International Journal of Clinical Obstetrics and Gynaecology

ISSN (P): 2522-6614
ISSN (E): 2522-6622

www.gynaecologyjournal.com
2025; 9(2): 100-103
Received: 20-01-2025
Accepted: 23-02-2025

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Posterior reversible encephalopathy syndrome in obstetrics: Pathophysiology, diagnosis, and management considerations

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DOI: https://www.doi.org/10.33545/gynae.2025.v9.i2b.1601

Abstract
Introduction: Posterior Reversible Encephalopathy Syndrome (PRES) is a reversible clinicoradiological condition characterized by vasogenic edema, predominantly in the posterior cerebral hemispheres, and is frequently associated with hypertensive disorders in obstetrics, such as preeclampsia and eclampsia. Its recognition is critical due to its potential for significant maternal morbidity if untreated.
Objectives: This study aimed to investigate the pathophysiology, clinical presentation, diagnostic features, and management outcomes of PRES in obstetric patients, with a focus on a small cohort (n = 9) over a two-year period, to enhance understanding and guide clinical practice in this high-risk population.
Methods: A retrospective observational study was conducted at a tertiary obstetric center from March 2023 to March 2025. Nine pregnant or postpartum women diagnosed with PRES via clinical symptoms (e.g., seizures, headache) and MRI-confirmed bilateral posterior hyperintensities were included. Data on demographics, clinical features, laboratory findings, treatments, and outcomes were analyzed descriptively due to the small sample size.
Results: All 9 patients had preeclampsia or eclampsia, with seizures (77.8%), headache (66.7%), and visual disturbances (55.6%) as predominant symptoms. Mean systolic blood pressure at diagnosis was 172±15 mmHg. Management with antihypertensives (labetalol or hydralazine) and magnesium sulfate achieved symptom resolution within a median of 5 days (IQR: 3-7 days), with radiographic resolution in 8 patients (88.9%) at 4-6 weeks. One patient exhibited persistent mild memory deficits.
Discussion: Findings align with PRES's association with hypertensive crises in obstetrics, highlighting seizures as a key feature and the efficacy of standard therapies. The small cohort limits generalizability, but outcomes suggest reversibility with prompt intervention, though long-term effects warrant further study.
Conclusion: PRES in obstetrics is manageable with early diagnosis and treatment, emphasizing the need for vigilance and multidisciplinary care. Larger studies are needed to refine predictive and therapeutic approaches.

Keywords: Posterior reversible encephalopathy syndrome, PRES, obstetrics, preeclampsia, eclampsia, seizures, MRI, magnesium sulfate, maternal outcomes

Introduction
Posterior Reversible Encephalopathy Syndrome (PRES), first delineated by Hinchey *et al*. in 1996, is a reversible neurotoxic condition associated with acute disruptions in cerebral autoregulation and endothelial integrity [1]. Characterized by symptoms such as headache, seizures, visual disturbances, and altered mental status, PRES has emerged as a critical concern in obstetrics due to its frequent association with hypertensive crises during pregnancy and the postpartum period. Preeclampsia and eclampsia, affecting 5-8% of pregnancies globally, are primary precipitants, with PRES reported in up to 92% of eclamptic patients with neurological symptoms [2]. The syndrome's reversible nature underscores the importance of timely diagnosis and intervention, yet its overlap with other neurologic emergencies complicates management in obstetric settings. This article synthesizes current knowledge on PRES, augmented by a small-scale study (n = 9) conducted over two years, to elucidate its pathophysiology, clinical features, diagnostic approaches, and therapeutic strategies in the obstetric context.

Pathophysiology
The pathogenesis of PRES remains incompletely elucidated, with two dominant hypotheses shaping the discourse: the hyperperfusion theory and the hypoperfusion theory.

The hyperperfusion theory posits that acute hypertension surpasses the upper limit of cerebral autoregulation (typically 150-160 mmHg mean arterial pressure), leading to blood-brain barrier disruption and vasogenic edema, particularly in posterior watershed zones [3]. Conversely, the hypoperfusion theory suggests that endothelial dysfunction-driven by systemic inflammation, hypoxia, or cytotoxic injury-induces vasoconstriction, hypoperfusion, and secondary edema [4].

In obstetrics, preeclampsia and eclampsia amplify these mechanisms through placental ischemia and systemic endothelial injury. Elevated anti-angiogenic factors, such as soluble fms-like tyrosine kinase-1 (sFlt-1), and reduced placental growth factor (PlGF) disrupt vascular homeostasis, increasing permeability [5]. Pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), further exacerbate this cascade, particularly in severe preeclampsia [6]. Magnesium sulfate, a cornerstone of eclampsia management, may mitigate PRES severity by stabilizing endothelial function and lowering seizure thresholds, though its precise role remains under investigation [7]. Genetic factors, such as polymorphisms in the VEGF gene, have also been implicated as potential risk modifiers, suggesting a multifactorial etiology [8].

