Case report

A 28-year-old, 32 weeks primigravid woman, was admitted in a hospital of Dhaka city of Bangladesh with fever for 2 days and generalized bodyache. She had no remarkable medical history; she did not take any medications other than iron and calcium supplements and had not received any recent vaccinations. Initial hematology and biochemistry tests were normal except mild anemia (Hemoglobin 9.6 g/dl), but a rapid test for dengue nonstructural protein 1 antigen (NS1 Ag) was positive. She remained hemodynamically stable and did not experience any bleeding or have evidence of plasma leakage. No history of hypertension was found as per medical records. No other significant history was available. On Day 3 of afebrile period, she was discharged from hospital with normal vitals, platelet count was 96×10^9/L, PCV was 29.6%.

Day 5 of afebrile period, she was again brought to the emergency room with the history of two episodes of generalized tonic–clonic seizures, urinary incontinence, tongue bite and altered sensorium since 4 hours. She had a headache prior to the onset of seizures.

Because of low oxygen saturation resulting from compromised airway with minimal aspiration due to impaired consciousness (Glasgow Coma Scale score of E2V2M3), she was intubated in ER and brought to intensive care unit and put on mechanical ventilation. Heart rate was 90/min, blood pressure 125/75 mmHg, oxygen saturation was 95-96% with FiO_2 0.3, temperature 98^0F, pupil equal and reacting to light.

Laboratory examination revealed hemoglobin 10.6 g/dl, normal WBC 5. $\times10^9$/L, platelet count was 120×10^9/L and mildly elevated C-reactive protein 17 mg/dl (<5 mg/dl), SGOT 935 (<40), SGPT 319, and renal function tests were normal. Other blood tests, coagulation profile, autoantibodies were normal. Urine microalbumin was 58.9 mg/gm (<20).

Magnetic resonance imaging of the brain showed symmetrical parieto-occipital, fronto-parietal subcortical white matter hyperintensities in both brain hemispheres, and suggestive of posterior reversible encephalopathy syndrome (PRES) [Fig 1A]. A diagnostic lumbar puncture revealed clear color with normal opening pressure, WBC 3/mm^3 (N33%, L67%), sugar 40 mg/dl, protein 20 mg/dl. The differential diagnoses included eclampsia, encephalitis, or acute demyelinating encephalomyelitis (ADEM).

In ICU she was managed with intravenous fluid, empirical IV Ceftriaxone, intravenous levetiracetam, IV midazolam, and IV dexamethasone 5 mg 8 hourly for 3 days with other supportive treatment. In the next day of ICU admission, she improved symptomatically in the form of normal sensorium and normal vitals. She was extubated and stepped down. On day 5 of readmission, she was discharged. Follow up MRI brain at 17 days showed complete resolution of white matter oedema (Fig 1B).

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Received: 15 Oct., 2023 **Accepted:** 19 Oct., 2023

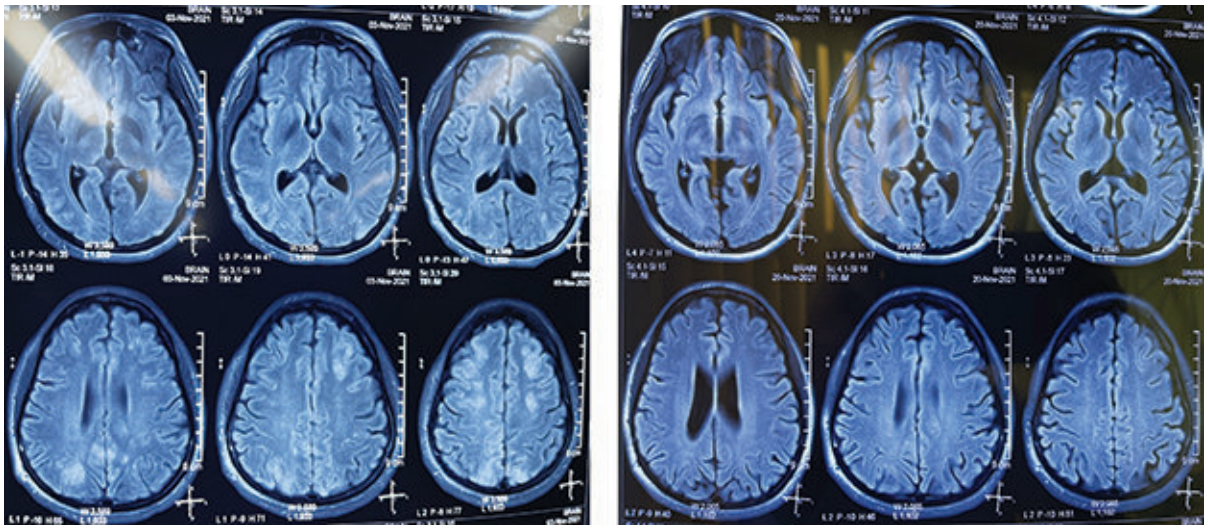

Fig.-1: *Fluid-attenuated inversion recovery (FLAIR) magnetic resonance images of the brain of a 28-year-old woman with dengue-associated posterior reversible encephalopathy syndrome A) symmetrical parieto-occipital, fronto-parietal subcortical white matter hyperintensities in both brain hemispheres B) Almost complete resolution of abnormal findings 17 days later.*

Discussion:

The diagnosis did not fit with eclampsia because of absence of hypertension and significant proteinuria; not even fit with dengue encephalitis because of a lack of CSF pleocytosis and the normal protein levels. MRI which showed early reversible white matter changes rather than delayed multifocal discrete lesions associated with ADEM, were diagnostic of dengue-associated posterior encephalopathy syndrome (PRES)[1].

PRES is an acute neurologic syndrome, typically seen in patients with blood pressure fluctuations or metabolic derangement[1]. The pathophysiology of PRES is thought to involve disruption to cerebral blood flow autoregulation, endothelial dysfunction, and vasogenic edema[1].

The predominant involvement of the posterior circulation territories is thought to result from the relatively sparse sympathetic innervation of vertebrobasilar circulation. Another hypothesis is one of the endothelial dysfunctions secondary to circulating toxins originating from immunosuppressant therapy, sepsis, autoimmune disease and pre-eclampsia/ eclampsia[2].

Though wide spectrum of neurological features is described in dengue, PRES has been suspected in only few cases[3,4]; possibly because of underreporting due to limited access to neuroimaging services in dengue endemic areas. In literature search, dengue associated PRES in pregnancy is not reported before.

This case emphasizes the importance of considering PRES in a dengue patient present with neurologic symptoms and PRES should be distinguished from encephalitis and ADEM, and from eclampsia in the last trimester of pregnancy.

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