

Subarachnoid Hemorrhage and Posterior Reversible Encephalopathy Syndrome (PRES): A Case Report Associated with Eclampsia and Hypertension

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ABSTRACT

Posterior Reversible Encephalopathy Syndrome (PRES) is thought to occur when there is a sudden elevation in blood pressure that surpasses the autoregulatory capacity of the cerebral vasculature. This condition leads to endothelial dysfunction, resulting in the breakdown of the blood brain barrier and the development of vasogenic edema. A 26-year-old nulliparous woman at 30 weeks of gestation presented to the emergency department with severe headache and seizures. Blood pressure monitoring showed 200/110 mmHg, prompting an emergency cesarean section due to suspected eclampsia. After ultrasound measurements indicated early fetal growth restriction consistent with 27 weeks, a 1000 g female infant (<1 percentile) with Apgar scores of 5 and 7 was delivered under general anesthesia. MRI revealed a subarachnoid hemorrhage and findings consistent with posterior reversible encephalopathy syndrome. The patient received antihypertensive and antiepileptic treatment in the Intensive Care Unit. In a subsequent pregnancy 11 months later, she underwent cesarean delivery at 34 weeks due to preterm labor, delivering a 2550 g male infant with Apgar scores of 7 and 8. No recurrence of PRES was observed. This case highlights the potential for reversible progression of PRES and subarachnoid hemorrhage during pregnancy with timely diagnosis and appropriate management.

Keywords: Posterior reversible encephalopathy syndrome, subarachnoid hemorrhage, cerebral edema, eclampsia, maternal neurological complications

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a rare clinical condition that can be diagnosed radiologically and is most commonly characterized by reversible subcortical vasogenic edema in the parieto-occipital regions (1). The true prevalence of PRES remains unknown; however, it is most commonly observed in association with hypertensive disorders, renal dysfunction, immunosuppressive therapy, and preeclampsia/eclampsia (2). Early diagnosis and treatment of underlying causes, such as hypertensive emergencies like preeclampsia, eclampsia, or systemic inflammation, are vital for full clinical and radiological recovery within days or weeks. Delayed intervention may result in serious complications, including massive posterior fossa edema and brainstem compression (1, 2). Encephalopathy is a common feature, varying from subtle confusion to deep stupor, while

generalized tonic-clonic seizures occur in nearly 60–75% of cases (2).

The pathophysiology of PRES and subarachnoid hemorrhage (SAH) remains incompletely understood and is likely multifactorial. One theory attributes SAH to hypertension-induced vessel rupture from blood pressure fluctuations, while another suggests severe hypertension overwhelms cerebral autoregulation, disrupting the blood-brain barrier and causing fluid leakage into brain tissue (3). Another proposed mechanism involves systemic endothelial dysfunction and vasoconstriction, which circulating toxins can trigger (4). Intracranial hemorrhage occurs in an estimated 10–25% of PRES cases. Among these, intraparenchymal hemorrhage is the most frequently observed pattern, whereas sulcal subarachnoid hemorrhage is the second most common (2).

Preeclampsia is a pregnancy-specific hypertensive disorder that typically occurs after 20 weeks of gestation. It is characterized by new-onset

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hypertension along with proteinuria or signs of end-organ dysfunction (5). Additionally, eclampsia refers to the occurrence of generalized tonic-clonic seizures in a woman with preeclampsia, without the presence of other neurological conditions (6). Both preeclampsia and eclampsia are well-recognized clinical precipitating factors of PRES, due to their shared pathophysiological mechanisms involving endothelial dysfunction and vasogenic cerebral edema.

This case aims to emphasize the need for prompt recognition of atypical presentations of PRES in obstetric patients by presenting a rare case associated with eclampsia and subarachnoid hemorrhage, and to highlight the importance of timely neuroimaging and multidisciplinary management in achieving favorable outcomes.

Case Report

A 26-year-old G1P0A0 nulliparous woman at 30 weeks of gestation presented to the emergency department with a severe headache and a seizure earlier that day. During her evaluation, she experienced a second generalized tonic-clonic seizure with an epileptic pattern, and her blood pressure was recorded at 200/110 mmHg. Ultrasonographic evaluation revealed fetal biometric measurements below the 5th percentile, consistent with 27 weeks of gestation, indicating early-onset fetal growth restriction.

The patient had no history of surgery or known chronic illness. She was receiving insulin therapy for gestational diabetes mellitus. First-trimester genetic screening and second-trimester level II ultrasound had not been performed. Upon admission, laboratory tests showed: hemoglobin 12.3 g/dL, platelets 261×10^9 /L, white blood cells 14.4×10^9 /L, creatinine 0.96 mg/dL, uric acid 9.2 mg/dL, ALT 15 U/L, AST 22 U/L. Spot urine analysis revealed +3 proteinuria with a protein/creatinine ratio of 23 mg/mg. Blood type was A Rh-positive.

