International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Sheetal B et al. Int J Reprod Contracept Obstet Gynecol. 2025 Jun;14(6):1744-1749
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20251459

Original Research Article

Posterior reversible encephalopathy syndrome: a diagnostic dilemma

Sheetal B.[1], Usha Doddamani[1], Sohail Shali[2], Pooja H. Sangundikar[1]*, Shradha[1]

[1]Department of Obstetrics and Gynaecology, Gulbarga Institute of Medical Sciences, Kalaburagi, Karnataka, India
[2]Department of Anaesthesia, Gulbarga, Institute of Medical Sciences, Kalaburagi, Karnataka, India

Received: 08 October 2024
Revised: 16 May 2025
Accepted: 17 May 2025

***Correspondence:**
Dr. Pooja H. Sangundikar,
E-mail: poojah7777@gmail.com

ABSTRACT

Background: Incidence of eclampsia 1 in 2000 to 3250. Seizures can occur antepartum, intrapartum, postpartum. PRES characterised by features like visual disturbances, headache, vomiting, seizures and altered sensorium .Hypertension major cause of PRES. Study aims incidence of PRES, asses clinical presentation, neuroimaging in PRES patients and maternal outcome.
Methods: PRES is neurological disorder characterized by symptoms like visual disturbances, headache, vomiting, seizures and altered sensorium. Eclampsia by definition have seizures as part of clinical syndrome. In our study, all patients diagnosed with PRES admitted in obstetric ICU from January 2023 to June 2023 at Gulbarga institute of medical sciences are included. They underwent CT scan for diagnosis of PRES. Abnormal findings included brain hypodense areas in posterior parietal lobe and occipital areas, posterior temporal lobes. To avoid misdiagnosis, it requires careful attention to clinical and radiographic presentation. Main criteria for PRES are Presence of neurologic symptoms or findings, presence of risk factors for PRES, absence of other possible causes of encephalopathy, Reversible course on follow up.
Results: Out of 22 patients admitted in obstetric ICU, 14 cases revealed PRES on neuroimaging. Eclampsia occurred in 20 antepartum and in 2 postpartum patients. Headache being predominant symptom, followed by altered mental status, visual disturbances.
Conclusions: Our study revealed the common finding of PRES in patients with eclampsia which suggested that PRES is a core component of pathogenesis of eclampsia in pregnancy. Hence early diagnosis and treatment is essential to avoid irreversible neurological damage and maternal morbidity and mortality.

Keywords: CT brain, Eclampsia, PRES, Pregnancy

INTRODUCTION

Incidence of eclampsia is 1 in 2000 to 3250, deliveries in developed countries and 1 to 5% in India.[1-4] Eclamptic seizures can occur antepartum, intrapartum or postpartum. Posterior reversible encephalopathy syndrome (PRES) was first described in 1996 by Hinchey et al.[5] PRES is a neurological disorder characterized by various symptoms such as visual disturbances, headaches, vomiting, seizures, and altered sensorium. Patients with eclampsia, by definition, have seizures as part of the clinical syndrome. However, patients with preeclampsia can also present with other neurological signs and symptoms. It was first named this syndrome reversible posterior leukoencephalopathy syndrome.[5] This name was superseded by 'posterior reversible encephalopathy syndrome' in 20006.

PRES has been associated with many conditions including eclampsia, severe hypertension, autoimmune disease, treatment with cytotoxic medications, post transplantation immunosuppression, and infection with sepsis to name a few. Generalized seizures are mostly common clinical features of PRES, but patients can present with signs of encephalopathy such as altered mental status, headache,

nausea, and vomiting.[7] Visual disturbances are also common, varying from mild blurry vision to complete cortical blindness.[8] Increased blood pressure is cause for majority of cases, But BP may be normal or only mildly increased in 20-30% of patients.[9] In the series of 36 patients presented by Lee et al, the mean SBP presented as 187 mmHg (range, 80–240 mmHg) within 24 hours.[8] In patients having PRES neuroimaging reports have been explained in scores of eclamptic patients since 1996 report of Hinchey et al.[5] Laboratory findings may differ, depending on pathology associated with condition Management includes symptomatic treatment including ICU care, control of hypertension, antiseizure measures and hemodynamic management. Although the syndrome is classically described as reversible entity with active management however permanent complications, mortalities and recurrence of symptoms have been reported. Hence, early diagnosis and prompt treatment is required for reduction of morbidity and mortality.[10]

