CE 1.5 ANCC Contact Hours

Abstract

Posterior reversible encephalopathy syndrome (PRES) is a newly defined syndrome; therefore, this transient clinical condition is not well known and probably underdiagnosed. It develops quickly with symptoms that are usually indistinguishable from eclampsia. Nurses need to be knowledgeable and aware of identifying symptoms and appropriate treatment. The condition is thought to share pathophysiology with eclampsia, and it is suggested that endothelial dysfunction combined with hypertension causes disruption in the blood brain barrier resulting in cerebral edema. Seizures develop secondary to cerebral edema, and mark later stages of the disease. Treatment is aimed at reducing blood pressure (BP) with antihypertensive therapy and seizure control with magnesium sulfate. When PRES is treated early, symptoms typically disappear within a few days and imaging studies normalize in several weeks. Permanent brain damage can occur if diagnosis and treatment are delayed. If PRES is suspected, thorough focused assessments and increased communication among the healthcare team are essential for patient care. When pregnant or postpartum women present with elevated BP accompanied with neurologic symptoms, imaging studies should be considered. An exemplar case is presented of a woman with normal prenatal course that is complicated by rapidly developing preeclampsia, eclampsia, and PRES.

Key words: Eclampsia; Hypertension; Posterior reversible encephalopathy syndrome; Preeclampsia; Seizure.

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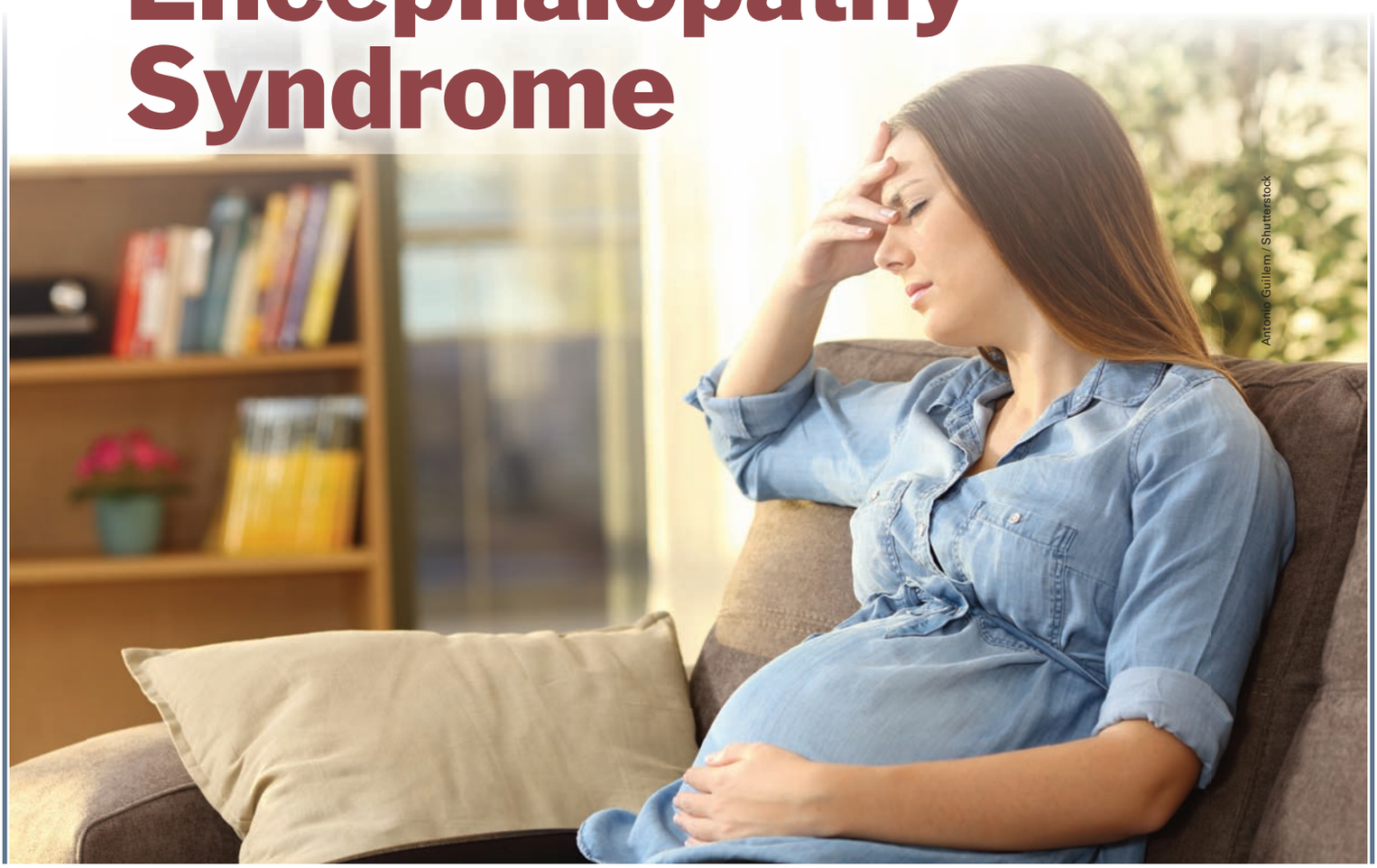
Posterior Reversible Encephalopathy Syndrome



