RESEARCH PAPER

Posterior Reversible Encephalopathy Syndrome (Pres) In Pregnancy With Eclampsia: A Case Report With Literature Review.

Jana Harshitha Sree Chandana[1], M C Metgud[2*], Kamal Patil [3], Mahadevi Savanur[4], Vinutha Hanji[5], Anusha Malapure[6]

[1]PG, JR 3, MS Obstetrics and Gynaecology, Jawaharlal Nehru Medical College, Belagavi
Email ID - jana.chandana26@gmail.com
[2]Professor, Department of Obstetrics and Gynaecology, Jawaharlal Nehru Medical College , Belagavi.
Email ID - metm67@gmail.com
[3]Professor, Department of Obstetrics and Gynaecology, Jawaharlal Nehru Medical College, Belagavi.
Email ID-kamalpatil1967@yahoo.co.in
[4]Associate Professor, Department of Obstetrics and Gynaecology, Jawaharlal Nehru Medical College, Belagavi.
Email ID- drdevim30@gmail.com
[5]Assistant Professor, Department of Obstetrics and Gynaecology, Jawaharlal Nehru Medical College, Belagavi. Email ID- vinuhanji@gmail.com
[6]Assistant Professor, Department of Obstetrics and Gynaecology, Jawaharlal Nehru Medical College, Belagavi.
Email ID- anushamalapure@gmail.com

ABSTRACT

Background: Posterior reversible encephalopathy syndrome (PRES) is an uncommon but serious neurological complication associated with severe preeclampsia and eclampsia. It is characterized by seizures, altered sensorium, visual disturbances, and characteristic neuroimaging findings involving predominantly the parieto-occipital regions of the brain. Early diagnosis and prompt management are essential to prevent irreversible neurological injury and maternal mortality.

Case Presentation: We report a case of a primigravida with eclampsia who underwent emergency lower segment caesarean section because of worsening maternal condition and recurrent generalized tonic–clonic seizures. Following surgery, the patient developed multiple recurrent seizure episodes associated with severe uncontrolled hypertension and neurological deterioration requiring intensive care admission. Mechanical ventilation and sedative support with midazolam infusion were initiated because of persistent ictal activity and risk of airway compromise. Neuroimaging with contrast computed tomography of the brain demonstrated bilateral fronto-parietal hypodensities suggestive of posterior reversible encephalopathy syndrome. The patient received multidisciplinary management including magnesium sulphate infusion, intravenous antiepileptic therapy, antihypertensive infusions, osmotherapy, ventilatory support, and continuous neurological monitoring. Gradual clinical improvement was observed with successful seizure control, stabilization of blood pressure, and recovery of neurological status. The patient was eventually extubated and discharged in stable condition with advice for neurological and obstetric follow-up.

Conclusion: This case highlights the importance of early recognition of PRES in patients with antepartum eclampsia presenting with recurrent seizures and hypertensive emergency. Prompt neuroimaging, aggressive seizure management, strict blood pressure control, and multidisciplinary intensive care are essential for favourable maternal neurological outcomes and prevention of permanent neurological complications.

Keywords: Posterior reversible encephalopathy syndrome; PRES; Eclampsia; Generalized tonic–clonic seizures; Postpartum seizures; Hypertensive encephalopathy;; Neurocritical care; Magnesium sulphate; Obstetric emergency.

How to cite this article: Chandana JHS, Metgud MC, Patil K, Savanur M, Hanji V, Malapure A. Posterior Reversible Encephalopathy Syndrome (Pres) In Pregnancy With Eclampsia: A Case Report With Literature Review. Int J Drug Deliv Technol. 2026;16(50s): 855-861. DOI: 10.25258/ijddt.16.50s.90

INTRODUCTION

Eclampsia is a severe and potentially life-threatening obstetric emergency characterized by the occurrence of generalized tonic–clonic seizures in a woman with preeclampsia that cannot be explained by other neurological conditions (1). It remains one of the major causes of maternal and perinatal morbidity and mortality worldwide, particularly in developing countries (1). Preeclampsia is a multisystem disorder of pregnancy associated with hypertension and end-organ dysfunction occurring after 20 weeks of gestation (2). When left untreated or inadequately controlled, it may progress to eclampsia, leading to serious maternal complications such as cerebrovascular accidents, pulmonary edema, renal failure, coagulopathy, and neurological dysfunction (3). Although eclampsia commonly occurs during the antenatal period, postpartum eclampsia is increasingly recognized as a significant clinical entity requiring urgent diagnosis and treatment (4). Posterior reversible encephalopathy syndrome (PRES) is an