Aims and objectives

To determine the incidence of PRES in patients with eclampsia. To assess clinical presentation and neuroimaging abnormalities in a series of patients admitted in obstetric ICU under eclampsia unit and its maternal outcome.

METHODS

Study type

This was a retrospective observational study.

In our study, all patients diagnosed with PRES admitted in obstetric ICU in the department of Obstetrics and Gynecology, Gulbarga Institute of Medical Sciences, Kalaburgi from January 2023 to June 2023 included in the study. They were subjected to CT scan for diagnosis of PRES. Data was gathered through a retrospective chart review of all patients diagnosed with PRES during this period. The research team reviewed all charts with PRES as diagnosis.

Inclusion criteria

All patients with diagnosis of eclampsia and those who developed PRES are included in the study.

Exclusion criteria

Patients with PRES with CVT, Acute infracts, Hemorrhage, Sepsis were excluded.

Abnormal findings included brain hypodense areas in posterior parietal lobe and occipital areas, posterior temporal lobes. Since it can be easily misdiagnosed, proper diagnosis requires careful attention to clinical and radiographic presentation. The main criteria for PRES are Presence of neurologic symptoms or findings, Presence of risk factors for PRES, Absence of other possible causes of encephalopathy, Reversible course on follow up.

Statistical analysis

The data collected was analysed statistically bt frequencies and percentages. Software used were Microsoft office 17 and SPSS 16.

RESULTS

Out of 22 patients admitted in obstetric ICU, 14 cases revealed PRES on neuroimaging. Eclampsia occurred in 20 antepartum patients and in 2 postpartum patients. Headache was most predominant symptom, followed by altered mental status, visual disturbances. No maternal mortality in both antepartum and post-partum cases.

Table 1 summarizes the incidence of PRES. The Chi-square test for PRES occurrence yielded a p value of 0.200, indicating no statistically significant difference in PRES prevalence. However, the comparison between antepartum and postpartum cases showed a highly significant association with a p-value of 0.00012, suggesting PRES was significantly more common in antepartum cases.

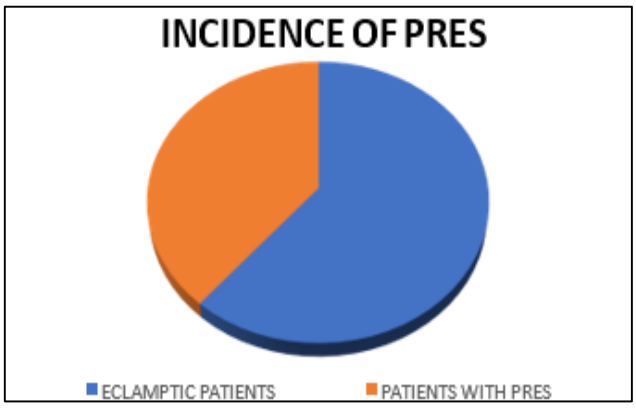

Figure 1: Incidence of PRES.