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Posterior reversible encephalopathy syndrome (PRES) is a transient clinical condition that is related to multiple factors and etiologies (Demirel, Kavak, Ozer, Bayar, & Erhan, 2014). It was first described by Hinchey in 1996, and is associated with a sudden increase in blood pressure (BP) related to a dysfunctional autoregulation of circulation in the brain leading to cerebral edema (Demirel et al.). Symptoms are usually indistinguishable from eclampsia: headache, visual abnormalities, nausea, vomiting, and focal neurologic deficits, progressing to impaired consciousness, seizure activity, and coma (Demir, Ozerkan, Ozbek, Yıldırım Eryilmaz, & Ocakoglu, 2012; Kurdoglu et al., 2015; Patil et al., 2015). Many conditions have been reported to cause PRES, including preeclampsia; eclampsia; hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome; autoimmune disease; and sepsis. Hypertension remains the most commonly identified trigger of PRES (Kurdoglu et al.; Patil et al.). The mechanism of PRES is poorly understood; however, it appears to share the same pathology of eclampsia (Demir et al.). Diagnosed with radiological studies, PRES is usually reversible if diagnosed and treated early; in the later stages, permanent brain damage and chronic epilepsy can occur (Kurdoglu et al.).

Symptoms of posterior reversible encephalopathy syndrome are usually indistinguishable from eclampsia.

Worldwide, approximately 10% of pregnancies are affected by hypertension, and about 18% of maternal deaths are attributed to hypertensive disorders of pregnancy (Kurdoglu et al., 2015; Too & Hill, 2013). There is a reported global eclampsia rate of 0.28% of births with a maternal mortality rate of 3.66% in pregnant women with eclampsia. Incidence and prevalence of PRES are thought to be underdetected and underreported because it is a condition that is often not recognized (Kurdoglu et al.).

Definition

Posterior reversible encephalopathy syndrome (PRES) is a transient clinical condition that is diagnosed by presence of neurological symptoms and specific radiological findings (Demirel et al., 2014; Kutlesič, Kutlesič, & Koratevič, 2015). Eclampsia is defined as presence of seizures and a diagnosis of preeclampsia (Kutlesič et al.). Seizures are caused by a transient disturbance in cerebral function (Hart & Sibai, 2013).

Preeclampsia affects between 5% and 8% of pregnancies and is one of the leading causes of maternal and fetal morbidity and mortality. It resolves only after delivery of the placenta (Stocks, 2014). The reported rate of eclampsia in the western world has decreased over the past 10 years; from 12.4 in 10,000 pregnancies down to 4 to 5 in 10,000 pregnancies (Hart & Sibai, 2013; Liu et al., 2011). Preeclampsia is defined as hypertension that develops after 20 weeks of gestation accompanied by proteinuria. However, according to the American College of Obstetricians and Gynecologists (ACOG) task force on hypertension in pregnancy, proteinuria is no longer necessary for the diagnosis of preeclampsia. If proteinuria is absent, preeclampsia can be diagnosed if the woman has any of the following: thrombocytopenia, renal insufficiency, elevated liver enzymes, pulmonary edema, or cerebral or visual symptoms (ACOG, 2013).