*Author for Correspondence: jana.chandana26@gmail.com

important neurological complication associated with severe preeclampsia and eclampsia (5,6). PRES is a clinic-radiological syndrome characterized by headache, altered mental status, seizures, visual disturbances, and characteristic neuroimaging findings of vasogenic edema involving predominantly the parieto-occipital regions of the brain (7). The exact pathophysiology is not completely understood, but endothelial dysfunction, cerebral autoregulatory failure, vasospasm, and abrupt severe hypertension are considered major contributing mechanisms (8). Early recognition of PRES is crucial because delayed diagnosis may result in irreversible neurological damage, intracranial haemorrhage, or death. Fortunately, with prompt treatment and blood pressure control, most patients demonstrate complete clinical and radiological recovery (9).

Neuroimaging plays a vital role in the diagnosis of PRES. Computed tomography (CT) and magnetic resonance imaging (MRI) typically reveal symmetrical hypodense or hyperintense lesions involving the posterior cerebral white matter, most commonly in the parieto-occipital regions and can also involve other areas like frontal, temporal lobes , cerebellum , brain stem and basal ganglia. MRI remains the gold standard for diagnosis; however, CT imaging may provide early supportive evidence in emergency settings (10).

Management of eclampsia complicated by PRES requires a multidisciplinary approach involving obstetricians, neurologists, intensivists, anaesthesiologists, and cardiologists (11). Immediate seizure control using magnesium sulphate and antiepileptic agents, aggressive blood pressure management, airway protection, ventilatory support when required, and close neurological monitoring are essential components of treatment (12).

The present case report describes a patient with eclampsia who developed recurrent generalized tonic–clonic seizures and radiological features suggestive of PRES following emergency lower segment caesarean section. The case highlights the importance of early diagnosis, neurocritical care, and multidisciplinary management in improving maternal outcomes.

CASE PRESENTATION

Patient Information and Clinical Background: A 28 year old primigravida at 32 weeks period of gestation was admitted in the labour room with eclampsia. The patient had severe hypertension associated with neurological manifestations in the form of recurrent generalized tonic–clonic seizures. Because of worsening maternal condition and unfavourable cervix, the patient underwent emergency lower segment caesarean section for maternal stabilization and safe delivery of the fetus. The pregnancy was complicated by severe hypertensive disorder with subsequent neurological involvement requiring multidisciplinary critical care management.

The patient delivered a preterm female baby weighing 1.5 kg. Following delivery, the neonate was shifted to the neonatal intensive care unit for management of respiratory distress on CPAP (Continue positive airway pressure) and prematurity and low birth weight. Intra-operatively there was signs of abruption of placenta (GRADE 0) with a retroplacental clot . The placenta and membranes were delivered completely and appeared healthy. The uterus was adequately contracted and retracted after delivery, and haemostasis was achieved successfully. Estimated intraoperative blood loss was approximately 300 mL. The abdominal incision was closed in layers, and the patient tolerated the surgical procedure initially.

Immediate Postoperative Neurological Deterioration: Following surgery, the patient developed multiple episodes of generalized tonic–clonic seizures associated with severe uncontrolled hypertension. Blood pressure recordings reached critically elevated levels, suggestive of hypertensive emergency associated with eclampsia. The patient became neurologically unstable with recurrent ictal episodes requiring urgent intervention by the neurology and intensive care teams.

Because of persistent seizure activity, the patient was transferred to the intensive care unit for advanced monitoring and management. Clinical examination during the acute neurological event revealed ongoing ictal activity with severe hypertension and tachycardia. In view of recurrent seizures and risk of airway compromise, ventilatory support was initiated. The patient was connected to mechanical ventilation and sedative therapy was started with intravenous midazolam infusion for seizure suppression and neuroprotection.

Magnesium sulphate infusion was initiated as the primary anticonvulsant therapy for eclampsia. Intravenous antiepileptic medications including levetiracetam and lacosamide were administered for additional seizure control. Strict blood pressure management protocols were initiated simultaneously to prevent further neurological injury.