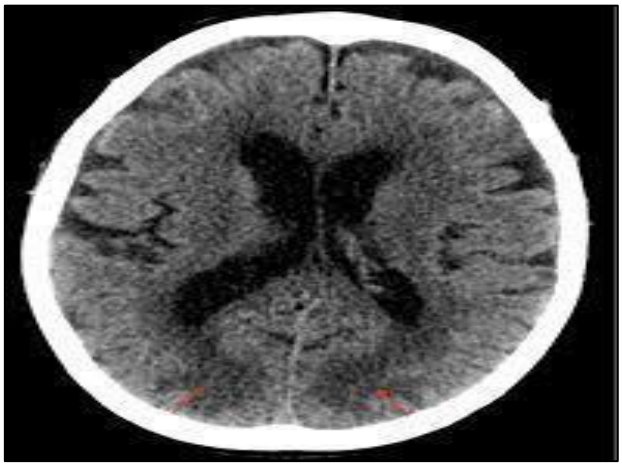

Figure 2: CT scan showing low attenuation of posterior cortical areas.

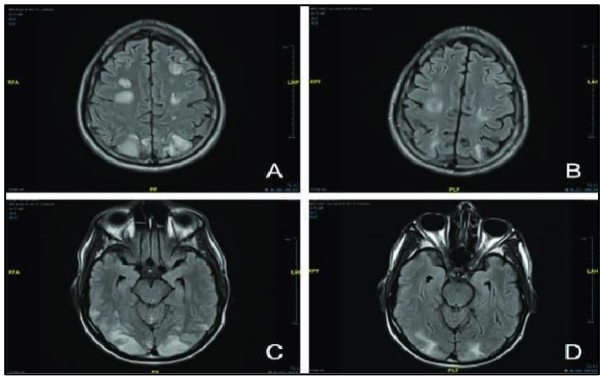

Figure 3 (A-D): MRI showing white matter hypodensities in parieto-occipital regions predominantly.

Figure 1 illustrates the distribution, confirming that 14 out of 22 eclamptic patients developed PRES. There were no maternal deaths recorded in either the antepartum or postpartum groups, indicating favourable outcomes despite the severity of symptoms. Table 2 details the distribution of clinical symptoms among patients. The most commonly reported symptom was headache and/or vomiting, noted in 21 out of 22 patients, followed by blurring of vision (20 patients), epigastric pain (16 patients), and anasarca (15 patients). All 22 patients presented with hypertension and proteinuria, and all experienced eclamptic seizures. Seizure frequency varied, with 17 patients experiencing 2-4 episodes, and 4 patients having more than 4 episodes. Notably, complications such as acute renal failure (ARF), pulmonary edema, disseminated intravascular coagulation (DIC), and encephalopathy were absent in all cases, suggesting effective early management. These findings indicate that seizures, headache/vomiting, hypertension, blurring of vision, and epigastric pain were the predominant clinical features in patients who developed PRES.

As shown in Table 3, Magnesium Sulphate ($MgSO_4$) was administered to all 22 patients as the first-line anti-seizure therapy. In 2 patients, Phenytoin was added to $MgSO_4$ to control seizures. This highlights the standard use of $MgSO_4$ in the management of eclamptic seizures, with additional agents used selectively. Figure 2 demonstrates a CT scan showing low attenuation in the posterior cortical areas, indicative of PRES-related edema. Figure 3 presents an MRI image revealing white matter hypodensities in the parieto-occipital regions, consistent with classic PRES findings. These imaging results reinforce the clinical diagnosis in patients presenting with neurological symptoms.

Table 1: Distribution of Incidence of PRES of our study participants.

PRES	Frequency
Positive	14
Negative	8
Chi square test	1.636
P value	0.200
Antepartum	20
Postpartum	02
Chi square test	14.727
P value	0.00012

Table 2: Distribution of Incidence of clinical feature of our study population.

Headache/ vomiting	21	Anemia	18
Epigastric pain	16	Abruption	2
Hypertension/proteinuria	22	ARF	0
Blurring of vision	20	Pulmonary oedema	0
Seizures-1 episode		DIC	0
2-4 episodes	17	Eclampsia	22
>4 episodes	4	PPH	15
Oliguria		HELLP	3
Anasarca	15	Encephalopathy	0

Table 3: Distribution of management of our study participants.

Treatment given	Number
MgSO4	22
Phenytoin	0
MgSO4+ Phenytoin	2

DISCUSSION

PRES in eclampsia is rare condition which is associated with headache, seizure, altered mental status and visual disturbances.

