Case Series

Posterior Reversible Encephalopathy Syndrome in Eclamptic women

Anam Sarwar[1], Ashwani Verma[2], Samrat Joshi[3], Leena Saini[4]

Abstract

Background : Posterior Reversible Encephalopathy Syndrome (PRES) is a neurological disorder characterised by reversible subcortical vasogenic oedema, typically presenting with headache, altered mental status, seizures and visual disturbances. The etiology is multifactorial and includes hypertension, immunosuppressive therapy, eclampsia and renal failure.

Aims and Objective : This case series aims to explore the clinical presentation, imaging findings, predisposing factors, management strategies and outcomes of patients diagnosed with PRES.

Materials and Methods : We conducted analysis of four cases of PRES admitted to National Institute of Medical Sciences, Jaipur. Clinical data, imaging studies, laboratory results, treatment modalities and patient outcomes were reviewed.

Results : In our study posterior reversible encephalopathy syndrome was associated with eclampsia and characterized by seizures, altered and loss of consciousness, visual disturbances. White matter abnormalities in posterior parietooccipital region on Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) were observed. Management usually includes stabilization of patient control of blood pressure and prevention of seizures.

Conclusion : Our case series underscores the diverse clinical manifestations and predisposing factors associated with PRES. Early recognition and prompt management are essential for optimizing patient outcomes.

Key words : Posterior Reversible Encephalopathy, Pres, Neurological Disorder, Clinical Presentation, Predisposing Factors, Management, Outcome.

Posterior Reversible Encephalopathy Syndrome (PRES) is a neurological syndrome associated with a number of conditions including pre eclampsia, eclampsia, severely high blood pressure, renal failure, SLE and the assumption of immunosuppressive agents[1,2].

The triggering events for PRES seems to be an abrupt increase in blood pressure leading to an acute disruption of blood brain barrier. However, cases in normotensive patients have also been reported[3,4].

PRES is first identified by Hinchy in 1996[5].

PRES is also characterized by headache, confusion, vomiting, altered consciousness, visual disturbances and seizures[5].

Previously this condition has been known by various names like reversible posterior leukoencephalopathy syndrome and reversible occipital parietal encephalopathy, but PRES is now widely accepted term[6,7] MRI and CT show diffuse abnormalities due to vasogenic oedema predominantly within territories of posterior circulations and primarily affecting the subcortical white matter of the parieto-occipital lobes[8]. Eclampsia is characterized by new onset seizures/convulsions in a women with pre eclampsia in the absence of any other causes. Both pre eclampsia and eclampsia may be associated with PRES[9-12].

Editor's Comment :

- PRES is a reversible warning from the Brain in women with pre-eclampsia and eclampsia. When severe hypertension and endothelial dysfunction overwhelm cerebral autoregulation, timely diagnosis and gentle blood-pressure and seizure control can restore normalcy.
- Missed antenatal care turns a preventable condition into a life-threatening event – early screening for pre-eclampsia protects not only the placenta and fetus, but also the mother's brain.

PRES is usually reversible, but permanent damage can occur if cerebral ischemia or haemorrhage occurs[1].

We present four cases of PRES in eclamptic women.

Case 1

A 22 year primigravida at 37 weeks 5 days period of gestation was attended at emergency department in state of altered sensorium (GCS score – E1V2M5) with blood pressure of 220/118 mmhg .

She was intubated according to RSI protocol and foley's catheterization was done.

Patient was given Injection Levipil 1 gm iv, Injection labetalol 20 mg iv along with Injection magnesium sulphate loading dose of 14 gm.