Neurological Evaluation and Neuroimaging Findings: Neurological consultation was obtained immediately because of recurrent seizures and altered sensorium. The neurology team advised aggressive anticonvulsant therapy, strict blood pressure regulation, neuroimaging evaluation, and close neurological monitoring. Recommendations included continuation of magnesium sulphate infusion, intravenous levetiracetam therapy, lacosamide administration, midazolam infusion, osmotherapy with mannitol, and corticosteroid administration with dexamethasone.

Computed tomography of the brain demonstrated ill-defined hypodense areas involving bilateral fronto-parietal regions. These findings were radiologically suggestive of posterior reversible encephalopathy syndrome (PRES) (Figures-1,2,3,4). Additional hypodense areas involving the anterior limb of the right internal capsule and right lentiform nucleus were also noted(Figures- 5). Magnetic resonance imaging of the brain was advised for further evaluation if clinically indicated.

The clinical presentation of recurrent seizures associated with severe hypertension and characteristic neuroimaging findings strongly supported the diagnosis of antepartum eclampsia complicated by PRES. Continuous neurological assessment and bedside monitoring were carried out throughout the intensive

care stay. Bedside electroencephalographic monitoring was also advised to evaluate ongoing cerebral activity and seizure control.

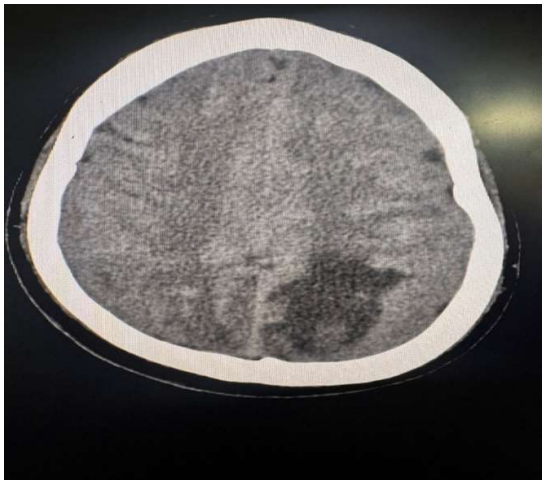

Fig 1: Plain and Contrast CT Brain Showing Bilateral fronto-Parietal Hypodensities Suggestive of Posterior Reversible Encephalopathy Syndrome (PRES) in Eclampsia

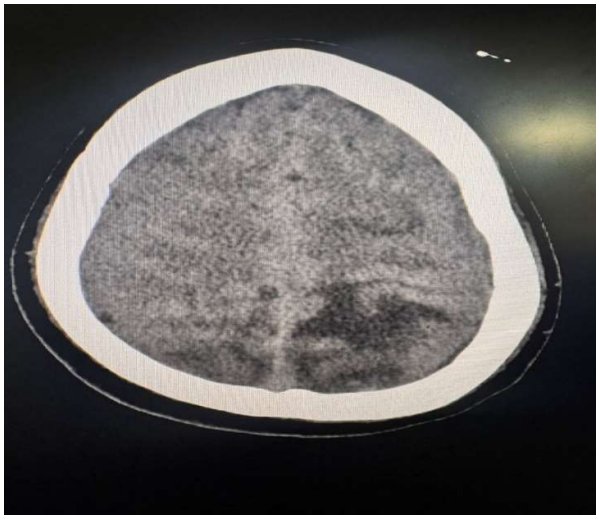

Fig 2: Axial Plain and Contrast CT Brain Image Demonstrating Bilateral Posterior Fronto-Parietal Hypodense Lesions Consistent with Posterior Reversible Encephalopathy Syndrome (PRES) Associated with Eclampsia

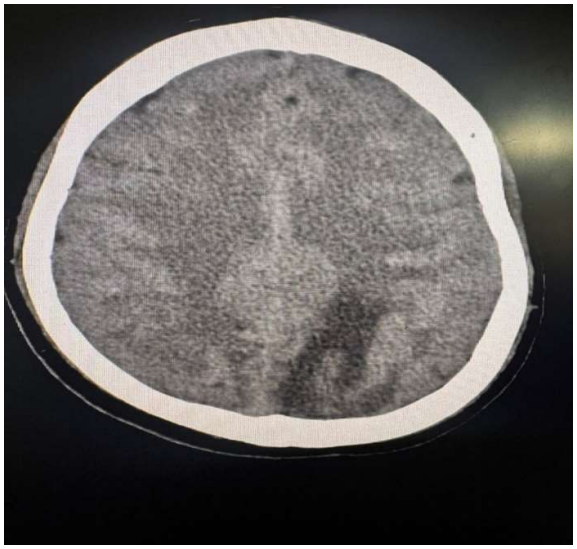

Fig 3: Axial CECT Brain Section Revealing Symmetrical Bilateral Fronto- parietal Hypodensities with Cortical–Subcortical Involvement Suggestive of Posterior Reversible Encephalopathy Syndrome (PRES) in Eclamptic Encephalopathy