It can present with focal deficits , mimicking stroke. The major cause is severe pre-eclampsia and eclampsia. The main pathogenesis is inability of brain circulation to auto regulate in response to acute changes in blood pressure. When unrecognised irreversible cytotoxic oedema and damage can occur.

The cerebral pathology of patients with severe preeclampsia who suffer seizure(s) may be due to microangiopathy of the cerebral vessels, often with hypertension contributing. Microangiopathy and/or malfunction of the cerebral vasculature may be related to circulating levels of antiangiogenic factors such as soluble fms-like tyrosine-1 (sFlt-1) and soluble endoglin (sEng), which have been demonstrated to be at higher levels in patients with eclampsia compared with those with severe preeclampsia.[11,12]

In emergency cases, CT scan helps in rapid evaluation of patient. It also helps in excluding any cerebral haemorrhage or space occupying lesion. However, it is not 100% sensitive to diagnose PRES syndrome. MRI is the imaging modality of choice.[13] It is characterised by presence of high signal intensity in white matter of posterior regions of both cerebral hemispheres. It is seen predominantly in parietal and occipital lobes. In our patient, NCCT head was done which showed hypo densities in bilateral parietal occipital lobe. In study by McKinney et al, atypical MRI findings of PRES syndrome has been described.[14] 76 patients with PRES syndrome were evaluated on MRI. It was found that most common region involved was parietooccipital (98.7%) followed by posterior frontal, temporal, thalamus, cerebellum, brainstem and basal ganglia. Atypical manifestations were found as enhancement, restricted diffusion and haemorrhage.

Generalized seizures are often the most common clinical manifestation of PRES.[15] Headache is the most common symptom. Visual disturbances can range from blurring of vision to hemianopsia or even cortical blindness. However, unlike many other studies and case reports we observed that signs of imminent eclampsia may not be present in patients of eclampsia with PRES syndrome. Hemiparesis, dystonias are rare features. Sluggish pupillary reflexes or myosis can be seen. Papilledema and haemorrhagic changes on fundus examination can be signs of raised intracranial pressure. Recurrence is reported in 3.8% of cases in a recent series.[16]

Control of hypertension is considered a core management component for PRES treatment.[2] Antihypertensive treatment using intravenous labetalol, hydralazine, or nicardipine is usually recommended for the prevention of severe systolic hypertension of more than 160 mm Hg and/or severe diastolic hypertension of more than 105-110 mmHg.[17,18] Gradual correction to target levels of 140-155 mmHg systolic and 90-105 mm Hg diastolic are advisable to protect the mother and to avert compromised uteroplacental blood flow.

The rate of increase in BP is a more important factor for development of PRES than the absolute rise in BP. There is development of vasogenic brain oedema mostly in the posterior circulation as a result of breakdown of blood brain barrier.[19,20] An acute rise in BP over a short period of time causes failure of normal autoregulatory mechanisms that usually control blood flow to the brain in a hypertensive state.[21-23] Likewise patients with chronic hypertension are less likely to develop PRES as their circulation is adapted to higher levels of BP. PIH patients don't seem to enjoy this advantage where increase in BP to high levels occurs in relatively short periods. Another theory suggests that acute rise in BP causes hypoperfusion leading to endothelial damage secondary to hypoxia and subsequent cytotoxic oedema.