Department of Obstetrics and Gynaecology, National Institute of Medical Sciences, Jaipur, Rajasthan 303121
[1]MBBS, Postgraduate Resident
[2]MS, Assistant Professor
[3]MD, Professor, Department of Anaethesia, Balvir Singh Tomar Institute of Medical Sciences and Research, Jaipur, Rajasthan 303002
[4]MS, Professor, Department of Obstetrics and Gynaecology, Balvir Singh Tomar Institute of Medical Sciences and Research, Jaipur, Rajasthan 303002 and Corresponding Author
Received on : 11/04/2024
Accepted on : 29/04/2024

How to cite this article : Posterior Reversible Encephalopathy Syndrome in Eclamptic women. Sarwar A, Verma A, Joshi S, Saini L. *J Indian Med Assoc* 2026; **124(1):** 56-61.

On per abdominal examination – Uterus was 36 weeks, cephalic presentation, uterus relaxed, fetal heart rate was 98 bpm.

Patient underwent emergency cesarean section under GA and delivered a female child. The neonate weighed 2.41 kg . Liquor was meconium stained and baby was born limp, bag and mask was done for 1 minute and was intubated, baby got admitted in NICU. APGAR score at 1 and 5 min was 4 and 5 respectively. Cord blood gas was normal.

Postdelivery patient was shifted to ICU for intensive monitoring.

On day 1 of post partum blood pressure values of 186/100 mmhg was seen . Treatment with infusion Labetalol at 5ml/hr started and titrated according to blood pressure.

Investigations showed haemoglobin value of 7.4 g/dl, platelet counts of 1,41,000 and urine albumin was 3+, Magnesium sulphate infusion was continued at 1gm/hr in post partum period for 24 hours.

MRI brain including T2-FLAIR and DWI showed radiological picture suggestive of PRES syndrome.

On neurology consultation Injection Mannitol 100 cc iv 8 hourly with Injection Levipil 500 mg i.v 8 hourly was started.

48 hours post partum patient was extubated, neurological examination was normal and blood pressure maintained at 130/80 mmhg on Tab Amlodipine 5 mg 12 hourly with Tab Nicardia 20 mg 8 hourly and adequate pain management. Patient was transferred to obstetric ward. Neonate outcome was good and was shifted to mothers side.

On 7th day patient was discharged from hospital in good health with anti hypertensive therapy. Follow up MRI at 1 month from the event was completely normal.

Histopathological examination of the placenta revealed chronic hypoxia acute inflammatory cells along the chorion.

Case 2

A 24 year primigravida at 27 weeks 3 days period of gestation with one episode of seizure along with tongue bite and one episode of vomiting with severe headache.

Patient was attended at emergency department and blood pressure was 220/118 mmhg and was semiconscious, drowsy and disoriented (GCS score – E3V2M6) on examination B/L pitting pedal edema was present. Patient had 2nd episode of GTCS at emergency department also. Patient was in post ictal phase with GCS score – E2V2M5. Patient was given Injection Levipil 1 gm iv, Injection Labetalol 20 gm i.v with Injection MgSo4 loading dose of 14 mg, oxygen support and shifted to ICU.

Per abdominal examination – Uterus – 24 weeks size, cephalic presentation, uterus relaxed, fetal heart sound-124 bpm.

