# POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN PREGNANCY- A CASE REPORT Heera Shenoy T<sup>1</sup>

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### **CLINICAL PRESENTATION**

A 23 year old primi referred at 34 weeks gestation with history of elevated blood pressure reading of 160/100mmHg. She was having regular ante-natal checkups and was diagnosed to be a Gestational Diabetic on insulin and Pregnancy Induced Hypertension on 2 drugs, (Labetalol and Methyl-DOPA). On admission, she was well-oriented, pulse rate 100/minute, respiratory rate 20/minute, afebrile, blood pressure 160/100 mmHg. Bilateral pitting pedal oedema of grade II was noted.

## **Obstetric Examination**

Singleton Pregnancy corresponding to gestational age, cephalic presentation, relaxed feel. Next morning, she developed severe headache not responding to medication, flashes of light and blurring of vision. She was treated with general supportive measures and 4 grams of Injection Magnesium sulphate was loaded IV along with 1 gram per hour infusion to prevent seizures. NST was nonreactive and there was evidence of deteriorating renal function by laboratory parameters. Emergency Caesarean section was done under General anaesthesia in view of impending eclampsia, unfavourable cervix and preterm premature rupture of membranes. An unasphyxiated male baby of weight 3075 grams which cried soon after birth was handed over to the neonatologist for preterm care. Maternal ascites was noted. The patient was stable during the operation. Intraoperative hypertension was controlled with IV Labetalol .On shifting her to SICU, she was disoriented, irritable and complained of acute loss of vision following which she developed generalized tonic clonic convulsions. BP recording was 170/120 mmHg and was on Magsulph infusion for 36 hrs.

She was desaturating despite supplemental oxygen and elective ventilation done in view of recurrent seizures. BP was controlled and serial lab data were normal in postpartum Period. Vision improved within 4 days with complete recovery of vision. She was discharged home on day 8 and was off hypertensive medications. Fundoscopy was normal. CT Head showed abnormal patchy ill-defined hypodensities involving bilateral occipitoparietal subcortical

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white matter and basal ganglia. MRI Head, Figures (1) and (2) revealed areas of high signal throughout the white matter involving the parietal regions and bilaterally to involve occipital and temporal lobes suggestive of PRES.



Figure 1. MRI (1) and (2)

## **DIFFERENTIAL DIAGNOSIS**

-Stroke (thrombotic, embolic, haemorrhagic)

- -Venous thrombosis
- -Toxic / metabolic Encephalopathy
- -Vasculitis
- -Demyelinating disorder

## **CLINICAL DIAGNOSIS**

Posterior Reversible Encephalopathy Syndrome / Reversible Posterior Leukoencephalopathy

## DISCUSSION OF MANAGEMENT

Posterior Reversible Encephalopathy Syndrome / Reversible Posterior Leukoencephalopathy Syndrome is a neuroimaging finding of reversible vasogenic subcortical tissue oedema without gross infarction. PRES with eclampsia is a rare condition.

## Incidence

Common in young than elderly and in females than in males.  $^{1} \ \ \,$ 

## **Theories of PRES**

Earliest theory suggested that overreaction of cerebral autoregulation results in reversible vasospasm which in turn results in potentially reversible brain ischemia particularly in vascular border zone territories.<sup>2</sup> Newer theory suggest that autoregulation maintain a constant perfusion to cerebral nervous system; despite alterations in systemic BP. This is by arteriolar dilatation and constriction. When there is

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marked elevation of systemic BP, the constricted arterioles are forced to dilate resulting in hypoperfusion of the brain thereby breakdown of blood brain barrier resulting in extravasation of fluid, macromolecules and even RBC's into the brain parenchyma. So, PRES represents vasogenic oedema rather than cytotoxic oedema in majority of cases.<sup>3</sup> Another theory states that at intravascular pressure just below those could rupture the capillary wall permeability through the endothelium increased markedly, it was most likely due to increased pinocytotic activity through the wall of capillaries which may act to relieve intravascular pressure thereby resulting in development of large haemorrhages. So PRES is due to hydrostatic oedema in this case.<sup>4</sup>