Pathophysiology

The pathophysiology of PRES and eclampsia is not fully understood; however, they have many factors in common (Kurdoglu et al., 2015). Endothelial dysfunction combined with a sudden rise of BP that surpasses cerebral autoregulation results in an increase in permeability of the blood brain barrier (BBB), which allows fluid and blood to leak into brain tissue, resulting in cerebral edema (Demirel et al., 2014; Patil et al., 2015; Razmara, Bakhadirov, Batra, & Feske, 2014).

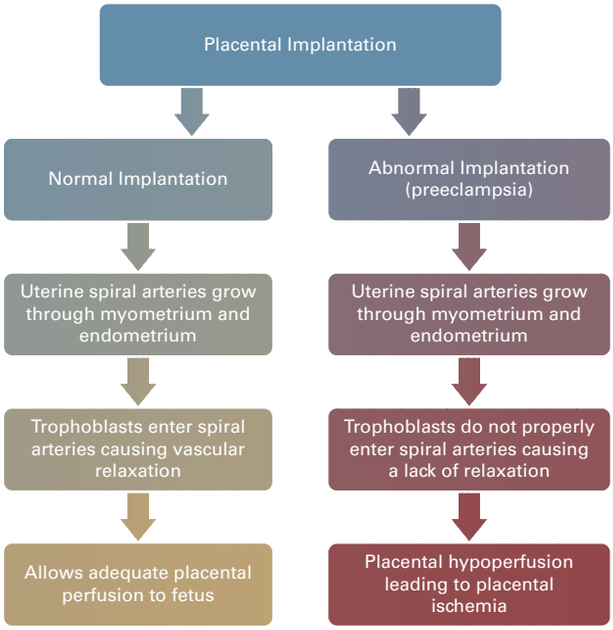
The current theory on eclampsia and its neurologic symptoms suggests that the symptoms are due to a failed autoregulation of the brain. Normally the brain is able to regulate a steady blood flow regardless of the body's constant fluctuation in mean arterial BP ranging from 60 to 150 mmHg (Ohno et al., 2013). However, blood vessels are injured when BPs increase beyond the brain's autoregulation capacity. These injured vessels, combined with hyperperfusion, lead to cerebral edema (Demir et al., 2012). Seizures mark later stages of the disease, developing secondary to cerebral edema and disruption of the BBB (Razmara et al., 2014).

Pathophysiology of preeclampsia is not fully understood, but is thought to contain both genetic and immunological factors. Normally when the placenta implants, uterine spiral arteries grow through the myometrium and into the endometrium. Trophoblasts then enter the spiral arteries and reduce elasticity causing relaxation of the vascular smooth muscle. This allows the placenta to adequately perfuse the fetus. However, with preeclampsia, trophoblasts do not properly enter the spiral arteries causing a lack of relaxation leading to hypoperfusion of the placenta that leads to placental ischemia (Figure 1). Placental ischemia is thought to cause an abnormal activation of the maternal vascular endothelium leading to potential multiorgan damage (Olson-Chen & Seligman, 2016; Stocks, 2014).

Risk Factors

Known risk factors for preeclampsia include maternal age over 40, nulliparity, a previous pregnancy with preeclampsia, family history of preeclampsia, previous pregnancy over 10 years ago, a new partner, obesity, chronic hypertension, renal disease, diabetes, and multiple gestation (Razmara et al., 2014). Risk factors for PRES are younger age, higher systolic and diastolic BPs, eclampsia, and lower platelets (Saraf et al., 2014).

Figure 1. Preeclampsia and Placentation



Signs, Symptoms, and Clinical Manifestations

Posterior reversible encephalopathy syndrome typically develops quickly, with symptoms reaching their peak around 12 to 48 hours after onset, commonly progressing over the next several days (Kurdoglu et al., 2015). Symptoms of PRES are usually indistinguishable from eclampsia and include the following: headache, blurry vision, cortical blindness, nausea, vomiting, impaired consciousness, and seizure activity (Demir et al., 2012; Kurdoglu et al.; Patil et al., 2015) (Table 1). Seizure activity is the most frequent symptom and is present in 90% of PRES cases. This is often the first symptom to appear (Demir et al.). Symptoms of PRES usually resolve within a week; however, some cases have persisted longer (Kurdoglu et al.).