For preoperative eclampsia prophylaxis, the patient received a 6 g loading dose of magnesium sulfate (MgSO_4), followed by a maintenance infusion at 2 g/hour. A single 6 mg dose of betamethasone was administered as an antenatal corticosteroid. During her admission to the obstetric clinic, the patient experienced a third eclamptic seizure, which was promptly managed with a 10 mg bolus dose of benzodiazepine. She exhibited disorientation and somnolence. Due to her altered mental status and limited intravenous antihypertensive options, nitroglycerin infusion was initiated at a starting dose

of 5 mcg/min. Due to suspected eclampsia, emergency cesarean delivery was performed. Under general anesthesia, a 1000 g female infant (<1st percentile) was delivered with Apgar scores of 5 and 7 at one and five minutes, respectively.

At two hours postoperatively, the patient remained disoriented and confused. In addition to the bilateral frontal subcortical hyperdense areas consistent with subarachnoid hemorrhage observed on cranial computed tomography (CT), magnetic resonance imaging (MRI) with FLAIR (T2-weighted) sequences demonstrated bilateral symmetrical hyperintense cortical and subcortical lesions, predominantly in the occipital lobes, diffusely extending into the cerebellum and other cerebral regions. The radiology department performed a specialist consultation, and the findings were interpreted as consistent with PRES (Figure 1).

The patient was admitted to the intensive care unit. In addition to uterotonic therapy, she was started on antihypertensive treatment with beta-blocker esmolol infusion, targeting systolic blood pressure below 130 mmHg. Antiepileptic therapy was initiated with a loading dose of 2000 mg levetiracetam, followed by a maintenance dose of 500 mg twice daily.

Following treatment, her blood pressure stabilized at 140/90 mmHg. The patient became oriented and cooperative, with resolution of her headache. No progression of intracranial hemorrhage was observed on follow-up imaging, and surgical intervention was deemed unnecessary. After stable blood pressure and improved mental status, and with regression of hemorrhagic findings on follow-up CT, the patient was discharged on postoperative day 11.

At 8 months postoperatively, follow-up MRI revealed no pathology secondary to the previously diagnosed condition. The hyperintense areas predominantly seen in the posterior regions on T2-weighted images, as well as the diffuse cortical and subcortical hyperintensities throughout the cerebral hemispheres including the cerebellum, which were consistent with PRES, had completely resolved (Figure 2). Minimal ischemic changes were observed in the left frontal subarachnoid region, likely secondary to the prior subarachnoid hemorrhage.

Three months after the cesarean section, the patient conceived again. During her second pregnancy, she presented to the emergency department seven times due to headaches; only symptomatic treatment was administered, and no specific pathology was detected. At 34 weeks of gestation, due to the onset of preterm labor, an emergency cesarean section was performed, delivering a 2550 g male infant (75th percentile) with Apgar scores of 7 and 8. In this second pregnancy,

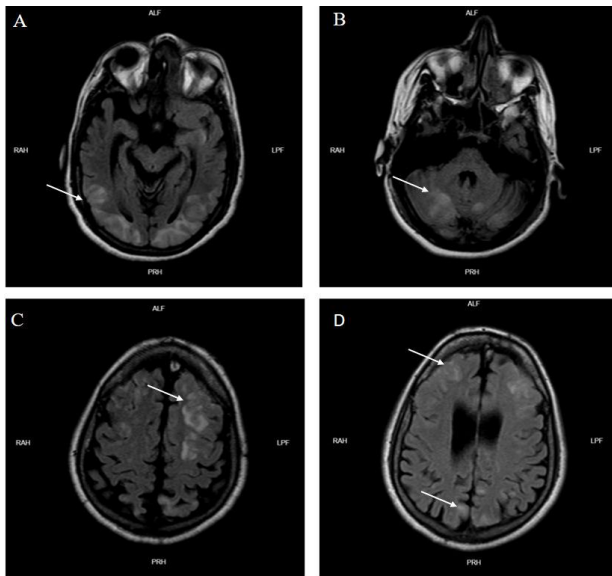


Fig. 1. A. FLAIR (T2-weighted) sequence demonstrating bilateral symmetrical hyperintense cortical and subcortical lesions predominantly in the occipital lobes, consistent with vasogenic edema. B. Hyperintense lesions observed over the cerebellum. C. Scattered hyperintense lesions throughout the cerebral regions. D. Evidence of subarachnoid hemorrhage in the frontoparietal lobes with associated cerebral lesions in the occipital region.

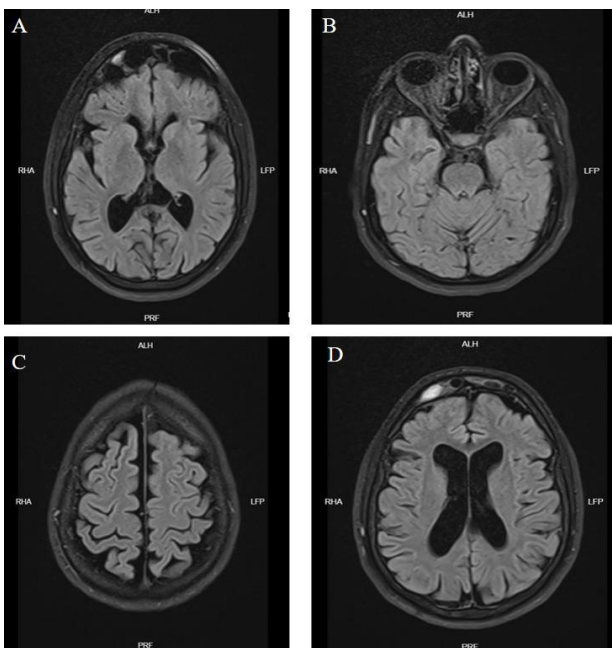


Fig. 2. Normal post-treatment MRI demonstrating complete resolution of widespread hyperintense lesions previously observed in the posterior and cerebral regions, including the frontoparietal subarachnoid hemorrhage area, consistent with recovery from subarachnoid hemorrhage and posterior reversible encephalopathy syndrome (PRES)