Clinical Presentation

PRES in obstetric patients typically manifests in the third trimester or early postpartum period, aligning with the peak incidence of hypertensive disorders. The clinical triad includes:

- **Headache:** Severe, bilateral, and often refractory to analgesia, reflecting cerebral edema and pressure changes.
- **Seizures:** Generalized tonic-clonic seizures occur in 70-90% of eclampsia-associated PRES cases, often as the presenting symptom [2].
- **Visual Disturbances:** Symptoms range from blurred vision to cortical blindness, attributed to occipital lobe involvement.

Additional signs, such as confusion, lethargy, or focal deficits, may mimic stroke or encephalopathy of other etiologies. In pregnancy, PRES frequently coexists with preeclampsia or eclampsia, though rare triggers like immunosuppressive therapy (e.g., cyclosporine) or autoimmune diseases may apply in non-obstetric contexts [3]. The overlap with HELLP syndrome or cerebral venous sinus thrombosis necessitates careful differentiation.

Diagnostic Evaluation: Diagnosis of PRES integrates clinical assessment with neuroimaging. Magnetic Resonance Imaging (MRI) is the gold standard, revealing bilateral, symmetrical hyperintensities on T2-weighted and Fluid-Attenuated Inversion Recovery (FLAIR) sequences, predominantly in the parietal and occipital lobes [9]. Diffusion-Weighted Imaging (DWI) typically shows no restriction, distinguishing PRES from ischemic stroke, though cytotoxic edema may appear in 10-15% of cases [10]. Computed Tomography (CT) may detect hypodensities in emergencies but lacks MRI's sensitivity.

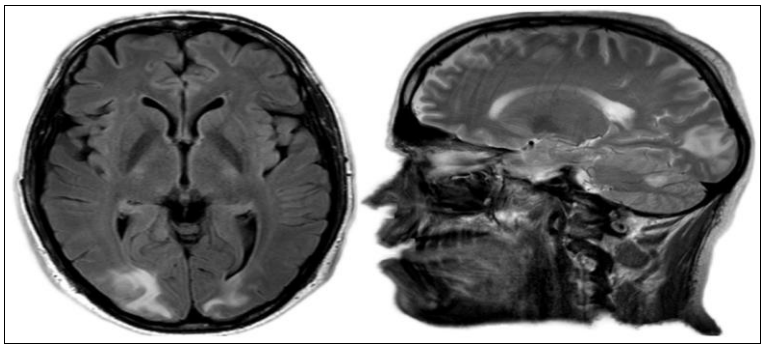

Fig 1: Axial FLAIR MRI sequence and Saggital T2 MRI sequence showing PRES.

In obstetric patients, laboratory markers of preeclampsia-proteinuria (>300 mg/24h), thrombocytopenia (<100,000/µL), and elevated transaminases-support the diagnosis [11]. The sFlt-1/PlGF ratio, a biomarker of preeclampsia severity, may predict PRES risk, though its role requires further validation [12]. Differential diagnoses, including reversible cerebral vasoconstriction syndrome (RCVS) and hemorrhagic stroke, must be excluded via imaging and clinical correlation.

Objectives

This study aimed to investigate the pathophysiology, clinical presentation, diagnostic features, and management outcomes of PRES in obstetric patients, with a focus on a small cohort (n = 9) over a two-year period, to enhance understanding and guide clinical practice in this high-risk population.

Methods

Study Design and Population

This retrospective observational study was conducted at a tertiary care obstetric center from March 1, 2023, to March 1, 2025. Eligible participants were pregnant or postpartum women diagnosed with PRES based on clinical symptoms (e.g., seizures, headache, visual changes) and radiological confirmation. Inclusion criteria included: (1) age 18-45 years, (2) MRI evidence of bilateral posterior hyperintensities on T2/FLAIR sequences, and (3) presentation during pregnancy or within 6 weeks postpartum. Exclusion criteria encompassed alternative neurologic diagnoses (e.g., stroke, venous thrombosis) confirmed by imaging or follow-up. Nine patients met these criteria over the two-year period.