Women who develop eclampsia typically present with complaints of a persistent frontal or occipital headache, visual disturbances, right upper quadrant pain/epigastric pain, and an altered mental state (Demir et al., 2012). Women with eclampsia will have at least one of the above symptoms in 59% to 75% of cases; headaches are reported in 50% to 75% of cases (Hart & Sibai, 2013). Eclampsia occurred most commonly during labor (39.7%) and postpartum (43.6%) (Ohno et al., 2013).

Diagnosis & Treatment

The gold standard for diagnosing PRES is completed with magnetic resonance imaging (MRI), typically showing posterior cerebral edema with the parietal–occipital lobe being the most highly affected region. Most women with PRES make a full recovery within a few weeks. If PRES is treated early and aggressively, symptoms usually disappear in a few days, whereas neuroimaging abnormalities may take several weeks to resolve. However, if treatment is delayed, permanent brain damage can occur (Demir et al., 2012; Demirel et al., 2014). In a recent study that evaluated seven women with severe preeclampsia/eclampsia and PRES, all but one patient improved neurologically within 2 to 5 days after diagnosis. The MRI findings were normal in all patients 1 month later. Treatment should focus on removing the PRES-triggering factors, hypertension being of utmost importance. Reduction in BP and control of seizures are paramount for full recovery (Patil et al., 2015). Current recommendations for treatment consist of antihypertensive agents and a magnesium sulfate infusion. Magnesium sulfate causes vasodilation that increases cerebral blood flow and prevents ischemia (Demirel et al.). Magnesium sulfate also prevents edema by reducing cerebral endothelial permeability and protects the BBB. This helps prevent PRES and its related neurological symptoms (Demir et al.; Kutlesič et al., 2015). Mannitol is also a medication used in PRES patients that focuses on decreasing intracranial pressure. One study compared effectiveness of magnesium sulfate and mannitol in 62 women with eclampsia and PRES. Women receiving magnesium sulfate had both better neurological outcomes and shorter hospital stays. Magnesium sulfate is recommended as the first-line treatment for both women with preeclampsia and those with PRES (Demir et al.).

Women with eclampsia may require supplemental oxygen as hypoventilation and subsequent respiratory acidosis often occur. Even though the seizure typically only lasts a few minutes, and the patient does not breathe during a seizure, oxygenation is a primary intervention to prevent prolonged hypoxemia and acidosis. Management of eclampsia revolves around preventing further seizures with magnesium sulfate. Currently, a 6 g loading dose infused over 15 to 20 minutes, followed by a 2 g/hour is the recommendation for women with eclampsia. Ten percent of patients will have a second seizure (Hart & Sibai, 2013).

Decreasing BP is of prime importance because a severely elevated systolic BP is a significant predictor of cerebral

Table 1. Neurologic Symptoms of PRES and Eclampsia

Headache
Blurry vision
Cortical blindness
Nausea & vomiting
Impaired consciousness
Seizure activity
Neurological deficits
Coma

Note. Adapted from Demir et al. (2012), Kurdoglu et al. (2015), and Patil et al. (2015).

complications (Kutlesič et al., 2015). Clinicians generally agree that severe hypertension of greater than 160/110 mmHg that is persistent and lasts for greater than 15 minutes should be treated (Too & Hill, 2013). However, it is important to decrease BP slowly as a rapid decrease in BP can decrease uteroplacental blood flow and adversely affect the fetus (Stocks, 2014). Antihypertensive medications should be used in women with severe range BPs, see Table 2 (Archer, Druzin, Shields, & Peterson, 2013; Hart & Sibai, 2013; Olson-Chen & Seligman, 2016). Target BP ranges (140 to 160 mmHg for systolic BP and 90 to 105 mmHg for diastolic BP) ensure adequate uteroplacental blood flow and maternal cerebral perfusion (Hart & Sibai). It is important to remember that preeclampsia is a progressive disease and thus frequent assessments are needed to determine whether severity of the disease is increasing (ACOG, 2013). Magnesium sulfate reduces risk of seizures by more than 50% and therefore is the treatment of choice. The infusion is usually given until 24 hours after birth (Kutlesič et al.; Stocks).

If hypertension is not treated, serious complications such as intracerebral hemorrhage, hypertensive encephalopathy, myocardial ischemia, and cardiac failure can occur. When eclampsia develops, the woman is at risk for intracerebral hemorrhage, cardiac arrest, and death (Stocks, 2014). Perinatal complications for PRES and cerebrovascular disorders in pregnancy include lower Apgar scores, increased risk for stillbirth, infant death, prematurity, and fetal hypoxia (Hart & Sibai, 2013; Kurdoglu et al., 2015).

