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CASE REPORT

Late Postpartum Pre-eclampsia with Posterior Reversible Encephalopathy Syndrome

Kshitij Mohini¹, Naqiya Bellary², Richa Chandra³, Lucetta Amelia Dias⁴

^{1,3,4}Junior Resident, ²Assistant Professor, Department of Obstetrics and Gynaecology, D Y Patil Hospital, Kolhapur, Maharashtra, India

ABSTRACT:

Posterior Reversible Encephalopathy Syndrome (PRES) is a clinic neuroradiological entity presenting with headache, confusion, visual disturbances or blindness, and seizures. Parieto-occipital white matter changes due to vasogenic edema can be observed on imaging modalities. It rarely occurs without seizures and after delivery. We report a 27-year-old multi gravida with a history of pre-eclampsia in term pregnancy complicated by PRES with seizures at the postpartum period. Clinical improvement with complete resolution without any complications was observed on 11th day after delivery. Posterior Reversible Encephalopathy Syndrome is reversible when early diagnosis is established and appropriate treatment is started without delay.

Key words: PRES (Posterior reversible encephalopathy syndrome, RPL (Reversible posterior leukoencephalopathy).

Corresponding author: Dr. Kshitij Mohini, Junior Resident, Department of Obstetrics and Gynaecology, D Y Patil Hospital, Kolhapur, Maharashtra, India

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NTRODUCTION Reversible leuko-encephalopathy posterior syndrome (RPLS) or posterior reversible encephalopathy syndrome (PRES) was first described by Hinchey in 1996. She described it as a reversible syndrome manifested as headache, altered mental functioning, seizures, and loss of vision associated with white matter changes, suggestive of edema mainly in the posterior regions of the cerebral hemispheres, but also involving the brainstem, cerebellum, and other cerebral areas. Postpartum preeclampsia is a rare and underrecognized condition occurring in 5.7% of all cases of pregnancy-induced hypertension.¹

Causes of Posterior Reversible Encephalopathy Syndrome:

- 1. Hypertensive encephalopathy
- 2. Eclampsia
- 3. Renal failure with hypertension
- Immunosuppressive agents and cytotoxic drugs-Cyclosporin A, Interferon alfa, Intravenous immunoglobulins, Cisplatin, Cytarabine Erythropoietin, Tacrolimus
- 5. Collagen vascular diseases- e.g. Systemic lupus erythematosus etc
- Others- hypercalcemia, postdural puncture and spinal anaesthesia

CASE REPORT

A 27 year old multigravida with post natal day 9 of full term normal vaginal delivery came to D Y Patil Hospital casualty with referral letter from Gauri Maternity Home in view of postpartum convulsion with focal neurological deficit with complaints of headache and giddiness since the previous night. She was apparently alright when she developed headache, giddiness since last night which was acute in onset and gradually progressive. She visited the local doctor with the same complaint; where upon single reading of elevated blood pressure, patient was referred to Gauri Hospital and admitted there. Patient delivered a male 9 days back, a normal full term vaginal delivery and a healthy baby of 2.8 kg weight, at the same hospital. She developed 3 episodes of generalised tonic clonic convulsions early in the morning next day. She was given inj MgSO4 24 gms in total by Pritchard Regimen and then referred to DY Patil Hospital. She has triplets, all females, 9 years back, delivered vaginally. She also has a history of one intrauterine death, preterm vaginal delivery 11 years back. On arrival to DY Patil Hospital, patient was stable, conscious, oriented to time, place and person. Pedal edema was present, urine protein on dipstick +3, deep tendon reflex exaggerated, BP- 170/100 mmhg, P-70beats/min. She also showed involuntary movements of left leg with slight twitching which resolved on third day. All laboratory investigations were within normal limits. Her MRI report was suggestive of Posterior Reversible Encephalopathy Syndrome. Patient was admitted in ICU with strict monitoring of TPR, BP, urine output (after catheterizing the patient), Urine protein and deep tendon reflexes. Medicine opinion was taken and she was started on T.Levetiracetam. Patient was given T.Labetalol 100mg twice daily with T. Nifedipine 10mg stat dose if diastolic BP>100mmhg. Opthalmology opinion was taken which showed no abnormality in fundus. Patient was shifted to ward on post natal day 13 and discharged on post natal day 16.

DISCUSSION

PRES is a reversible neurological entity characterized by the presence of white matter edema affecting the occipital and parietal lobes. The exact incidence of PRES is unknown. ¹⁰ It has become better recognized with the progress in imaging modalities. The more popular theory suggests that hypertension leads to failure of autoregulation, subsequent hyperperfusion and vasogenic edema. The other theory suggest that vasoconstriction and hypoperfusion leads to brain ischemia and subsequent vasogenic edema. ²

PRES is characterized by high signal intensity on T2-weighted and FLAIR images, predominantly in the posterior regions, which is caused by subcortical white matter vasogenic edema. Supplemental diffuse weighted imaging (DWI) and apparent diffuse coefficient (ADC) map images are helpful in distinguishing vasogenic from cytotoxic edema, which represents foci of irreversible ischemia. Atypical features have been seen in neuroimaging like significant anterior, cortical, brainstem lesions, recurrent RPL episodes, foci of permanent injury, hemorrhage into lesions, and unilaterally.³

Preeclampsia usually occurs between 20 weeks of gestation and 48 hours postpartum. If it occurs after 48 hours and upto 30 days after delivery, it is called late postpartum preeclampsia or eclampsia. The syndrome is usually reversible within 7 days, after controlling the blood pressure. Offending immunosuppressive agents should either be discontinued or the dose should be reduced.

Delay in initiating the appropriate treatment may result in permanent damage to the brain. Patients experiencing seizures become seizure free after resolution of imaging abnormalities and do not require chronic antiepileptic treatment. Therapeutic strategy depends on the cause of PRES and clinical picture. Most important are blood pressure regulation (labetalol, nitropruside, diuretics), control of epileptic attacks (phenytoin), anti-edema therapy (mannitol). Induction of vaginal delivery in eclampsia and discontinuation of cyclosporine therapy. In most cases there are no neurological manifestations after the 7th day, but some studies showed normalization of clinical finding after one year and more.⁷ PRES has been reported to be reversible¹; however, irreversible brain damage can sometimes occur due to late recognition or incorrect treatment.8,9

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