Data Collection: Data were extracted from electronic medical records, including demographics (age, gestational age, parity), clinical features (symptoms, blood pressure), laboratory results (proteinuria, platelet count, liver enzymes), and MRI findings. Treatment details-antihypertensive agents, magnesium sulfate use, and delivery timing-were recorded. Outcomes assessed included time to symptom resolution, radiographic improvement on follow-up MRI (4-6 weeks post-diagnosis), and maternal complications (e.g., neurologic deficits). Data were de-identified and stored in a secure database per institutional ethical guidelines.

Statistical Analysis
Due to the small sample size (n = 9), descriptive statistics were employed. Continuous variables (e.g., blood pressure, time to resolution) were reported as means±standard deviations (SD) or medians with interquartile ranges (IQR), based on normality assessed via the Shapiro-Wilk test. Categorical variables (e.g., seizure occurrence, delivery mode) were expressed as frequencies and percentages. No inferential analyses were performed given the cohort size. Data were analyzed using SPSS version 28.0 (IBM Corp., Armonk, NY).

Ethical Considerations
The study received approval from the Institutional Review Board. Informed consent was waived due to the retrospective design and use of electronic and paper data.

Results
Patient Characteristics
Nine women were diagnosed with PRES over the two-year period, representing an incidence of 0.3% among 3,000 deliveries. The mean age was 29.4±5.2 years (range: 22-38 years). Six patients (66.7%) were primiparous, and 7 (77.8%) presented in the third trimester (median gestational age: 34 weeks, IQR: 32-36 weeks), while 2 (22.2%) presented postpartum (days 3 and 5). All had preeclampsia (n = 7) or eclampsia (n = 2), with hypertension and proteinuria confirmed in each case.

Clinical and Laboratory Findings
Seizures were the most frequent symptom (n = 7, 77.8%), followed by headache (n = 6, 66.7%) and visual disturbances (n = 5, 55.6%). Mean systolic blood pressure at diagnosis was 172±15 mmHg (range: 150-195 mmHg), and diastolic was 108±12 mmHg (range: 90-130 mmHg). Laboratory findings included proteinuria in all patients (median: 1,200 mg/24h, IQR: 800-2,500 mg/24h), thrombocytopenia in 3 (33.3%), and elevated AST (>70 U/L) in 4 (44.4%). MRI confirmed PRES in all cases, with bilateral occipital and parietal hyperintensities; no DWI restriction was observed.

Management and Outcomes
Antihypertensive therapy was administered to all patients: labetalol (n = 6, 66.7%) or hydralazine (n = 3, 33.3%), achieving target blood pressure within a median of 4 hours (IQR: 2-6 hours). Magnesium sulfate was given to 8 patients (88.9%)-all 7 with seizures and 1 prophylactically-using a 4 g loading dose and 1-2 g/hour infusion for 24-48 hours. Delivery occurred in the 7 antepartum cases (5 cesarean, 2 vaginal) within 24 hours of diagnosis.

Symptoms resolved within a median of 5 days (IQR: 3-7 days), with radiographic resolution on follow-up MRI in 8 patients (88.9%) at 4-6 weeks. One patient (11.1%), a 32-year-old with eclampsia, showed persistent occipital hypointensity and mild memory difficulties at 3 months. Two patients (22.2%) required ICU admission for refractory hypertension and seizures, but no deaths occurred.

Management
Management of PRES in obstetrics focuses on addressing the underlying trigger and preventing complications:

1. **Blood Pressure Control:** Labetalol (20-40 mg IV every 10-15 minutes, max 300 mg) or hydralazine (5-10 mg IV every 20-30 minutes) targets systolic blood pressure of 140-150 mmHg [13]. Nicardipine infusion is an alternative for refractory cases.
2. **Seizure Management:** Magnesium sulfate (4-6 g IV loading dose, 1-2 g/hour infusion) is standard for eclampsia-related PRES, with adjuncts like lorazepam if needed [14].
3. **Supportive Care:** ICU monitoring may be required for severe cases, with fluid balance and airway management prioritized.
4. **Delivery:** Antepartum PRES prompts expedited delivery post-stabilization, guided by maternal and fetal status [13].

Resolution typically occurs within days to weeks, though delays may lead to permanent deficits in 10% of cases [15].

Discussion
PRES represents a critical intersection of obstetric and neurologic pathology, with preeclampsia and eclampsia as dominant triggers. This study's findings align with prior reports, showing seizures as the predominant symptom (77.8% vs. 70-90% in literature [2]) and high recovery rates with prompt intervention. The small sample (n = 9) limits generalizability but reflects the rarity of PRES, even in a tertiary center. The sFlt-1/PlGF ratio's potential as a predictive biomarker warrants exploration, as does the long-term impact of PRES, given one patient's persistent deficits [12] [16].

