

Peripartum Posterior Reversible Encephalopathy Syndrome: A rare pathology requiring a multidisciplinary approach

Savannah Nightingale¹; Kameron Tavakolian²; Anton Mararenko²; Sarah Esposito³; Swapnil Patel²

- 1. Robert Wood Johnson/Monmouth Medical Center, NJ, United States
- 2. Hackensack Meridian Jersey Shore University Medical Center, NJ, United States
- 3. Saint Georges University Medical Center, Grenada

Introduction

Posterior reversible encephalopathy (PRES) is a well-recognized disease process that can present in a variety of patient populations. A diagnosis is made based on presenting clinical picture and diagnostic neuroimaging. However, in pregnancy PRES can be difficult to distinguish from other disease processes such as Eclampsia. Initial presentation with new-onset seizure activity is seen in approximately 74-87% of patients who are diagnosed with PRES. The acute onset of this disease process can result in significant maternal and fetal morbidity and mortality. We present a rare case of a healthy 28-year-old gravid patient with a prenatal course complicated by new-onset seizure activity, with an ultimate diagnosis of PRES.

Case Presentation

A 28-year-old nulligravid at 29 weeks and 1-day gestation with no medical history presented to the ED following a witnessed seizure. Initial vitals revealed a blood pressure of 168/108 mm/Hg. She was treated with 20 mg intravenous Labetalol and continuous Magnesium Sulfate infusion. Following transfer to labor and delivery, an additional 140 mg intravenous Labetalol was required for persistent severe-range blood pressures. Upon stabilization, a primary cesarean section was performed without complication. Postpartum course was complicated by acute agitation and persistent severerange blood pressure. The patient was transferred to the ICU for continuous IV Clevidipine infusion. CT scan of the head revealed decreased attenuation in the occipital lobes bilaterally. MRI of the brain (figure 1) demonstrated areas of increased signal in the subcortical white matter of the bilateral occipital lobes, left temporal lobe, and left frontal cortex. The patient was ultimately diagnosed with PRES. Mentation improved to baseline with strict blood pressure control and she was eventually discharged with referral for outpatient neurology follow-up.

Discussion

PRES was first described by Hinchey et al. in 1996, which analyzed 15 patients. This study included three postpartum patients who developed Eclampsia. Initial symptoms include seizures, encephalopathy, and neurological deficits. The two most used diagnostic modalities are non-contrast CT scan and T2-MRI. Neuroimaging reveals vasogenic edema, located primarily in the parieto-occipital regions, which is largely symmetric, bilateral, and subcortical. As the density of the autonomic nervous system is present in the anterior circulation, the posterior blood-brain barrier remains vulnerable to hyperperfusion, resultant cerebral dysregulation, and vasogenic edema. While this condition can be reversible, prompt intervention is necessary. In pregnant patients, intervention is through immediate maternal stabilization with the administration of Magnesium Sulfate and use of anti-hypertensives as indicated. If there is minimal disruption of the maternal-fetal oxygenation pathway, maternal stabilization is achieved prior to immediate cesarean delivery. The diagnosis and management of PRES rely on a successful interplay between specialties. This case highlights the importance of this relationship which results in both decreased fetal and maternal morbidity and mortality.

References

1. Gewirtz AN, Gao V, Parauda SC, Robbins MS. Posterior Reversible Encephalopathy Syndrome. Curr Pain Headache Rep. 2021 Feb 25;25(3):19. doi: 10.1007/s11916-020-00932-1. PMID: 33630183; PMCID: PMC7905767.

The authors have no conflict of interest regarding this presentation to disclose

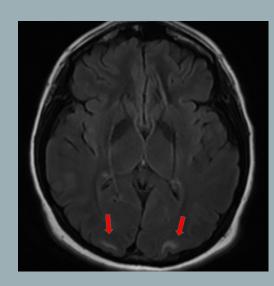


Figure 1. Brain MRI without contrast demonstrating areas of increased T2/FLAIR signal in the subcortical white matter of the bilateral occipital lobes (red arrows), left temporal lobe as well as single focus of increased T2/FLAIR left frontal cortex.