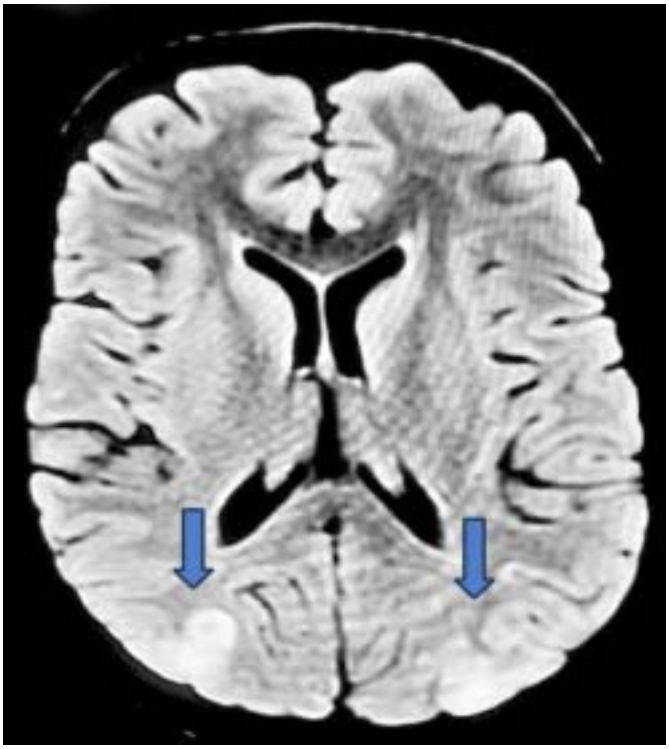

Fig 1 — T2W – FLAIR MRI Patchy area of hyperintensity in cortical subcortical location of parieto occipital region with faint diffusion restriction

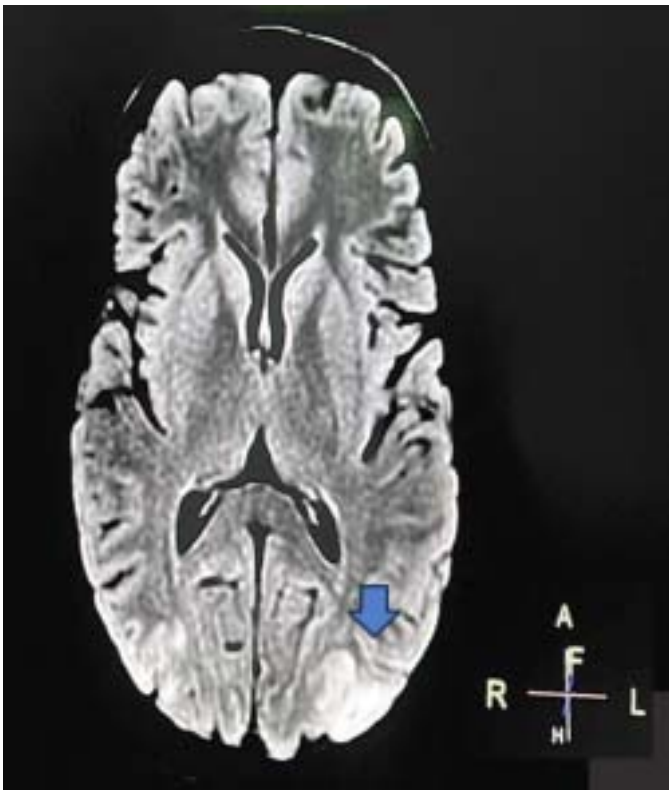

Fig 2 — FLAIR MRI : cortical – subcortical hyperintense lesions in parieto occipital region

Per vaginal examination – Cervical Os closed, uneffaced cervix, vertex high up, no leaking or bleeding per vaginum.

Steroid coverage (Dexamethasone) for fetal lung maturity was given.

Neurology opinion advised Injection levipil 500 mg 8 hourly with Intravenous Magnesium Sulphate infusion for next 24 hours. USG fetal well being was done which was suggestive of single live intrauterine fetus of 26 weeks 2 days with AFI – 8.2 cm, placenta posterior upper, estimated fetal weight of 734 gms. USG fetal color doppler showed absent diastolic flow in umbilical artery with raised PI value in mean maternal uterine artery.

Urine albumin level was 1155.7, UACR – 9472.95 . Blood investigations included Hb – 10.5 g/dl, WBC count of 12.32, platelet – 193, renal and liver functions was normal. As BP was not controlled she was started on injection labetalol infusion at 5ml/hr and nifedipine orally. Magnesium sulphate maintenance dose i.e 1 gm/hr by continuous infusion.

Neonatology opinion for fetal outcome and probable prognosis was done and patient was planned for Caesarean section which was performed under general anaesthesia. The newborn weighted 695 gms and APGAR score of 2 at 1 min and 4 at 5 min, with arterial base excess – 11 millimoles/L and was admitted to NICU.