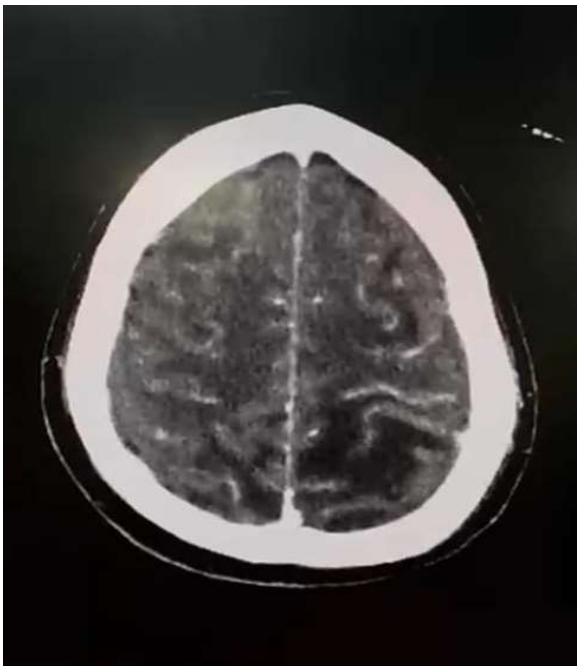

Fig 4: Axial Plain and Contrast CT Brain Image Showing Bilateral Cortical and Subcortical Fronto-Parietal Hypodensities with Edematous Changes Suggestive of Posterior Reversible Encephalopathy Syndrome (PRES) Secondary to Eclampsia

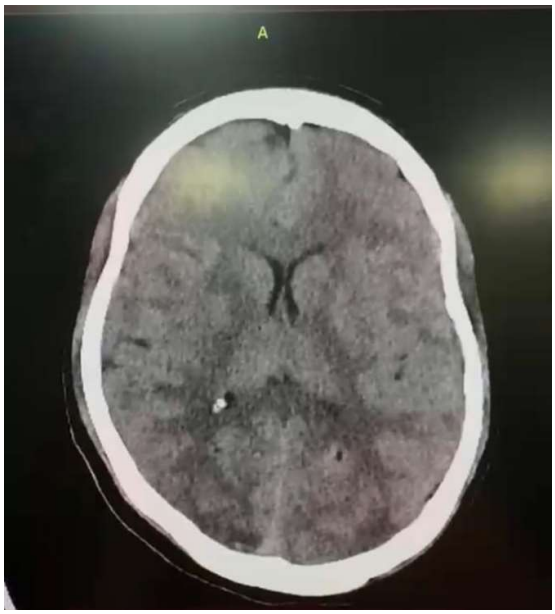

Fig 5: Plain and CECT Brain Image Demonstrating Hypodense areas involving anterior limb of right internal capsule and right lentiform nucleus,Characteristic of Posterior Reversible Encephalopathy Syndrome (PRES) in Severe Eclampsia

Intensive Care Management: The patient required prolonged intensive care management because of recurrent seizures, hypertensive emergency, and neurological instability. Continuous infusion of antihypertensive medications was initiated for blood pressure stabilization. Nitro-glycerine infusion and labetalol infusion were administered with titration according to blood pressure recordings. The target blood pressure was maintained below critical hypertensive levels to prevent progression of cerebral edema and neurological complications.

Sedation was maintained using midazolam infusion because of ongoing seizure activity. Magnesium sulphate infusion was continued as part of standard eclampsia management. Additional antiepileptic therapy with levetiracetam and lacosamide was continued throughout the hospital stay. Mannitol infusion was administered to reduce cerebral edema and intracranial pressure.

The patient initially required ventilatory support because of recurrent seizures and altered sensorium. Gradual neurological improvement allowed weaning from ventilatory support, after which CPAP trial was initiated successfully. Continuous Glasgow Coma Scale monitoring, respiratory monitoring, hemodynamic assessment, and neurological evaluation were performed during recovery.