Case Presentation

At 1140, a 16-year-old woman having her first baby presented to the obstetrical emergency room at 38/6 weeks gestation. She reported a headache for the last 2 days that had begun to worsen over the last 3 hours. Her prenatal record revealed that she did not receive prenatal care until 20 weeks gestation, but she had no significant prenatal complications, no abnormal lab values, and no abnormal vital signs throughout her prenatal visits. The only significant medical history reported was a childhood history of asthma.

On assessment, she reported positive fetal movement, nausea, seeing spots in her field of vision, and increased swelling in her legs and feet. Initial vital signs were 153/93 mmHg, 105 bpm, 18 respirations, and a temperature of 98.6 °F. Fetal heart rate (FHR) baseline was 120 bpm, minimal to moderate variability, accelerations, no decelerations, and irregular contractions palpating mild. The nurse's assessment findings revealed 3+ deep tendon reflexes (DTRs), 3+ pitting edema in lower extremities bilaterally, no clonus, and an occipital and temporal headache rating 9/10 on pain scale. Repeat BP was 180/101 mmHg, and the routine urinalysis revealed 3+ protein. The OB hospitalist was notified of the patient arrival, complaint, her history, and nurse's assessment. Orders were received to admit to labor and delivery and to initiate magnesium sulfate for preeclampsia. The admission process was started and the following BP was 159/103 mmHg.

At 1215, she had a 5-minute eclamptic generalized seizure with rigidity and jerking. During the seizure, she was repositioned into the left lateral position, and the nurse called for additional help. While postictal, the patient was reported to be combative, moving constantly, and attempting to remove fetal monitors. During this time, there was a 5-minute prolonged FHR deceleration. At 1220, an intravenous (IV) was started, admit labs were drawn, a 6 g magnesium sulfate bolus was initiated, and the patient was being prepped for a primary cesarean. The FHR returned to 120 bpm baseline with minimal to moderate variability, no decelerations or accelerations. At 1239, a second eclamptic seizure occurred and lasted for 2 minutes. It appeared to be generalized clonic with mild rigidity. Initial maternal lab values were within normal limits and included: creatinine clearance 0.7 mL/min, aspartate aminotransferase 22 units/L, alanine aminotransferase 13 units/L, platelets 361 × 10^9/L,

Table 2. Antihypertensive Medications

Labetalol	Hydralazine	Nifedipine
Alpha- and beta-blocker that reduces blood pressure (BP) by dilating arterioles and decreasing heart rate	Arterial dilator that reduces BP; may cause tachycardia	Calcium channel blocker; may cause tachycardia and headaches
20 mg IV push over 2 min. Increase every 10–15 min Example: 20 mg–40 mg–80 mg–80 mg	5–10 mg IV push over 2 min. May give every 15–20 min	10–20 mg PO every 30 min
Onset is 2–5 min	Onset is 5–20 min	Onset is 5–20 min
Maximum effect in 5 min	Maximum effect 15–30 min	Maximum effect 30–60 min
Consider alternative drug after 220 mg	Consider alternative drug after 25 mg	Consider alternative drug after 50 mg
Maximum dose 220–300 mg	Maximum dose 25 mg	Maximum dose 50 mg in 1 hr
Contraindicated: asthma, cocaine, and amphetamine	Avoid in severe headache and tachycardia	Avoid in severe headache and tachycardia

Note. Adapted from Archer et al. (2013), Hart & Sibai (2013), and Olson-Chen & Seligman (2016).

prothrombin time 8.8 seconds, fibrinogen 426 mg/dL, hemoglobin 9.8 g/dL, and hematocrit 33.2%.

At 1241, she was transferred postictal to the operating room (OR) for an emergent cesarean. On entering the OR, the FHR was reported to be in the 50s. At 1247, a third eclamptic seizure occurred and lasted for 3 minutes. The patient was then intubated and general anesthesia was induced. Surgery started at 1251. Maternal vital signs included BPs ranging from 180/90 to 190/110 mmHg and a heart rate (HR) between 130 and 140 bpm. The baby was born at 1254 with Apgar scores of 1 at 1 minute, 4 at 5 minutes, and 6 at 10 minutes. Surgery was completed at 1350. Blood loss was estimated to be 800 mL. Umbilical artery gasses were pH 6.76, HCO_3 17.8 mEq/L, PCO_2 > 90 mmHg, base excess (BE) -17, and the baby was transferred to the neonatal intensive care unit (NICU). The baby was diagnosed with suspected hypoxic ischemic encephalopathy upon admission to the NICU and transferred to a level 4 NICU for whole body cooling therapy.