Postdelivery patient was shifted to ICU for intensive monitoring.

On postpartum day 1 patient had blood pressure values of 180/110 mmhg and heart rate of 92/min with SpO2 at 100% on ventilatory support treatment with infusion labetalol at 5ml/hr and infusion fentanyl was given. Injection Levipil was continued with infusion of magnesium sulphate maintenance dose.

Patient was extubated 24 hours postoperatively and maintained a blood pressure of 160/100 mmhg with heart rate of 84/min on injection NTG infusion and amlodipine 5 mg orally 12 hourly and tablet labetalol 200 mg 8 hourly along with tablet levipil 500 mg 8 hourly.

Patient complained of severe headache and showed persistently raised blood pressure on neurology consultation Injection Mannitol 100 cc IV 8 hourly with Injection levipil 500 mg iv 8 hourly was started.

MRI brain with DW showed patchy area of hyperintensity on T2W/FLAIR in cortical and subcortical location of parieto occipital region s/o likely PRES.

Bed side 2D echo and USG renal doppler was normal.

72 hours after delivery neurological examination was normal and blood pressure maintained at 140/90 mmhg on Tab Amlodipine 5 mg 12 hourly with Tab Nicardia 20 mg 8 hourly and adequate pain management.

On fifth day of delivery patient was shifted to PNC ward neurological examination was normal and blood pressure of 130/80 mmhg.

Neonate had poor prognosis, died on day 6 due to refractory septic shock.

Antecedent cause – Extreme pre maturity and RDS. FLAIR MRI : cortical – subcortical hyperintense lesions in parieto occipital region

On day 8th she was discharged from the hospital in good health and anti hypertensive therapy.

On follow-up after 15 days pedal edema was significantly reduced and pre conceptional counselling was don.

Placental examination showed chronic hypoxia, multiple infarcts and chronic villitis inter-villitis.

Case 3

A 37 year primigravida at 36 weeks 5 days period of gestation with HIV positive on antiretroviral therapy (lamivudine, tenofovir, dolutegravir) since 4th month of gestation. She was attended at emergency department referred from periphery health centre in unconscious state with history of abnormal body movement since 4 hours, multiple episodes of vomiting since last night. On examination GCS score – E1V2M4, blood pressure was 220/120 mmhg, heart rate – 48/min, SpO2 – 48 % on room air. Tongue bite and bilateral pitting edema was present.

She was intubated according to RSI protocol and foley's catheterization done.

Patient was given Injection levipil 1 gm iv, Injection Labetalol 20 gm iv and Magnesium sulphate loading dose

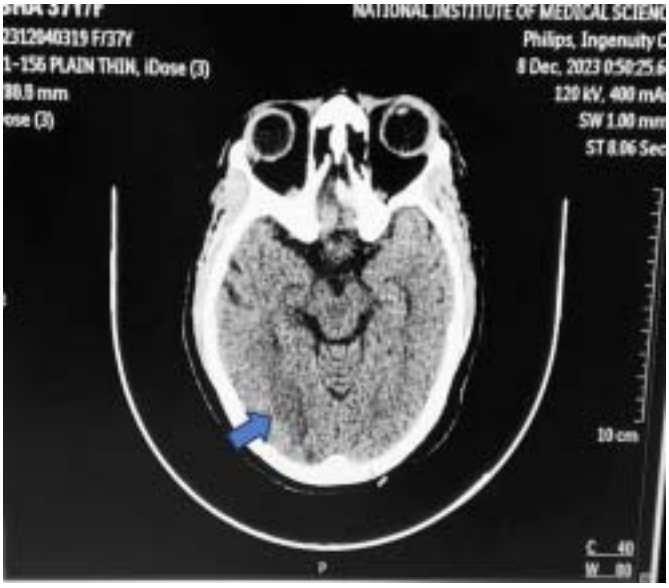

Fig 3 — NCCT scan of brain: Mild hypodensity is seen in the right occipital

of 14 gm. Postintubation GCS score – E1VTM1 (under sedation) SpO2 – 100% maintained at FiO2 – 100%, blood pressure – 170/100 mmhg and heart rate of 76/min.