the patient was started on low-dose acetylsalicylic acid (ASA) at 100 mg/day. She continued oral antiepileptic treatment with levetiracetam due to chronic epilepsy. No anti-hypertensive therapy was required, and no signs of preeclampsia were observed.

Discussion

Although the exact pathophysiological mechanism of PRES remains unclear, the presence of underlying autoimmune conditions that may support endothelial dysfunction is often implicated. The location, extent, and severity of vasogenic edema, observed as hyperintense lesions on MRI, can result in a wide range of clinical presentations with varying intensities (7). In addition to autoimmune diseases such as systemic lupus erythematosus, acute blood pressure elevations due to conditions like eclampsia or primary renal disease are frequently identified as underlying causes. It is believed that severe hypertension contributes to endothelial injury and disruption of the blood-brain barrier (8, 9).

Because preeclampsia was considered the most likely underlying etiology in this case, low-dose aspirin prophylaxis was recommended for a subsequent pregnancy. Aspirin at low doses inhibits platelet cyclooxygenase-1 (COX-1), reducing thromboxane A2 (TxA2) synthesis while sparing prostacyclin production, thereby improving uteroplacental blood flow and modulating endothelial dysfunction. This mechanism is particularly relevant in patients at high risk of preeclampsia, in whom an imbalance between vasoconstrictive and vasodilatory pathways contributes to abnormal placentation and maternal systemic vascular dysregulation (10).

In our case, although no autoimmune background was present, eclamptic episodes characterized by headache and seizures were considered the primary etiological factor. The continuation of headache throughout the day suggests prolonged exposure to elevated blood pressure, which is consistent with literature data indicating the potential development of cerebral vasogenic edema under such conditions.

In the differential diagnosis of PRES during pregnancy, it is essential to consider HELLP syndrome (Hemolysis, Elevated Liver enzymes, and Low Platelet count) and reversible cerebral vasoconstriction syndrome (RCVS), as both may present with overlapping symptoms such as headache, altered mental status, and seizures (11). Reports indicate, that PRES occurs in about 17–38% of individuals with reversible cerebral vasoconstriction syndrome (2).

Endothelial damage and disseminated intravascular coagulation (DIC) from HELLP syndrome can lead to cerebral complications. Therefore, PRES and HELLP should be seen as interconnected conditions rather than mutually exclusive. Diagnosis requires a holistic approach, considering clinical observations, laboratory results, and imaging data. Key laboratory findings help differentiate the two: HELLP syndrome typically presents with hemolysis (elevated LDH and indirect bilirubin), thrombocytopenia (platelet count $<100,000/\text{mm}^3$), and significantly elevated liver enzymes (AST/ALT), while these findings are generally not present in PRES (12). In RCVS, severe headache is the predominant clinical symptom and often occurs without accompanying neurological deficits. Although both conditions are reversible, RCVS typically resolves within 1–3 months as the vascular constriction subsides, whereas the clinical symptoms and imaging findings of PRES may resolve completely within days as cerebral edema regresses (11).

Another diagnosis that should be considered clinically due to neurological symptoms is meningitis. However, the absence of neck stiffness, fever, and an increase in infective parameters such as WBC, CRP, and sedimentation rate in the clinical evaluation exclude meningitis in the preliminary diagnosis (13).

The primary goal of treatment is to reduce the mean arterial pressure to premorbid levels. In emergency management, intravenous nicardipine (5-15 mg/hour) and labetalol (2-3 mg/minute) are considered first-line agents. Epilepsy is recognized as a long-term complication of PRES, as there have been reports of patients developing hippocampal sclerosis or permanent focal changes, and even temporal lobe epilepsy months to years after a PRES episode (14). Long-term neurological deficits are observed in about 10–20% of individuals diagnosed with PRES (2).

Although it is well established that early diagnosis and treatment of the underlying cause in PRES can lead to full clinical and radiological recovery from vasogenic edema, delayed intervention has been associated with irreversible cytotoxic edema and chronic neurological sequelae (9-15).

In conclusion, this case represents an instance of eclampsia complicated by both subarachnoid hemorrhage and posterior reversible encephalopathy syndrome (PRES). This case report reinforces the existing literature by supporting the reversibility of PRES, as evidenced by the absence of symptoms and lack of MRI abnormalities during the subsequent pregnancy. Timely and accurate diagnosis, combined with appropriate management, is essential to prevent

permanent neurological damage both clinically and radiologically.

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