The intubated patient was taken to the intensive care unit (ICU) at 1355. On admission to ICU, her vital signs were BP 180/76, HR 107, and O_2 75% to 98%. A head computerized tomography (CT) scan and neurology consult were ordered. Initial maternal blood gases were pH 7.34, CO_2 34.3, HCO_3 18, BE -7.1, and O_2 170. At 1545, the woman was transferred to radiology for a CT scan, and at 1610 she had a fourth generalized clonic seizure that lasted 5 minutes. The ICU nurse then contacted the physician, administered Lorazepam 2 mg IV push, and applied oxygen following the seizure. The woman was transferred back to the ICU following the CT scan at 1645. The CT report showed multifocal areas of low density in right cerebrum with concerning subacute infarcts. An MRI was recommended by radiologist for further evaluation of the CT findings. A neurology consult was completed and additional orders for electroencephalography (EEG), MRI, and magnetic resonance venography (MRV) were obtained. The neurologist assessment findings found an intact neuro check and 2+ DTR, 4+ dependent edema bilaterally.

The neurologist's plan included continuing magnesium for seizure activity, Lorazepam as needed for additional seizures and strict control of BP. Following the MRI at 2105, the radiologist reported findings on FLAIR imaging were as follows: multifocal areas of abnormal posterior cerebral and cerebellar signal, likely edema caused by PRES. Lab results were within normal limits with exception of serum potassium level of 6 mmol/L. At 2229, the MRV was completed and was negative as no thrombi were found in the cerebral vessels. At 2255, the ICU nurse documented a fifth seizure that lasted approximately 2 minutes. Lorazepam 2 mg IV push was administered and the physician was notified.

On postoperative day one, all maternal labs and blood gases were within normal limits and EEG showed no seizure activity. The woman was extubated and placed on nasal cannula. Nursing assessment documented that patient denied headache, 2+ DTRs present, and intact neuro check. The patient's only complaint at this time included abdominal incision pain rating 6/10. Her BP ranged from 112/76 to 148/87 mmHg and HR was in the 110 bpm. At 1930, the nasal cannula was removed and she was able to maintain adequate oxygenation on room air. At 2200, she was able to ambulate without difficulty. At 0020, magnesium sulfate was discontinued. During the following 3 days, she continued to improve, vital signs normalized, and she was discharged from the hospital in stable condition with no additional seizures. The baby was discharged from the NICU after 14 days of stay in stable condition.

Nursing Implications

Because of the similarities between PRES and eclampsia, there should be a heightened suspicion of PRES, and imaging should be considered when pregnant women or postpartum women present with elevated BP and neurological symptoms. Focused assessments, increased communication with physicians, and patient care will be enhanced as more nurses become knowledgeable and aware of PRES. Nurses play an integral role in assessment. It is often based on this assessment that providers choose to further evaluate abnormalities. By communicating these assessment findings to the provider, nurses are instrumental in advocating for and facilitating more aggressive surveillance and early treatment for their patients, thereby preventing further complications.

Treatment and management of preeclampsia should take a multidisciplinary team approach and focus on treating the elevated BP and minimizing complications to both mother and fetus. Careful consideration needs to be taken in planning the timing of birth by weighing risks versus benefits to both the mother and fetus. Simulation and mock eclamptic codes can help facilitate a smooth multidisciplinary approach.

Education for patients with PRES is a crucial component of nursing care. Whether the woman is in the antepartum or postpartum phase, all patients with hypertension in pregnancy should receive thorough discharge instructions that include important signs and symptoms to report to their provider. These include: elevated BP, sudden weight gain, blurred vision or light sensitivity, swelling of hands or feet, nausea or vomiting, lightheadedness, headaches, abdominal pain, and seizures (Kellicker, 2016).

With more research and education, it is likely that the reported cases of PRES will continue to increase. It is important that awareness increase because swift and aggressive treatment is crucial to prevent permanent neurological damage to mother and fetus. ✣

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