Per abdominal examination – Uterus – 36 weeks size, cephalic presentation, uterus relaxed, fetal heart was 87 bpm Per vaginum examination – Cervix 1 cm dilated, uneffaced, vertex -1 station, membranes present flat, leaking present clear and no bleeding.

Patient underwent emergency ceasrean section under general anaesthesia and delivered a male child. Baby did not cry immediately after birth and did not had respiratory efforts so bag and mask ventilation given for 60 sec, after which baby cried and was shifted to NICU on CPAP for post resuscitation care. APGAR at 1 and 5 min was 4 and 7. The birth weight of neonate was 2.5 kg. As baby was maintaining saturation well ,was shifted to room air next day. Cord blood gas was normal.

Postdelivery patient was shifted to ICU for intensive monitoring

On postpartum day 1 - Patient had blood pressure of 150/110 mmhg and heart rate of 92/min with SpO2 of 100% on ventilatory support under sedation with Injection fentanyl infusion, injection atracurium injection, Injection levipil 500 mg iv 12 hourly and MgSo4 infusion maintenance dose was continued.

Patient was extubated 48 hours post operatively and had persistenly raised blood pressure (invasive – arterial) of 170/110 mmhg with heart rate of 94/min with continuous intravenous infusion of injection NTG and injection labetalol.

Haemoglobin was 9.9 g/dl, platelet count - 154, TLC – 11.37, D.dimer – 2.85.

PT/INR - 12.8/0.95, Thyroid stimulating hormone – 8.53, anti TPO – 4.57.

Tab Eltroxin 37.5 mcg was started and patient resumed taking anti retroviral drugs.

Patient complained of blurring of vision on 3rd day postpartum and dizziness, fundoscopy examination revealed signs of advanced hypertensive retinopathy. On retina multiple flame shaped haemorrhages and cotton wool spots with hard exudates were present.

NCCT of brain showed mild low density in right occipital white matter.

Renal doppler and 2D echo was normal.

On 4th day post delivery patient complained of pain upper abdomen and nausea.

Ultrasonography of abdomen showed pancreas appear bulky and free fluid in morrisons pouch s/o Ascites.

Amylase – 148, lipase – 640, C reactive protein – 6.5, procalcitonin – 0.096, lifer function tests and renal function test was normal, electrolytes were normal. Gastrology opinion adviced adequate analgesics and liquid diet for the patient.

On day 8th her blood pressure was 140/90 mmhg, pulse rate – 82/min, SpO2 – 98 % on room air with oral Tab Nifidipine 20 mg 6 hourly, clonidine 100 mg 8 hourly, Tab Metroprolol 50 mg 12 hourly.

Her vision had completely returned and pain abdomen was subsided. Patient was shifted to obstetrics ward on 11th day and was discharged on oral anti hypertensives. Follow-up of MRI after 1 month from the event was completely normal. Neonate outcome was good.

Pathological examination of placenta revealed chronic hypoxia, fibrous stroma of chorionic villi.

Case 4 :

A 27 year G2P1L1 at 30 weeks 5 days period of gestation was attended at emergency department referred from peripheral health centre with altered sensorium since 10 mins and abnormal body movements with history of 2 episodes of seizure and vomiting (GCS score – E2V1M2), her blood pressure was 180/108 mmhg, heart rate – 148/min, SpO2 – 90% on room air.

IV line was secured with two large bore IV cannula.