Magnesium sulfate's efficacy in this cohort supports its dual role in eclampsia and PRES management, though its mechanism remains speculative [7]. Diagnostic challenges persist due to overlap with conditions like RCVS, emphasizing the need for MRI. Future research should focus on larger cohorts, biomarker validation, and postpartum neurocognitive outcomes.

Conclusion
PRES is a reversible yet potentially life-threatening complication in obstetrics, closely tied to hypertensive disorders. This study of 9 cases over two years underscores the importance of early diagnosis via MRI and management with antihypertensives and magnesium sulfate. Multidisciplinary collaboration is essential to optimize maternal outcomes, with ongoing research needed to refine prevention and treatment strategies.

Conflict of Interest: None.

References
1. Hinchey J, Chaves C, Appignani B, *et al*. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334(8):494-500. DOI: 10.1056/NEJM199602223340803
2. Brewer J, Owens MY, Wallace K, *et al*. Posterior reversible encephalopathy syndrome in 46 of 47 patients with eclampsia. Am J Obstet Gynecol. 2013;209(5):468.e1-468.e6. DOI: 10.1016/j.ajog.2013.06.015
3. Fugate JE, Rabinstein AA. Posterior reversible encephalopathy syndrome: Clinical and radiological manifestations, pathophysiology, and outstanding questions. Lancet Neurol. 2015;14(9):914-25. DOI: 10.1016/S1474-4422(15)00111-8
4. Bartynski WS. Posterior reversible encephalopathy syndrome, part 2: Controversies surrounding pathophysiology of vasogenic edema. AJNR Am J Neuroradiol. 2008;29(6):1043-1049. DOI: 10.3174/ajnr.A0929
5. Levine RJ, Maynard SE, Qian C, *et al*. Circulating angiogenic factors and the risk of preeclampsia. N Engl J

Med. 2004;350(7):672-683. DOI: 10.1056/NEJMoa031975
6. Euser AG, Cipolla MJ. Magnesium sulfate for the treatment of eclampsia: A brief review. Stroke. 2009;40(4):1169-1175. DOI: 10.1161/STROKEAHA.108.527788
7. Stevens AM, Ray DW, Zeggini E, *et al*. Genetic variants in the VEGF pathway as potential mediators of preeclampsia susceptibility. Hypertension. 2012;60(3):e18-e19. DOI: 10.1161/HYPERTENSIONAHA.112.196576
8. McKinney AM, Short J, Truwit CL, *et al*. Posterior reversible encephalopathy syndrome: Incidence of atypical regions of involvement and imaging findings. AJR Am J Roentgenol. 2007;189(4):904-912. DOI: 10.2214/AJR.07.2024
9. Lamy C, Oppenheim C, Mas JL. Posterior reversible encephalopathy syndrome. Handb Clin Neurol. 2014;121:1687-1701. DOI: 10.1016/B978-0-7020-4088-7.00109-8
10. Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. Obstet Gynecol. 2003;102(1):181-192. DOI: 10.1016/S0029-7844(03)00475-7
11. Zeisler H, Llurba E, Chantraine F, *et al*. Predictive value of the sFlt-1:PlGF ratio in women with suspected preeclampsia. N Engl J Med. 2016;374(1):13-22. DOI: 10.1056/NEJMoa1414838
12. American College of Obstetricians and Gynecologists. Hypertension in pregnancy. Obstet Gynecol. 2013;122(5):1122-1131. DOI: 10.1097/01.AOG.0000437382.03963.88
13. Duley L, Gülmezoglu AM, Henderson-Smart DJ, *et al*. Magnesium sulphate and other anticonvulsants for women with pre-eclampsia. Cochrane Database Syst Rev. 2010;(11):CD000025. DOI: 10.1002/14651858.CD000025.pub2
14. Roth C, Ferbert A. Posterior reversible encephalopathy syndrome: Long-term follow-up. J Neurol Neurosurg Psychiatry. 2011;82(7):773-777. DOI: 10.1136/jnnp.2010.226530
15. Postma IR, Bouma A, Ankersmit IF, *et al*. Neurocognitive functioning following preeclampsia and eclampsia: A long-term follow-up study. Am J Obstet Gynecol. 2014;211(1):37.e1-37.e9. DOI: 10.1016/j.ajog.2014.01.042

How to Cite This Article
Barbhuiya SM, Ali SKA. Posterior reversible encephalopathy syndrome in obstetrics: Pathophysiology, diagnosis, and management considerations. International Journal of Clinical Obstetrics and Gynaecology. 2025;9(2):100-103.