Supportive intensive care measures included intravenous fluids, electrolyte correction, nutritional supplementation, physiotherapy, spirometry, nebulization therapy, and stress ulcer prophylaxis. Broad-spectrum antibiotic coverage with piperacillin-tazobactam was initiated to prevent secondary infections. Anticoagulant prophylaxis with low molecular weight heparin was also administered during hospitalization.

Cardiovascular Assessment: During hospitalization, the patient developed episodes of bradycardia requiring cardiology evaluation. Cardiology consultation and echocardiographic assessment were performed to evaluate cardiac function and exclude structural abnormalities. Echocardiography demonstrated preserved left ventricular systolic function with an ejection fraction of approximately 60%. No regional wall motion abnormalities were identified. Mild valvular abnormalities including trivial mitral regurgitation and trivial tricuspid regurgitation were noted without significant hemodynamic compromise. No active cardiac intervention was considered necessary, and conservative monitoring was advised.

Blood pressure management remained an important component of treatment because severe hypertension was considered a major contributing factor for neurological complications and PRES. Oral antihypertensive medications including clonidine and nicardipine were gradually introduced as the patient stabilized clinically.

Laboratory Investigations: Comprehensive laboratory investigations were performed throughout hospitalization to assess hematological, renal, hepatic, coagulation, and metabolic status.

Hematological investigations revealed fluctuating leukocytosis (TLC count – 20.2k) with neutrophilic predominance(Neutrophils count – 92) suggestive of stress response and inflammatory activation associated with eclampsia and critical illness. Peripheral smear examination showed neutrophilic leukocytosis without toxic granules. Platelet counts remained within acceptable limits (Platelet count – 1,51,000) and platelet morphology was reported as normal. Hemoglobin levels showed mild postoperative reduction (Hb-9.0 gms) but remained hemodynamically acceptable throughout the hospital course.

Renal function tests demonstrated preserved renal function with normal creatinine (Creatinine- 0.95) levels. Serum urea was mildly elevated (Urea levels-33.7 mg/dl) during the acute phase. Electrolyte monitoring revealed variations in potassium, magnesium, sodium, chloride, bicarbonate, and calcium levels, all of which were corrected appropriately during intensive care management. Serum magnesium levels were closely monitored because of therapeutic magnesium sulphate administration.

Liver function tests demonstrated mild elevation of transaminases (AST -88U/L and ALT- 86 U/L) with elevated lactate dehydrogenase levels(550 U/L). Serum albumin (2.8g/dl) and total protein levels (5.2 g/dl)were reduced, likely secondary to systemic illness, inflammatory response, and nutritional compromise. Bilirubin levels remained within normal limits.

Coagulation profile including PT/INR, activated partial thromboplastin time, thrombin time, fibrinogen levels, and D-dimer assessment remained largely within acceptable ranges. Elevated D-dimer levels(890 ng/dl) were noted during the acute illness phase, likely reflecting systemic inflammatory and hypercoagulable response associated with eclampsia and postoperative state.

Urine examination demonstrated proteinuria(urine albumin =2+) consistent with hypertensive disease of pregnancy. Additional urinary parameters including glucose, ketone bodies, nitrite, casts, and crystals were unremarkable. Microscopic haematuria was present without significant pyuria.

Serological screening investigations including HIV, hepatitis B surface antigen, and VDRL were non-reactive.

Recovery Phase and Clinical Improvement: With aggressive multidisciplinary management, the patient gradually showed neurological and hemodynamic improvement. Seizure frequency reduced progressively with anticonvulsant therapy and blood pressure stabilization. Midazolam infusion was gradually tapered and discontinued as seizure control improved. Antihypertensive infusions were converted to oral medications once the patient achieved stable blood pressure recordings.

The patient was successfully extubated and maintained satisfactory oxygen saturation on room air. General physical examination during recovery showed bilateral pedal edema without pallor or icterus. Breast examination revealed soft, non-tender, secretory breasts consistent with postpartum physiological changes. Abdominal examination demonstrated a well-involuted uterus with healthy postoperative wound dressing and absence of active bleeding. Lochia remained healthy throughout the recovery period.

The patient tolerated oral medications and nutritional supplementation well. Ambulation and supportive rehabilitation measures were gradually initiated. Neurology team advised continuation of oral antiepileptic medications after discharge along with close neurological follow-up.

Discharge Status and Follow-Up Advice: At the time of discharge, the patient was conscious, oriented, and

hemodynamically stable with controlled blood pressure and no further seizure episodes. She was advised continuation of oral antiepileptic therapy with levetiracetam and lacosamide along with antihypertensive medications including nicardipine and clonidine-based therapy. Steroid tapering schedule and supportive medications including calcium supplementation, protein supplementation, nebulization therapy, and nutritional rehabilitation were advised.

Detailed discharge counselling was provided regarding warning symptoms including headache, seizures, abdominal pain, fever, vaginal bleeding, and neurological symptoms requiring immediate medical attention. The patient was advised strict blood pressure monitoring, fluid restriction, adequate rest, avoidance of strenuous physical activity, and regular follow-up in neurology and obstetric outpatient departments.

The final diagnosis was antepartum eclampsia complicated by posterior reversible encephalopathy syndrome with recurrent generalized tonic–clonic seizures requiring neurocritical intensive care management following emergency caesarean section for eclampsia and preterm delivery.

DISCUSSION

Posterior reversible encephalopathy syndrome (PRES) is an important neurological complication associated with severe preeclampsia and eclampsia and is characterized by seizures, altered sensorium, visual disturbances, and radiological evidence of vasogenic edema involving the posterior cerebral regions. In the present case, the patient developed recurrent generalized tonic–clonic seizures following emergency caesarean section performed for eclampsia, and neuroimaging findings were suggestive of PRES. Similar observations were described by Magley and Hinson (2024), who reported that eclampsia is a life-threatening obstetric emergency characterized by unexplained generalized seizures occurring in association with hypertensive disorders of pregnancy (1). The current patient demonstrated severe hypertension with recurrent seizures in the postpartum period, highlighting the neurological severity associated with eclampsia.

The pathophysiological mechanisms underlying PRES are believed to involve endothelial dysfunction, cerebral vasospasm, impaired autoregulation, and breakdown of the blood–brain barrier. These mechanisms were extensively discussed by Martini et al. (2025), who emphasized the role of systemic inflammatory activation and vascular endothelial injury in severe preeclampsia (3). Similar pathogenic mechanisms were evident in the present case, where uncontrolled hypertension likely contributed to cerebral edema and neurological deterioration. Fox et al. (2019) also noted that severe preeclampsia is associated with multiorgan endothelial dysfunction leading to complications involving the central nervous system, cardiovascular system, kidneys, and liver (2).

The postpartum period is increasingly recognized as a vulnerable phase for the development of eclampsia and neurological complications. According to Katsi et al. (2024), postpartum eclampsia may occur even after delivery and requires urgent seizure control and aggressive antihypertensive management to prevent maternal morbidity and mortality (4). In the current case, despite emergency caesarean section, the patient developed recurrent generalized tonic–clonic seizures with hypertensive emergency requiring intensive care admission and ventilatory support. Prompt initiation of magnesium sulphate infusion, antihypertensive therapy, and antiepileptic medications played a vital role in stabilizing the patient.

Radiological evaluation remains essential for confirming PRES. Classical neuroimaging findings include symmetrical vasogenic edema involving the parieto-occipital regions. Similar imaging characteristics were described by Gaillard et al. (2008) and further supported by Triplett et al. (2022), who identified bilateral posterior cerebral white matter involvement as the hallmark radiological feature of PRES (6,8). In the present case, computed tomography of the brain demonstrated bilateral fronto-parietal hypodensities consistent with PRES, correlating closely with the clinic-radiological descriptions available in the literature.

A retrospective review conducted by Shaikh et al. (2021) demonstrated that recurrent seizures and severe hypertension were among the most common presenting manifestations in patients with eclampsia-associated PRES (5). Their study also highlighted that early neuroimaging and timely critical care management significantly improve maternal neurological outcomes. Similar findings were observed in the present patient, where rapid neurological consultation, neuroimaging evaluation, and aggressive seizure management contributed to progressive recovery. Goyal and Jeswani (2022) additionally reported that seizures are the predominant neurological presentation in PRES patients, especially in hypertensive emergencies associated with pregnancy (7).