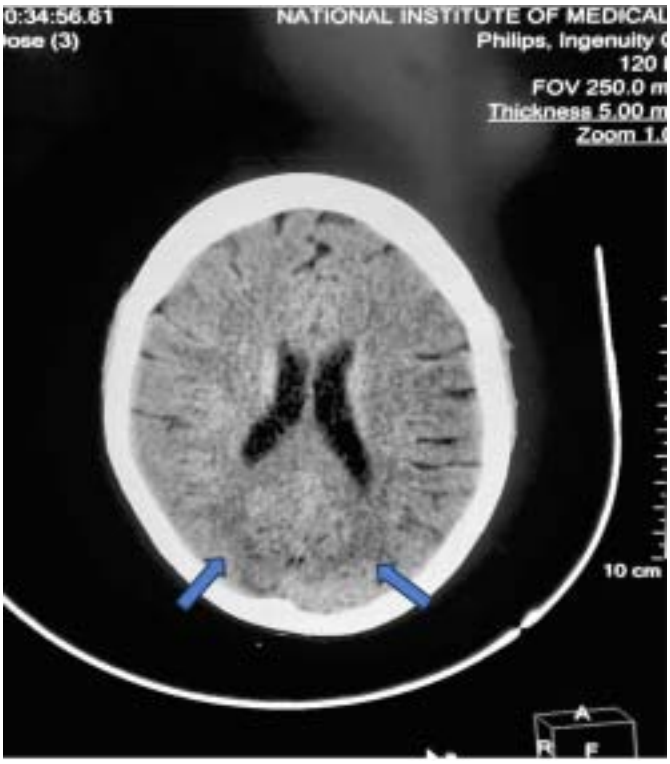

Fig 4 — NCCT scan of brain : Low density in bilateral occipital white matter

She was intubated according to RSI protocol , Ryle's tube with foley's catheter was inserted in triage Patient was given Injection Levipil 1 gm iv, Injection labetalol 20 gm iv and Injection magnesium sulphate 14 gm loading dose given.

Following which GCS score – E1VTM1 (under sedation) SpO2 – 100% maintained at FiO2 – 100%, blood pressure – 128/180 mmhg and heart rate of 110/min.

Arterial blood gas analysis showed metabolic acidosis.

Per abdominal examination – Uterus –28 weeks size, cephalic presentation, uterus relaxed, fetal heart rate was 150 bpm.

Per vaginum examination – cervix 1 cm dilated, uneffaced, vertex -1 station, membranes present flat, no leaking or bleeding.

For steroid coverage 1st dose was given and senior paediatrician was informed. Patient underwent caesarean section under GA and delivered a female child. The neonate weighed 1.13 kg. Liquor was absent and baby cried immediately after birth. APGAR at 1 and 5 min was 6 and 8. Cord blood gas was normal. Neonate was shifted to NICU due to respiratory distress on NIPPV and OG feed was started.

Postdelivery patient was shifted to ICU for intensive monitoring. On first day post partum blood pressure values of 178/116 mmhg was seen. Treatment with infusion labetalol at 2 ml/hr was given.

Blood investigations post delivery showed haemoglobin value of 6.6 g/dl, platelet count of 2,36,000 and urine albumin as 3+, renal and liver functions was normal. Magnesium sulphate infusion was continued at 1gm/hr in post partum period for 24 hours for eclampsia prophylaxis.

Bed side renal doppler was done and showed normal findings.

2D Echo was done which showed grade 1 left ventricular diastolic dysfunction for which patient was managed conservatively.

36 hours post partum neurological examination was normal and blood pressure maintained at 140/100 mmhg on infusion labetalol which was titrated according to blood pressure.

1 unit packed RBC was transfused.

NCCT brain was done, radiological picture suggestive of PRES syndrome.

On neurology consultation Injection Levipil 500 mg IV 8 hourly and Injection Midazolam 1ml/kg was started.

48 hours post partum blood pressure maintained at 140/90 mmhg on Tab Amlodipine 5 mg 12 hourly with Tab Nicardia 20 mg 8 hourly and adequate pain management.

On fifth day of delivery patient was shifted to PNC ward neurological examination was normal and blood pressure of 130/90 mmhg.

On 7th day she was discharged on oral anti hypertensives. Follow-up of MRI after 1 month from the event was completely normal. Neonate outcome was good.

Pathological examination of placenta revealed chronic hypoxia , fibrous stroma of chorionic villi.

Discussion

PRES is a remarkably heterogenous disorder, the severity and extent of symptom depends upon the involved area of the brain, therefore recognizing various manifestations of PRES is important[8].

There are various medical causes or clinical entities associated with causation of PRES which include hypertensive encephalopathy, pre eclampsia, eclampsia, acute or chronic renal diseases, hemolytic uremic syndrome, use of cytotoxic and immunosuppressant drugs, blood transfusion, and electrolyte disturbances[13]. However the ones which predominate in the causation of PRES are pre eclampsia and eclampsia[3]. Pathogenesis of brain lesion till now have been explained by two theories. The first theory proposes that hypertension can cause abnormal cerebral vascular autonomic modulation which causes vasodilatation as blood increases, this is mediated by endothelium, which causes hyper perfusion within the white matter. The second theory suggests that hypertension causes activation of autoregulatory system which causes vasoconstriction and decreases perfusion which causes ischemia[5].

In some rare cases PRES had been identified in the patient with normal blood pressure. It can be due to endothelial activation as an immune response causing production of molecule which alter the normal homeostasis of blood brain barrier, fluid leakage and oedema occurs due to weakening of vessel tight junctions. This scenario describes hypertension as a secondary syndrome of the underlying mechanism and not the cause[14].

Diagnosis of PRES is usually done by computed tomography and MRI. In our cases hyperintensity cortico subcortical location of parieto occipital region was seen in T2W FLAIR MRI in two cases and hypodensity in occipital lobe on NNCT in two cases.

Early diagnosis of PRES and reduction of blood pressure and early initiation of therapy is necessary . Blood pressure and seizure control remains the mainstay of therapy. Aggressive blood pressure control is not advised, because this may reduce the blood pressure below the autoregulatory range and may lead to ischemic events[15].

Labetalol and nicardipine are the are the first line drug for

lowering blood pressure in PRES patients[16]. Magnesium sulphate therapy should be initiated as soon as eclampsia or PRES in pregnancy is suspected , as it treats seizures[17].

The prognosis of PRES is good and 75% - 90 % of patients fully recover[18].

In our study, we discussed about four cases, three out of four women were nulliparous, one was in late second trimester and other three were is third trimester. All women were unbooked cases and presented as eclampsia and three women were intubated in emergency. All women underwent caesarean section under general anaesthesia out of which one had poor fetal outcome (died on day 6 due to refractory septic shock, Antecedent cause – extreme pre maturity and RDS) rest three had good fetal outcome.

On histopathological examination of placenta signs of chronic hypoxia and impaired placentation was seen, indicating presence of chronic disease that could have been diagnosed at earlier stages during routine check-ups.

Conclusion

In this study four cases of PRES in antenatal women with eclampsia were presented.

All the women were unbooked and none of the women had undergone early screening for pre eclampsia, so effective pre eclampsia screening in first trimester is needed, based on combination of clinical, biophysical, biochemical markers and uterine artery doppler and would allow the administration of therapy to improve placentation and to reduce the risk of PRES as although PRES is reversible but also has potential for serious complication.

Funding : None.

Conflict of Interest : None.

References

1 Hinchey J, Chaves C, Appignani B — A reversible posterior leukoencephalopathy syndrome. *The New England Journal of Medicine* 1996; **334(8):** 494500, View at: Publisher Site | Google Scholar

2 Onder AM, Lopez R, Teomete U — Posterior reversible encephalopathy syndrome in the pediatric renal population. *Pediatric Nephrology* 2007; **22(11):** 1921-9. View at: Publisher Site | Google Scholar

3 Kastrup O, Maschke M, Wanke I, Diener HC — Posterior reversible encephalopathy syndrome due to severe hypercalcemia. *J Neurol* 2002; **249:** 1563-6. [PubMed] [Google Scholar]

4 Ay H, Buonanno FS, Schaefer PW, Le DA, Wang B, Gonzalez RG, *et al* — Posterior leukoencephalopathy without severe hypertension: utility of diffusion-weighted MRI. *Neurology* 1998; **51:** 1369–76. [PubMed] [Google Scholar]

5 Rho JD, Kim YH, Shin JH, Kim TK — Case report: A case of posterior reversible encephalopathy in postpartum preeclampsia. *Medicine (Baltimore)* 2023; **102(47):** e36023. doi: 10.1097/MD.0000000000036023. PMID: 38013383; PMCID: PMC10681536.

6 Liman TG, Bohner G, Heuschmann PU — The clinical and radiological spectrum of posterior reversible encephalopathy syndrome: the retrospective Berlin preS study. *J Neurol* 2012; **259:** 155–64.doi:10.1007/s00415011-6152-4 pmid:http://www.ncbi.nlm.nih.gov/pubmed/21717193 CrossRefPubMedGoogle Scholar.

7 Brewer J, Owens MY, Wallace K — Posterior reversible encephalopathy syndrome in 46 of 47 patients with eclampsia. *Am J Obstet Gynecol* 2013; **208:** 468.e1–468.e6.doi:10.1016/j.ajog.2013.02.015 pmid:http://www.ncbi.nlm.nih.gov/pubmed/23395926 PubMedGoogle Scholar.

8 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:** 494-500. [PubMed] [Google Scholar].

9 Gestational hypertension and preeclampsia: ACOG Practice Bulletin, Number 222. Obstet Gynecol. 2020, 135:e237-e260. 10.1097/AOG.0000000000003891.

10 Verma AK, Garg RK, Pradeep Y — Posterior encephalopathy syndrome in women with eclampsia: predictors and outcome. *Pregn Hypertens* 2017; **10:** 74-82. 10.1016/j.preghy.2017.06.004.

11 Junewar V, Verma R, Sankhwar PL — Neuroimaging features and predictors of outcome in eclamptic encephalopathy: a prospective observational study. *Am J Neuroradiol* 2014; **35:** 1728-34. 10.3174/ajnr.A3923

12 Fisher N, Saraf S, Egbert N, Homel P, Stein EG, Minkoff H — Clinical correlates of posterior reversible encephalopathy syndrome in pregnancy. *J Clin Hypertens (Greenwich)* 2016; **18:** 522-7. 10.1111/jch.12656.

13 Stott VL, Hurrell MA, Anderson TJ — Reversible posterior leukoencephalopathy syndrome: a misnomer reviewed. *Inter Med J* 2005; **35(2):** 83-90

14 Marra A, Vargas M, Striano P, Del Guercio L, Buonanno P, Servillo G — Posterior reversible encephalopathy syndrome: The endothelial hypotheses. *Medical Hypotheses* 2014; **82(5):** 619-22,ISSN 03069877,https://doi.org/10.1016/j.mehy.2014.02.022.

15 Katwal S, Ghimire A, Bhusal A, Bajracharya A — Posterior reversible encephalopathy syndrome in postpartum patients with gestational hypertension: A case report emphasizing early recognition and management. *Radiology Case Reports* 2013; **18(12):**

16 [19]G Servillo, F Bifulco, E De Robertis, O Piazza, P Striano, F Tortora, *et al* — Posterior reversible encephalopathy syndrome in intensive care medicine. *Intensive Care Med* 2007; **33:** 230-6.

17 Striano P, Striano S, Tortora F, Robertis ED, Palumbo D, Elefante A, *et al* — Clinical spectrum and critical care management of posterior reversible encephalopathy syndrome (PRES). *Med Sci Monit* 2005; **11(11):** CR549CR553.

18 Roth C, Ferbert A — Posterior reversible encephalopathy syndrome: long-term follow-up. *J Neurol Neurosurg Psychiatry*; **81(7):** 773-7.Google Scholar