## **CLINICAL FEATURES**

- Headache<sup>5</sup> (53%): constant, non-localised, moderate to severe, not relieved with analgesics.
- Altered consciousness (92%): Mild somnolence to confusion and agitation, may progress to coma.
- Visual disturbances<sup>6</sup> (39%): Blurred vision, hemianopia, aura, visual hallucination and cortical blindness<sup>7</sup> which is characterized by intact pupillary reflex and normal funduscopic findings. A high degree of suspicion is required in patients of late postpartum eclampsia because it occurs between 48 h postpartum and 1 month after delivery, frequently in women who have had a normal pregnancy, delivery, and no signs of preeclamptic syndrome.
- Seizures<sup>8</sup> (87%): Generalised tonic clonic seizures. Multiple seizures are more common than single events.

## The Diagnostic Criteria for Posterior Reversible Encephalopathy Syndrome<sup>9</sup> are-

- 1) The presence of neurologic symptoms or findings.
- 2) Presence of risk factors for PRES.
- 3) Absence of other possible causes of encephalopathy.
- 4) Reversible course on follow up.

## Investigation

Neuroimaging is essential for diagnosis of PRES which predominantly affect the posterior circulation territory. The radiologic finding is vasogenic oedema, and it is most common in the occipitoparietal regions.

CT head shows Bilateral white matter oedema and hypodensities in the posterior cerebral Hemisphere (white and grey matter). Brain MRI showed increased T2 and FLAIR (Fluid Attenuated Inversion Recovery<sup>10</sup> signal due to Cerebral oedema in posterior region of cerebral hemisphere. The preferential involvement of the parieto occipital lobes is thought to be due to the relatively poor sympathetic innervation of the posterior circulation. The increased signal is usually bilateral and symmetrical. But sometimes it may be single and unilateral. Lesions are mostly parieto occipital (98.7%) but temporal (68.4%), frontal (78.9%), cerebellum (34.2%), brainstem (18.4%) involvement can occur.<sup>11</sup> In approximately 56% of patients, vertebra-basilar territory such as thalamus and cerebellum are involved. Findings are not usually confined to a single vascular territory. With

prompt treatment, resolution of the findings is mostly observed. It has been found that bilateral symmetrical vasogenic oedema of parieto-occipital lobe was the most MRI abnormality<sup>12</sup> and no significant differences were seen in the severity of oedema between patients with and without visual loss.<sup>2</sup>

## Diagnosis

Treatment includes aggressive BP control,<sup>13</sup> manage underlying comorbidities and seizure Prophylaxis. The essence of controlling BP is, to reduce the mean arterial BP by 20-25% in first 2 hours and diastolic BP-100 mmHg as rapid reduction in BP can worsen end organ perfusion and cerebral function.<sup>14</sup> Systolic ( $\geq$ 160) / severe diastolic (>110) hypertension lasting >15 min is considered a hypertensive emergency. The degree of systolic hypertension may be the most important predictor of cerebral injury and infarction. Treatment goal is to maintain 140-160 / 90-100 mmHg and prevent repeated, prolonged exposure to severe systolic hypertension and loss of cerebral vasculature autoregulation. IV labetalol or IV hydralazine are the most commonly used drugs.

### Prognosis

Most cases of PRES are reversible<sup>15</sup> and have complete neurological recovery with removal of inciting factors and treatment of BP. Complete reversibility is the pathognomonic feature of PRES.

## FINAL DIAGNOSIS

In our case of PRES due to Eclampsia, Magnesium Sulphate is preferred agent for seizure prophylaxis due to neuroprotective effects by stabilization of both neuronal membranes and cerebral circulation. Obstetrician must be aware and vigilant and investigate suspicious acute cerebral complaints with radiological findings in antepartum/intrapartum/postpartum patients especially with hypertension.

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