Sepsis and septic shock can precipitate PRES by endothelial derangement and microcirculatory disturbances. The brain imaging shows oedema of white matter and reversible ischaemic changes predominantly in areas perfused by posterior brain circulation, that is, the parietooccipital areas.[24] The findings include non-enhancing white matter abnormalities that appear as areas of low attenuation on CT. MRI is the investigation of choice. On MRI multifocal T2 hyperintensities are seen mainly in parietal and occipital cortex but can also involve other parts of the cortex, thalamus, basal ganglia, cerebellum and brain stem. Diffusion Weighted Images (DW1) and Apparent Diffusion Coefficient mapping (ADC) can differentiate between vasogenic and cytotoxic oedema.[25,26]

Ischaemic changes on DW1 and ADC images are associated with worse prognosis.[27] Focal regions of symmetric hemispheric oedemas are often seen. Haemorrhage, focal haematoma or isolated subarachnoid haemorrhage is seen in 15% of cases.[28] Prophylactic MgSo4 should be started on appearance of imminent symptoms. It is important to treat the aetiology or else this can progress to irreversible neurological sequelae even death. Blood pressure should be controlled by using I.V labetalol or oral nifedipine.[29] However, care should be taken not to cause a sudden hypotension as this may adversely affect the brain perfusion. An immediate reduction of mean arterial pressure by 20-25% in the first two hours should be aimed at followed by gradual reduction thereafter.

MgSo4 is the treatment of choice in eclampsia. If the patient is antenatal, delivery should be expedited. In a case of status eclamptic us I.V lorazepam is the recommended drug used along with invasive ventilation and other general measures. Treated in time, most patients exhibit complete

neurologic recovery within two weeks accompanied by resolution of the radiologic lesions. Prognosis is poorer if there is a large area of involvement or associated ischaemia or infarct. Obstructive hydrocephalus is another known complication. Apart from preeclampsia eclampsia and acute kidney injury PRES can be seen in a variety of other clinical conditions.

This study is short retrospective study. Could not find any limitations.

