Case Report

Posterior Reversible Leukoencephalopathy Syndrome (PRES) in a patient with post eclampsia with status epilepticus.

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ABSTRACT:

Despite the limited number of instances and relatively low knowledge of Posterior Reversible Leukoencephalopathy Syndrome (PRES) among the population of Bangladesh, it is imperative to recognize the gravity of this health issue. The crucial aspect in achieving favorable results is the early detection of the condition, as it allows for the prompt elimination of the precipitating cause and the effective management of blood pressure. The present case study elucidates the significant discoveries pertaining to a patient with posterior reversible encephalopathy syndrome (PRES), as well as the diagnostic and therapeutic strategies used. A female individual who had just undergone a surgical operation known as a cesarean section exhibited symptoms including headache, recurrent convulsions, and loss of consciousness. Subsequent medical evaluation led to the diagnosis of posterior reversible encephalopathy syndrome (PRES). This section presents an examination of the diagnostic process using clinical and radiological characteristics, as well as an overview of the therapy plan. The fast diagnosis and timely beginning of therapy procedures have been identified as crucial factors for achieving early recovery and favorable functional outcomes.

Introduction:

Posterior reversible leukoencephalopathy syndrome (PRES) is a clinicoradiological syndrome, first described by Hinchey et al. in 19961. The most common clinical manifestations are headache, altered alertness arising from drowsiness to stupor, seizures, vomiting, mental abnormalities including confusion and diminished spontaneity and speech, and abnormalities of visual perception1,2. Common risk factors associated with PRES include abrupt elevations of blood pressure, impaired renal function, preeclampsia/eclampsia, autoimmune diseases, infection, transplantation, and chemotherapeutic agents2. Diagnosis of PRES relies on history, clinical examination, and radiologic findings of symmetric bilateral hyper-intensities on T2-weighted magnetic resonance imaging (MRIs) representing vasogenic edema1. Early recognition is the key as timely removal of the precipitating factor and control of blood pressure is important to achieve favorable outcomes2.

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Case presentation:

A 23-year-old female, housewife hailing from Narayangonj, presented with repeated convulsions and an altered level of consciousness to the emergency room. She had a history of cesarean section four days back and was discharged with advice. However, she developed severe headaches followed by repeated convulsions for 3 hours on the day of discharge. She was immediately brought to the emergency where she was found to be repeatedly convulsing for 2-3 minutes without any responsiveness.
She had a tongue bite but no urinary and bowel incontinence. Her eyes roll up with a vacant look. She is non-responsive to any vocal commands. She is normotensive and her pregnancy period was uneventful. She is a nonsmoker and non-alcoholic. She had a history of taking oral contraceptive pills. She had no family history of hypertension. At the presentation, she was unconscious, GCS 3. She had an elevated blood pressure of 180/120 mm Hg; his heart rate was 87 beats per minute, and his SpO2 was 93% on room air. She was initially managed with intravenous diazepam 10 mg followed by another dose. As the convulsion was not controlled she was shifted to the ICU where she was managed by loading phenytoin and Levetiracetam. Significant lab findings in a CT scan of the brain revealed ill-defined hypoattenuation involving the cortex and subcortical white matter of both parietal and occipital lobes. A few tiny hyperdense foci are evident in the right parietal lobe features consistent with posterior reversible leukoencephalopathy syndrome. All other hematological and biochemical parameters were within normal limits. Furthermore, more MRI of the brain revealed extensive bilateral symmetrical T2W-FLAIR hyperintensity in both parieto-occipital and both frontal regions confirming the PRES diagnosis. She was managed in the ICU with intravenous diazepam, phenytoin, and magnesium sulfate her seizures were controlled. Her blood pressure was controlled gradually with amlodipine and labetalol. Intravenous dexamethasone was also given to prevent vasogenic edema. Her condition began to improve within 24 hours with control of seizures and blood pressure. After 48 hours she became well-oriented and fully conscious with GCS 15.

MRI of the Brain showed extensive bilateral symmetrical T2W-FLAIR hyperintensities with no perilesional edema noted in both parieto-occipital and both frontal regions.

**Discussion:**

The spectrum of clinical features, typical radiological changes, and various risk factors are crucial in making a diagnosis of PRES. The most common clinical symptoms and signs are headache, altered alertness ranging from drowsiness to stupor, seizures, vomiting, mental abnormalities including confusion and diminished spontaneity and speech, and abnormalities of visual perception. The onset is usually subacute but may be heralded by a seizure. Seizures are common at the onset of neurologic symptoms but can also develop later. Seizures may begin focially but usually become generalized. In this case, the patient developed repeated generalized tonic-clonic convulsions for 2-3 hours along with impaired consciousness. This indicates status epilepticus. Abnormalities of visual perception are nearly always detectable. Patients often report blurred vision. Hemianopia, visual neglect, and frank cortical blindness may occur. The most common abnormality on neuroimaging in patients with PRES was edema involving the white matter in the posterior portions of the cerebral hemispheres, especially bilaterally in the parieto-occipital regions. Similarly in this case, a CT scan of the brain revealed ill-defined hypodensity involving the cortex and subcortical white matter of both parietal and occipital lobes. MRI of the brain showed extensive bilateral symmetrical T2W-FLAIR hyperintensity in both parieto-occipital and both frontal regions confirming the PRES diagnosis. Rapid rises in blood pressures eventually overcome the autoregulatory capabilities of the cerebral vasculature causing vascular leakage and resultant vasogenic edema. Another proposes that endothelial dysfunction is the primary culprit, which may be caused by various endogenous or exogenous toxins leading to vascular injury with resultant development of vasogenic edema. Prompt recognition is the key as timely removal of the precipitating factor is important to achieve favorable outcomes. In patients with acute hypertension, gradual reduction of blood pressure should be performed (no more than 20–25% in the first few hours) to avoid the risk of cerebral, coronary, and renal ischemia. Seizures are very common and the most common antiepileptics that have been used during hospitalization include benzodiazepines, levetiracetam, and phenytoin, and upon discharge levetiracetam and phenytoin, with the majority of them on a single agent.
Conclusions:

PRES is an acute neurotoxic syndrome and the prognosis is highly variable. Patients with preeclampsia/eclampsia are found to have early recovery and good functional outcomes if prompt treatment is provided.

Reference: