Section: Obstetrics and Gynaecology

Original Research Article

# AN OBSERVATIONAL STUDY ON POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN SEVERE PREECLAMPSIA AND ECLAMPSIA

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## Abstract

**Background:** The aim of this study was to determine incidence of PRES among the patient with severe preeclampsia& eclampsia; to determine the predictors of posterior reversible encephalopathy syndrome (PRES) and to assess the impact of PRES on maternal and foetal outcome. **Materials and Methods:** This prospective observational study was carried out over a period of 12 month from 1st April, 2020 to 31st march, 2021 at Midnapore Medical College and hospital. We included consecutive women with severe preeclampsia and eclampsia. Maternal outcome was assessed using WHO maternal near miss identification criteria. **Result:** 95 consecutive women with severe preeclampsia and eclampsia were included. Among 95 patients with neurological symptoms, 55 out of 73 (75.34%) eclamptic patients and 2 out of 22 (9.09%) severe pre-eclamptic patients had posterior reversible encephalopathy syndrome on brain imaging study. 49 (85.96%) patients with antepartum eclampsia, 1 (100%) patient with intrapartum eclampsia and 5 (33.33%) patients with postpartum eclampsia had PRES on imaging study. Predictors of posterior encephalopathy syndrome were antepartum eclampsia, altered sensorium, vision impairment, headache, vomiting, decreased haemoglobin, low platelet count, high SGOT and SGPT leveland higher proteinuria. Patients with PRES had severe maternal outcome (31.4% vs 13.1%; P=0.02). Perinatal death was higher among patients with PRES (40.3% vs 15.7%; P=0.033). **Conclusion:** PRES appears to accompany severe preeclampsia and eclampsia in over half of all cases. Some clinical pictures and certain biochemical characteristics suggest PRES in patients with severe preeclampsia and eclampsia. PRES was associated with poorer maternal and foetal outcome.

## INTRODUCTION

Hypertensive disorders are one of the most common medical complications of pregnancy.[1]Among pregnant women the incidence of preeclampsia and eclampsia is 43.3% and 10.8% respectively in India.[2]Eclampsia is a significant cause of maternal and foetal mortality and morbidity in our country.[3]The posterior reversible encephalopathy syndrome (PRES) is defined as a group of some neurological signs and symptoms which is associated with typical magnetic resonance imaging (MRI) features. The characteristic findings on MRI are hyperintensity and oedema on T2-weighted images involving mainly in the occipital and parietal lobes.[4]Hinchey et al. first described Posterior Reversible Encephalopathy Syndrome (PRES) in1996.The authors described a condition marked by headache, altered mental status, seizures, and visual changes in a case series of 15 patients.Extensive white-matter changes which are suggestive of oedema of the posterior cerebral region was also found in the imaging study of these patients.[5]Hinchey et al. first named this syndrome as reversible posterior leukoencephalopathy syndrome. But it has also been known by many other names including PRES, hyper perfusion encephalopathy, hypertensive encephalopathy and brain capillary leak syndrome. PRES has been associated with many conditions like eclampsia, severe hypertension, autoimmune disease, post-transplantation immune suppression, treatment with cytotoxic medications and infection with sepsis to name a few.[3]Regardless of the underlying disease, the similar clinical presentation and imaging findings are usually seen. Clinical findings of PRES include blood pressure (BP) elevation, headache, seizures, altered mental status, decreased alertness


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and blurring of vision similar to the clinical findings in eclampsia.The syndrome is primarily characterized by vasogenic oedema of the subcortical white matter with predominant involvement of parenchyma supplied by the posterior circulation and is potentially reversible.[5]Aclear relationship between clinical features and specific imaging findings with severity or location of oedema has not been supported by any conclusive evidence.Although some studies have reported some correlations of various clinic-laboratory parameters with PRES and maternal-foetal outcomes in women with PRES.[1]In eclampsia, the incidence of posterior leukoencephalopathy syndrome is not precisely known. Limited obstetric literature has been found on PRES like some cases/case series.[5]

So, we have focused on to determine the incidence of PRES and whether the presence of PRES can be predicted from some particular clinical aspects of eclampsia and severe preeclampsia. Since the reversibility of the syndrome depends on early recognition and management of PRES, so by recognizing and managing early we can determine which patient with eclampsia/ severe preeclampsia with PRES may have better maternal and foetal outcomes.

## MATERIALS AND METHODS

Our study was conducted in the department of Obstetrics and Gynaecology, Midnapore Medical College and Hospital, west Bengal, over a period of 18 months between 1st April 2020 to 30th September 2021. This tertiary care hospital is equipped with round the clock ultrasonography and laboratory facilities for blood parameters examination, nephrology department with haemodialysis facilities, one dedicated high dependency unit with 12 beds and necessary equipment and blood bank with round the clock availability of blood and blood products. The Institutional Ethics Committee has been approved the study, and the legal guardian of all the women or the woman herself have been given the informed consent to be included in the study. Patients with severe preeclampsia & eclampsia during antepartum, intrapartum and postpartum period undergoing neuroimaging via magnetic resonance imaging (MRI)/Computed tomography (CT) performed after 24hour of last episode of convulsion and after completion of magnesium sulphate dose for usual clinical indication were included in the study. Following admission in eclampsia room a detailed history taking and a rapid general examination was done and documented. Obstetrics examination like estimation of height of uterus, frequency and duration of uterine contraction, lie & presentation of foetus, rate & regularity of foetal heart rate and vaginal examination was done. The patients with severe preeclampsia and eclampsia have been managed by the following steps. (1) consultant obstetrician/specialist were included in management plan. (2) magnesium sulphate according to Pritchard regimen started immediately and continued for 24 h. (3) Treatment of high blood pressure was done by using intravenous inj. Labetalol. (4) Patients have been monitored by measuring blood pressure and urine output until they were discharged from ward.[5] Progress of labour and foetal heart sound was monitored using partograph until delivery.[6] Input–output chart was maintained and deep tendon reflexes, respiratory rate were monitored in regular interval when magnesium sulphate was used.[7] Blood investigations CBC, urea, creatinine, liver function tests, serum electrolytes and urine for albumin and ophthalmoscopic examination were done in all women as routine investigation.[8] After stabilization of blood pressure and convulsions a senior obstetrician has taken the decision for delivery according to individual case merit.

Following conservative management those who were allocated for vaginal delivery, induction of labour was done with misoprostol tablet 25 mcg vaginally every 4 h with a maximum of five doses. Women were monitored in active labour using partograph and augmentation with injection oxytocin/ARM (or both) was done if uterine contractions were <3 in 10 min.

**Evaluation**

All the included women were subjected to magnetic resonance imaging of brain on 1.5 Tesla machine. Brain imaging was done within seventh day of onset of clinical symptoms. Some patients were subjected to CT scan of brain for whom MRI was not possible. Brain imaging was evaluated by an expert neuro-radiologist, after imaging women were classified as having normal neuroimaging, neuroimaging suggestive of posterior encephalopathy syndrome or any other alternative diagnosis.

**Follow- up and Outcome Assessment**

Women were followed for 7 days till discharge. Outcome assessment was done using WHO maternal near miss identification criteria. Severe maternal outcomes are maternal near miss cases and maternal deaths.

Maternal death is defined as death of a woman while pregnant or within 42 days of termination of pregnancy, irrespective of the duration and the site of the pregnancy, from any cause related to or aggravated by the pregnancy or its management, but not from accidental or incidental causes.

A maternal near-miss case is defined as “a woman who nearly died but survived a complication that occurred during pregnancy, childbirth or within 42 days of termination of pregnancy”. In practical terms, women are considered near miss cases when they survive life-threatening conditions (i.e. organ dysfunction).

Foetal death was defined as delivery of a foetus with no signs of life. Neonatal death was defined as death of baby within first 7 days of life.


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## RESULTS

We evaluated 102 consecutive patients who had features of severe preeclampsia and eclampsia and underwent neuroimaging shortly after their admission in this study.6 patients were excluded from the study because 4 MRI reports were not available and 2 patients lost during follow up. Another patient was also excluded because of presence of cerebral infract on MRI. Out of remaining 95 patients 73 had eclampsia and 22 had features of severe preeclampsia. 11 patients, who were comatose, were subjected to NCCT of brain. [Figure 1].

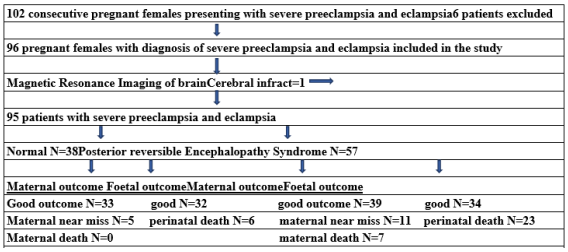



**Figure 1: flowchart of patient selection and outcome**

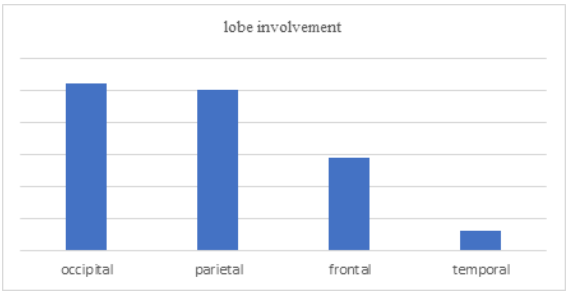



**Figure 2: Diagram shows the distribution of the study population by brain lobe involvement**

Out of 95 patients 74 were subjected to MRI and 11 were subjected to NCCT brain.47 (49.47%) patients among those who were subjected to MRI and 10 (10.52%) patients among those who were subject to NCCT of brain had features of PRES [Table 1].We found that 57 (60%) out of 95 patients had radiological evidence of PRES. So, the overall incidence of PRES was 60%.In our study, we noted that 75.34% of patients with eclampsia and 9.10% of patients with severe preeclampsia had posterior encephalopathy syndrome on imaging.

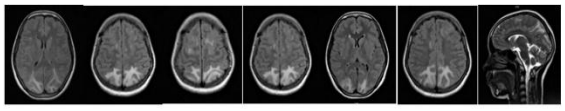
We also found that the hyperintense lesion on T2-weighted and FLAIR sequences involved occipital lobe (54.73%) and parietal lobe (52.63%) more frequently followed by frontal lobe (30.52%) and temporal lobe (6.31%).Occipital and parietal lobe involved with near about same frequency. [Figure 2].

We noted that the occurrence of posterior reversible encephalopathy syndrome was associated with antepartum eclampsia [p- 0.04], altered sensorium [p-0.018], headache [p-0.013], blurring of vision [p-0.004], vomiting [p-0.012], higher Hb% [p-0.006], low platelet count [p-0.043], high SGOT [p-0.005] and SGPT [p-0.002] level. [Table 2]

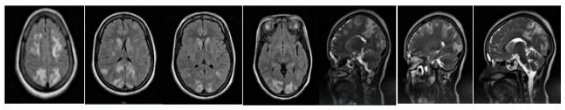
We evaluated maternal outcome by using WHO maternal near miss identification criteria. We noted 7 maternal deaths all of whom had PRES and 16 maternal near miss (total 23 severe maternal outcome) among 57 patients with posterior reversible encephalopathy syndrome. Among patients with severe maternal outcome 9 had CNS dysfunction, 5 had pulmonary dysfunction, 5 had haematological dysfunction, 3 had renal dysfunction and 1patient had severe PPH.

The factors associated with severe maternal outcome were altered sensorium [p<0.05], headache[p-0.01], vision impairment[p<0.05], vomiting[p-0.01], GCS ≤8 at admission[p<0.05], PRES on MRI[p-0.02], not having regular antenatal check-up (4 or more) [p-0.01], high blood urea[p<0.05] and creatinine level[p<0.05], high SGOT[p<0.05] and SGPT[p<0.05] level. [Table 3]

There is 23/57 (40.35%) perinatal death in patients with PRES as compared with 6/38 (15.78%) in patients withoutPRES.We also noted that some factors associated with pregnant women indicates poor foetal outcome, like vision impairment(p-0.046), GCS ≤8 at the time of admission(p<0.05), status of regular antenatal check-up(p-0.015), PRES on MRI(p-0.010), high blood urea level(p-0.017).



**Patient 1:** Mrs. S. Murmu, 21year primigravida, admitted on 16-01-20 with multiple episodes of convulsion.



Patient 2: Mrs. P. Mandi, 25 years primigravida, admitted on 02-01-20 with multiple episodes of convulsion.

**Figure 3: T2 and FLAIR images showing classical bilateral hyperintensities involving fontal, parietal and occipital lobe.**

**Table 1: Distribution of the study population according to radiological findings (N=95)**

| Radiological findings | Frequency | Percentage (%) |
|---|---|---|
| PRES (MRI) | 47 | 49.47 |
| NO PRES (MRI) | 37 | 38.94 |
| PRES (NCCT) | 10 | 10.52 |
| NO PRES (NCCT) | 1 | 1.05 |
| Total | 95 | 100 |


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**Table 2: Uni-variate analysis comparing clinical and laboratory features of posterior encephalopathy syndrome and normal neuroimaging patients**

| Variables | PRES(N=57) | Normal MRI(N=38) | P value |
|---|---|---|---|
| Age (mean ±SD, median) | 20.38 ±3.89, 19 | 21.55 ±3.70, 21 | 0.07 |
| Primigravida | 47(82.45%) | 26(68.42%) | 0.44 |
| Antepartum eclampsia | 49(85.96%) | 8(21.05%) | 0.04 |
| Altered sensorium | 34(59.64%) | 1(2.63%) | 0.01 |
| Headache | 51(89.47%) | 6(15.78%) | 0.01 |
| Blurring of vision | 45(78.94%) | 3(7.89%) | 0.004 |
| Vomiting | 42(73.86%) | 3(7.89%) | 0.01 |
| GCS≤8 at admission | 11(19.29%) | 0 | 0.08 |
| Booked case | 31(54.38%) | 26(68.42%) | 0.38 |
| Preterm delivery | 20(35.08%) | 14(36.84%) | 0.88 |
| Hb (gm/dl) (mean ±SD, median) | 11.22 ±1.53, 11.2 | 10.41 ±1.52, 10.65 | 0.006 |
| Platelet (/mm³) (mean ±SD) | 133964 ±77138 | 159157 ±64295 | 0.04 |
| Urea (mg/ dl) (mean ±SD) | 32.3 ±12.5 | 30.8 ±23.1 | 0.35 |
| Creatinine (mg/dl) (mean ±SD) | 0.9 ±0.3 | 0.8 ±0.7 | 0.31 |
| Bilirubin (mg/dl) (mean ±SD) | 0.68 ±0.43 | 0.70 ±0.43 | 0.40 |
| SGOT (IU/L) (mean ±SD) | 66.5 ±52.5 | 43.1 ±34.8 | 0.005 |
| SGPT (IU/L) (mean ±SD) | 70.3 ±54.7 | 43.3 ±37.5 | 0.002 |
| Systolic BP (mean ±SD) | 146 ±24 | 152 ±30 | 0.14 |
| Diastolic BP (mean ±SD) | 95 ±17 | 92 ±16 | 0.24 |

**Table 3: Univariate analysis showing clinical, neuroimaging and laboratory parameters associated with poor maternal outcome**

| Variables | Good maternal outcome N=72 | Severe maternal outcome (maternal near miss +maternal death) N= 23(16 +7) | P value |
|---|---|---|---|
| Age (mean ±SD), median | 20 ±3, 19 | 21 ±3, 21 | 0.41 |
| Primigravida | 57 (78.08%) | 16 (21.91%) | 0.89 |
| Antepartum eclampsia | 41 (71.9%) | 16 (28.1%) | 0.16 |
| Altered sensorium | 19 (54.28%) | 16 (45.71%) | 0.001 |
| Headache | 38 (66.6%) | 19 (33.3%) | 0.01 |
| Vision impairment | 29 (60.4%) | 19 (39.5%) | <0.05 |
| Vomiting | 29 (64.4%) | 16 (35.5%) | 0.01 |
| GCS≤8 at admission | 2 (25%) | 6 (75%) | <0.05 |
| Un-booked case | 24 (63.1%) | 14 (36.9%) | 0.01 |
| PRES on MRI | 39 (68.45%) | 18 (31.4%) | 0.02 |
| Hb (gm/dl) (mean ±SD) | 10.9 ±1.5 | 10.9 ±1.7 | 0.41 |
| Platelet (/mm³) (mean ±SD) | 159708 ±70034 | 124130 ±98702 | 0.05 |
| Urea (mg/dl) (mean ±SD) | 28.02 ±8.9 | 45.47 ±31.02 | 0.006 |
| Creatinine(mg/dl) (mean ±SD) | 0.79 ±0.23 | 1.45 ±1 | 0.002 |
| Bilirubin (mg/dl) (mean ±SD) | 0.63 ±0.35 | 0.85 ±0.59 | 0.05 |
| SGOT (IU/L) (mean ±SD) | 45.61 ±36 | 99.47 ±58.59 | <0.05 |
| SGPT (IU/L) (mean ±SD) | 48.72 ±41.47 | 94.91 ±58.16 | <0.05 |
| Systolic BP (mean ±SD) | 150 ±27 | 143 ±25 | 0.13 |
| Diastolic BP (mean ±SD) | 95 ±17 | 90 ±13 | 0.08 |

## DISCUSSION

In our knowledge, this is the first prospective study included patients with preeclampsia and eclampsia with a large sample size conducted in India. Most of the patients were subjected to MRI except few who have been undergone NCCT for imaging study because of their overall condition did not permit to do so.

Out of 95 patients 74 were subjected to MRI and 11 were subjected to NCCT brain.47 (49.47%) patients among those who were subjected to MRI and 10 (10.52%) patients among those who were subject to NCCT of brain had features of PRES.In our study, we noted that 75.34% (73/95) of patients with eclampsia and 9.10% (22/95) of patients with severe preeclampsia had posterior encephalopathy syndrome on imaging.We found that 57 (60%) out of 95 patients had radiological evidence of PRES. So, the overall incidence of PRES was 60%, which correlates with many previous studies.S. Singh et al. noted that 71.4% (5/7) women with eclampsia and 27.27% (3/11) women with preeclampsia having features of PRES in imaging study (1).D. HosapatnaBasavarajappa, et al. found that 86.7% (13/15) of patients with eclampsia and 23.3% (14/60) of patients with severe preeclampsia had neuroradiological diagnosis of PRES(6).Mai et al.noted that PRES occurred in 87.50% eclampsia patients (21/24) and 46.03% preeclampsia patients (58/126)(4).Bahadur et al.have got 75% (9/12) incidence of PRES among patients with eclampsia and 7% (3/41) among patients with preeclampsia.[7]

We also found that the hyperintense lesion on T2-weighted and FLAIR sequences involved occipital lobe (54.73%) and parietal lobe (52.63%) more frequently followed by frontal lobe (30.52%) and temporal lobe (6.31%) represent typical or classical PRES.The white matter involved more frequently than grey matter, which is consistent with vasogenic oedema. Lesions are mostly bilaterally symmetric. D. Hosapatna Basavarajappa, et al.also noted that


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patients with PRES had majority of lesions distributed inparieto-occipital lobes accounting for an average of 75%.[6]While in a study, Bahadur et al. noted thatfrontal lobe is most common location for sub-cortical hyperintensities followed by parietal and occipital.The characteristic findings of PRES are not limited to occipital and parietal lobes, theoretically, any part of the brain can be affected. In many cases, deep grey matter and the brainstem of the brain are involved.[7]

We noted that the occurrence of posterior reversible encephalopathy syndrome was associated with antepartum eclampsia [p- 0.04], altered sensorium [p-0.018], headache [p-0.013], blurring of vision [p-0.004], vomiting [p-0.012], higher Hb% [p-0.006], low platelet count [p-0.043], high SGOT [p-0.005] and SGPT [p-0.002] level (table no. 32). These factors indicate multi-organ involvement and a severe disorder. These factors can be considered as predictors of PRES. Bahadur et al.found age, parity, and presence or absence of eclampsia (all variables with $p < 0.1$ on the univariate analysis) as the independent predictors of PRES.[7]D. Hosapatna Basavarajappa et al. noted patients with headache (p- 0.490), visual disorder (p−0.042), depression of consciousness (p – 0.006), seizure (p– 0.000) had abnormal MRI findings suggestive of vasogenic oedema.[6]

Maternal and foetal outcome in women with severe preeclampsia and eclampsia complicated with posterior reversible encephalopathy syndrome is not well established. We evaluated maternal outcome by using WHO maternal near miss identification criteria. We noted 7 maternal deaths all of whom had PRES and 16 maternal near miss (total 23 severe maternal outcome) among 57 patients with posterior reversible encephalopathy syndrome. Among patients with severe maternal outcome 9 had CNS dysfunction, 5 had pulmonary dysfunction, 5 had haematological dysfunction, 3 had renal dysfunction and 1 patient had severe PPH. It indicates posterior reversible encephalopathy syndrome is associated with severe maternal outcomes. The factors associated with severe maternal outcome were altered sensorium [p<0.05], headache[p-0.01], vision impairment[p<0.05], vomiting[p-0.01], GCS ≤8 at admission[p<0.05], PRES on MRI[p-0.02], not having regular antenatal check-up (4 or more) [p-0.01], high blood urea[p<0.05] and creatinine level[p<0.05], high SGOT[p<0.05] and SGPT[p<0.05] level. All these factors indicate a severe form of the disease with multiple organ malfunction. Verma AK et al. noted poor maternal outcome comprising 12 maternal death and 29 significant disabilities using modified Rankin scale.[3]While S. Singh et al. had not got any significant differences in maternal and foetal outcomes in women with PRES.[1]

We noted a poor foetal outcome too. We recorded more foetal neonatal deaths in the group having posterior reversible encephalopathy syndrome. There is 23/57 (40.35%) perinatal death in patients with PRES as compared with 6/38 (15.78%) in patients without PRES. This finding is comparable with many previous studies. Verma AK et al noted 35/74(47.3%)perinatal death among patients with PRES vs 3/17(17.6%)among patients without PRES.[3]While Chao et al. found all live births with a 5-min Apgar score of > 7, except one wherein congenital anomaly resulted in IUFD.[8]

**Study Strengths and Limitations**

Our study has several strengths and limitations. The largest study conducted in India with 104 sample sizes included only patients with features of eclampsia. In our study we included patients with both eclampsia and severe preeclampsia. The inclusion of women who did not have seizures in our study allowed us to demonstrate the occurrence of PRES among non-eclamptic women.

We did not perform follow up neuroimaging and thus, reversibility of neuroimaging findings was not recorded. Another limitation is we did not able to reach the proposed number of 113 sample sizes due to reduced admission during the covid pandemic. This is a single centre study, so study population is restricted to a specific area.

## CONCLUSION

We concluded that, in patient with severe preeclampsia and eclampsia, PRES is a very frequent clinico-imaging syndrome. The presence of PRES can be predicted from some clinical and laboratory findings. PRES is often associated with poor maternal and fetal outcome.

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