CONCLUSION

Our study revealed the common finding of PRES in patients with eclampsia which suggested that PRES is a core component of pathogenesis of eclampsia in pregnancy. Hence early diagnosis and treatment is essential to avoid irreversible neurological sequelle and maternal morbidity and mortality.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Ventura SJ, Martin JA, Sally C, MA. Division of Vital Statistics. Final date for 1998. National Vital Statistics Reports. 2000;48(3):56.
2. Pradeep MR, Lalitha S. Retrospective Study of Eclampsia in a Teaching Hospital. International J Rec Trend Sci and Technol. 2013;8(3):171-3.
3. Gupte S, Wagh G. Preeclampsia–Eclampsia. J Obstet Gynecol Ind. 2014;64(1): 4-13.
4. Anuja B, Sayali K, Sunita G, Anjali K, Sulabha J, Savita S. Eclampsia: Maternal and fetal outcome. J of South Asian Federation of Obstet Gynaecol. 2013;5(1):19-21.
5. Hinchey J, Chaves C, Appignani B,. J braeen, L Pao, A Wang, et al. reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334:494-500.
6. Casey SO, Sampaio RC, Michel E, Truwit CL. Posterior reversible encephalopathy syndrome: utility of fluid-attenuated inversion recovery MR imaging in the detection of cortical and subcortical lesions. American J Neuroradiol. 2000;21:1199-206.
7. Sraykov D, Schwab S. Posterior reversible encephalopathy syndrome. J Intensive Care Med. 2012;27:11-24.
8. Lee V, Wijdicks EFM, Manno E, Rabinstein A. Clinical spectrum of reversible posterior leukoencaphalopathy syndrome. Arch Neurol. 2008;65:205-10.
9. Bartynski WS. Posterior reversible encephalopathy syndrome, part 1: fundamental imaging and clinical features. AJNR Am J Neuroradiol. 2008;29:1036-42.
10. Rodrigo P, Paul EM, Joseph V. Posterior Reversible Encephalopathy Syndrome: A Review. Crit Care & Shock. 2009;12:135-43.
11. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334(8):494-500.
12. Vaisbuch E, Whitty JE, Hassan SS, Romero R, Kusanovic JP, David B Cotton, et al. Circulating angiogenic and antiangiogenic factors in women with eclampsia. Am J Obstet Gynecol. 2011;204:152.
13. Pedraza R, Marik PE, Varon J. Posterior reversible encephalopathy syndrome: a review. Critical Care & Shock. 2009;12(4):135–43.
14. McKinney AM, Short J, Truwit CL, McKinney ZJ, Kozak OS, SantaCruz KS, et al. Posterior reversible encephalopathy syndrome: incidence of atypical regions of involvement and imaging findings. AJR Am J Roentgenol. 2007;189:904-12.
15. Justin B, Michelle YO, Kedra W, Amanda AR, Rachael M, Majid K, et al. Posterior reversible encephalopathy syndrome in 46 of 47 patients with eclampsia. Am J Obstet Gynecol. 2013;208:468.
16. Sweany JM, Bartynski WS, Boardman JF. "Recurrent" posterior reversible encephalopathy syndrome: report of 3 cases—PRES can strike twice. J Comput Assist Tomogr. 2007;31(1):148–56.
17. Martin JN, Thigpen BD, Moore RC, Rose CH, Cushman J, May WL. Stroke and severe preeclampsia/eclampsia: a paradigm shift focusing on systolic blood pressure. Obstet Gynecol. 2005;105:246-54.
18. American College of Obstetricians and Gynecologists. Emergent therapy for acuteonset, severe hypertension with preeclampsia or eclampsia. ACOG Committee Opinion. 2011: 514.
19. Roth C, Ferbert A. Posterior reversible encephalopathy syndrome: long-term follow-up. J Neurol Neurosurg Psychiatry. 2010;81:773–7.
20. Karumanchi SA, Lindheimer MD. Advances in the understanding of eclampsia. Curr Hypertens Rep. 2008;10:305-12.
21. Xuan H, Jinfeng X, Zao C, Guoliang L, Hong J. Posterior reversible encephalopathy syndrome with involvement of the cervical cord and medulla: A case report. J Clin Diagn Res. 2015;9(1):51-2.
22. Ishikura K, Ikeda M, Hamasaki Y, Hataya H, Nishimura G, Hiramoto R, et al. Nephrotic state as a risk factor for developing posterior reversible encephalopathy syndrome in paediatric patients with nephrotic syndrome. Nephrol Dial Transplant. 2008;23(8):2531-36.
23. Cipolla MJ, Sweet JG, Chan SL. Cerebral vascular adaptation to pregnancy and its role in the neurological complications of eclampsia. J Applied Physiol. 2011;110(2):329–39.
24. Bartynski WS. Posterior Reversible Encephalopathy Syndrome, Part 1: Fundamental Imaging and Clinical Features. Am J Neuro Radiol. 2008;29(6):1036-42.

25. Patil V, Dhamangaonkar BR, Pattanshetti RC, Patil MM. Posterior reversible encephalopathy syndrome in early postpartum women: A case report. J Clin Diagnos Res. 2014;8(4):12.
26. Ibrahim NMAM, Manal EB. MRI imaging of posterior reversible encephalopathy syndrome associated with pregnancy. The Egyptian J Radiol Nuclear Med. 2014;45(2):505–10.
27. Covarrubias DJ, Luetmer PH, Campeau NG. Posterior reversible encephalopathy syndrome: prognostic utility of quantitative diffusion-weighted MR images. American J Neuroradiol. 2002;23(6):1038-48.
28. Prognostic Utility of Quantitative DiffusionWeighted MR Images. Am J Neuro Radiol. 2002;23(6):1038-48.
29. Stevens CJ, Heran MKS. The many faces of posterior reversible encephalopathy syndrome. Br J Radiol. 2012;85:1566–75.
30. Deshmukh US, Lundsberg LS, Culhane JF, Partridge C, Reddy UM, Merriam AA, et al. Factors associated with appropriate treatment of acute-onset severe obstetrical hypertension. American J Obst Gynecol. 2021;225(3):329-11.

Cite this article as: Sheetal B, Doddamani U, Shali S, Sangundikar PH, Shradha. Posterior reversible encephalopathy syndrome: a diagnostic dilemma. Int J Reprod Contracept Obstet Gynecol 2025;14:xxx-xx.