Early recognition and multidisciplinary management are essential because PRES is generally reversible if treated promptly. This concept was emphasized by Zelaya and Al-Khoury (2026), who stated that most patients recover completely with appropriate blood pressure control and seizure management (9). Mourid et al. (2026) further warned that delayed diagnosis may result in irreversible neurological injury, intracranial haemorrhage, or death (10). In the present case, timely neurocritical care management including magnesium sulphate therapy, osmotherapy, ventilatory support, and strict blood pressure regulation resulted in gradual neurological improvement and favourable maternal outcome without persistent neurological deficits.

Thus, the present case reinforces the strong association between severe antepartum eclampsia and PRES and highlights the importance of early diagnosis, prompt neuroimaging, aggressive seizure control, and multidisciplinary intensive care management in preventing adverse maternal neurological complications.

CONCLUSION

Posterior reversible encephalopathy syndrome (PRES) is a serious but potentially reversible neurological complication associated with severe preeclampsia and eclampsia. The present case highlights the occurrence of recurrent generalized tonic–clonic seizures in the antepartum and postpartum period following emergency caesarean section in a patient with severe preeclampsia. Early recognition of neurological deterioration, prompt neuroimaging evaluation, and aggressive multidisciplinary management were crucial in achieving favourable maternal recovery. Characteristic radiological findings involving bilateral fronto-parietal regions supported the diagnosis of PRES. Timely administration of magnesium sulphate, antiepileptic medications, antihypertensive therapy, ventilatory support, and neurocritical care measures contributed significantly to seizure control and neurological stabilization. This case emphasizes the importance of maintaining high clinical suspicion for PRES in antepartum eclamptic patients presenting with seizures and altered sensorium. Early diagnosis and intensive management can prevent irreversible neurological injury, reduce maternal morbidity, and improve overall clinical outcomes in such high-risk obstetric emergencies.

REFERENCES

1. Magley M, Hinson MR. Eclampsia. Treasure Island (FL): StatPearls Publishing; 2024. PMID: 32119279.
2. Fox R, Kitt J, Leeson P, Aye CYL, Lewandowski AJ. Preeclampsia: risk factors, diagnosis, management, and the cardiovascular impact on the offspring. J Clin Med. 2019;8(10):1625. doi:10.3390/jcm8101625
3. Martini C, Saeed Z, Simeone P, Palma S, Ricci M, Arata A, et al. Preeclampsia: insights into pathophysiological mechanisms and preventive strategies. Am J Prev Cardiol. 2025;23:101054. doi:10.1016/j.ajpc.2025.101054
4. Katsi V, Svigkou A, Dima I, Tsioufis K. Diagnosis and treatment of eclampsia. J Cardiovasc Dev Dis. 2024;11(9):257. doi:10.3390/jcdd11090257
5. Shaikh N, Nawaz S, Ummunisa F, Shahzad A, Hussain J, Ahmad K, et al. Eclampsia and posterior reversible encephalopathy syndrome (PRES): a retrospective review of risk factors and outcomes. Qatar Med J. 2021;2021(1):4. doi:10.5339/qmj.2021.4 .
6. Gaillard F, Sharma R, Silverstone L. Posterior reversible encephalopathy syndrome. Radiopaedia.org. 2008 May 2.
7. Goyal G, Jeswani J. Study of clinicoradiological profile in posterior reversible encephalopathy syndrome: an experience from North India. Indian J Crit Care Med. 2022;26(4):501-506. doi:10.5005/jp-journals-10071-24172
8. Triplett JD, Kutlubaev MA, Kermode AG, Hardy T. Posterior reversible encephalopathy syndrome (PRES): diagnosis and management. Pract Neurol. 2022;22(3):183-189. doi:10.1136/practneurol-2021-003194
9. Zelaya JE, Al-Khoury L. Posterior reversible encephalopathy syndrome. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026. PMID: 32119379.
10. Mourid MR, Oweidat M, Abady E, Shahid MA, Ata IM, Shaban NS, et al. Posterior reversible encephalopathy syndrome (PRES): a narrative review of pathophysiology, clinical insights, and advances in management. Int J Emerg Med. 2026;19(1):48. doi:10.1186/s12245-026-01138-9
11. Clift J. Eclampsia and pre-eclampsia. In: Core Topics in Critical Care Medicine. Cambridge: Cambridge University Press; 2010. p. 342-348.
12. Karunarathna I. Comprehensive management of eclampsia: an evidence-based clinical approach to seizure control, hypertensive crisis, and delivery optimization. Uva Clinical Research Lab - Eclampsia. 2025.