POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES)

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BACKGROUND
Posterior reversible encephalopathy syndrome (PRES) is a transient clinical neuroradiological entity characterized by clinical signs and symptoms including hypertension, generalized seizure activity, altered mental status, headache and vision changes; along with PRES findings characteristic on head computed tomography (CT) or magnetic resonance imaging (MRI) scan.\(^1\) Many causes of PRES have been reported in the literature including hypertensive encephalopathy, preeclampsia, eclampsia, renal failure, immunosuppressants, thrombotic thrombocytopenic purpura, systemic lupus erythematosus (SLE), and acute intermittent porphyria.\(^2\) The nomenclature for this syndrome has undergone several changes with one radiologic journal describing this entity as Eclamptic Encephalopathy.\(^3\) In the early postpartum period PRES is most often seen in association with severe elevated BPs, and with eclampsia. In the late postpartum period, it may be seen in the Emergency Department with a patient who presents with hypertension and seizures.

Preeclampsia and eclampsia are probably the most common causes of PRES. The true incidence of PRES complicating preeclampsia and eclampsia is unknown because neuroradiographic imaging is not routinely performed.

Symptoms: Neurologic symptoms commonly precede eclampsia. Headache and visual disturbance are the most common prodromal symptoms. These neurologic symptoms reflect the development of cerebral edema and vasospasm of cerebral and retinal vessels. Premonitory symptoms may provide an early warning of imminent eclampsia. Other common warning symptoms include nausea, vomiting, or epigastric/abdominal pain. These gastrointestinal symptoms are believed to reflect hepatocellular involvement from periportal or parenchymal necrosis, liver capsule stretching and/or hemorrhage.\(^4\)

Treatment: PRES is usually reversible with prompt diagnosis and treatment. Early recognition and effective treatment of blood pressure in patients with PRES in the acute setting along with seizure prophylaxis decreases long-term sequelae of this condition. The antepartum patient should be stabilized and then delivered.

However, in rare cases the reversible vasogenic edema associated with PRES can progress to irreversible ischemic damage, cerebral infarction or even death. Treatment is the same as for severe preeclampsia, and neurological consultation is recommended. The underlying pathophysiology has been attributed to failure of cerebral auto-regulation and endothelial dysfunction. This impairment of cerebral auto-regulation leads to disruption of the blood-brain barrier in the posterior circulation with resultant extravasation of fluids and protein across the altered blood-brain barrier.\(^5\) This process causes the characteristic lesions seen in the occipital and posterior parietal lobes on neuroradiologic
imaging (Photos A, B, C). These radiographic cerebral abnormalities appear as intense signals on T2-weighted MRI scans and as low-density areas on CT scans. (Photos are of same patient taken at the same time, different views.)

Photo A

Photo B

Photo C

Photos highlight involvement of the occipital region that explains the visual changes seen in preeclampsia/PRES. Photos used with kind permission of Thomas Archer, MD, University of California, San Diego, 2013.
**Diagnosis:** MRI is considered the most appropriate tool as an adjunct in the clinical setting to diagnosing PRES and demonstrating the characteristic brain lesions. MRI imaging is superior to CT imaging in patients with PRES or Eclamptic Encephalopathy. The hallmark feature is bilateral symmetrical vasogenic edema in the territories of the posterior cerebral circulation white matter (occipital and posterior parietal lobes). The posterior cerebral white matter edema is most evident on T2-weighted MRI images with fluid-attenuated inversion recovery (FLAIR). The predominance of occipital lesions corresponds well to the neurologic manifestation of temporary cortical blindness. Neuroradiologic imaging should be strongly considered in the postpartum period with a patient who presents with headache, hypertension, seizures, or atypical neurologic symptoms such as visual changes or inability to see (blindness), in order to rule out other differential diagnoses such as a mass lesion or cerebral venous thrombosis.

**KEY LEARNING POINTS**

1. Upon initial discharge from the hospital, inform the recently delivered patient that there is still a measurable risk of Preeclampsia/Eclampsia in the postpartum period for up to six (6) weeks after delivery, even with an uneventful pregnancy and delivery. Although delayed postpartum preeclampsia is usually seen within the first two weeks postpartum, some patients can present up to four to six (4-6) weeks after delivery. This risk may be increased in patients who have experienced preeclampsia prior to delivery. Therefore, delayed preeclampsia should be considered in any patient within the normal six-week postpartum period.

2. It is important for Emergency Department physicians/staff to have a high index of suspicion for postpartum Preeclampsia/Eclampsia and to have early involvement of obstetric staff in the care of these patients.

3. Upon presentation to the ED, a female patient should be queried as to whether she is currently pregnant or has recently been pregnant.

4. Antihypertensive medications should be implemented for systolic BP > 160 mm Hg and/or if diastolic BP > 105-110 mm Hg that persists for 15 minutes or greater and should be considered in the group of patients that have blood pressures that are > 155 systolic and 105 diastolic.

5. Treatment guidelines for severe hypertension are identical to those for severe preeclampsia/eclampsia.
RECOMMENDATIONS FOR QUALITY IMPROVEMENT:

1. Initiate established treatment algorithms for severe preeclampsia and eclampsia for control of hypertension and seizures.

2. First-line antihypertensive medication is intravenous Labetalol or Hydralazine. (See Antihypertensive Agent chapter, pg. 45)

3. Magnesium sulfate is the treatment of choice for controlling seizures in eclampsia and for prevention of recurrent seizures. This should be maintained for at least 24 hours after the last seizure. (See Magnesium Sulfate chapter, pg. 50)

4. If patient is pregnant, maternal and fetal monitoring is advised on labor and delivery as fetal status is secondary to maternal stabilization.

5. In these complex clinical cases, a multidisciplinary team of ED, OB, Anesthesia, and possibly Neurology or Critical Care is necessary.

6. Do not delay treatment to perform neuroradiologic imaging.

7. Neuroradiologic imaging is strongly advised in the postpartum period due to the numerous differential diagnoses and to exclude other intracranial pathology.

EVIDENCE GRADING
Level of Evidence: III

REFERENCES


