

# Posterior Reversible Encephalopathy Syndrome in a Patient with Eclampsia

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## INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is a serious and a rare clinical neurological condition with the characteristic imaging findings. Here we describe a case of 30-year-old female, diagnosed with PRES secondary to maternal antepartum eclampsia. The diagnosis was established after magnetic resonance imaging (MRI) was done. Suspicion holds key towards early diagnosis and treatment to reverse PRES. Pre-eclampsia and eclampsia are the commonest causes of PRES.<sup>[1-4]</sup> Pre-eclampsia is a condition characterized by hypertension, albuminuria (300 mg/day) and oedema of the feet, hands and face. It commonly occurs in the third trimester of pregnancy.<sup>(1)</sup> The condition is characterized by poor placental perfusion and multiple organ systems involvement.<sup>(2)</sup> PRES can also occur due to other causes like hypertension, sepsis and multi-organ failure, acute kidney injury, chronic renal failure, immunosuppressive drugs (example: tacrolimus, cyclosporine, chemotherapy) and illicit drugs (like cocaine), organ transplantation and autoimmune diseases.<sup>(3)</sup> PRES presents with rapid onset of symptoms including headache, seizures, altered consciousness, and visual disturbance. It is usually associated with acute hypertension which causes vasogenic oedema by damaging cerebral vasculature, resulting in extravasation of proteins and fluids.<sup>[4-6]</sup> MRI brain shows characteristic imaging that includes sub-cortical vasogenic oedema at the bilateral occipital and parietal regions.<sup>(7),(8)</sup>

## PRESENTATION OF CASE

A 30-year-old patient, day 6 PNC with P1L1A2 came to emergency department with chief complaints of blurring of vision since 30 minutes with history of generalized tonic clonic seizures post-delivery. She had a history of similar episodes, twice in her ANC period in 2<sup>nd</sup> trimester. Her BP was on higher side for which she was advised antihypertensives (no documents available). Patient was not compliant to treatment protocol.

Patient had developed eclampsia at around 7.5 months of gestation for which she underwent emergency caesarean section in another hospital. She had delivered male 1.5 kg, baby who was shifted to NICU in view of prematurity and low birth weight. Post-delivery, patient had 3-4 episodes of GTCS. Her CT brain done post-delivery showed symmetrical gross white matter hypodensities involving bilateral, occipital and parietal area and external capsules on both sides. She was referred to our hospital for further management.

On examination in this hospital, she was conscious, oriented, obeying commands, moving all 4 limbs. Her pulse-98/min, BP -170/110 mmHg, Respiratory Rate-20/min. bilateral pedal oedema was present. On P/A examination her uterus was well retracted. Bleeding per vaginum minimal.

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*DOI: 10.14260/jemds/2019/787*

*Financial or Other Competing Interests:  
None.*

*How to Cite This Article:*

*Agarwal H, Inamdar A, Inamdar SA, et al.  
Posterior reversible encephalopathy  
syndrome in a patient with eclampsia. J.  
Evolution Med. Dent. Sci. 2019;8(48):3643-  
3645, DOI: 10.14260/jemds/2019/787*

*Submission 21-08-2019,  
Peer Review 28-10-2019,  
Acceptance 05-11-2019,  
Published 02-12-2019.*



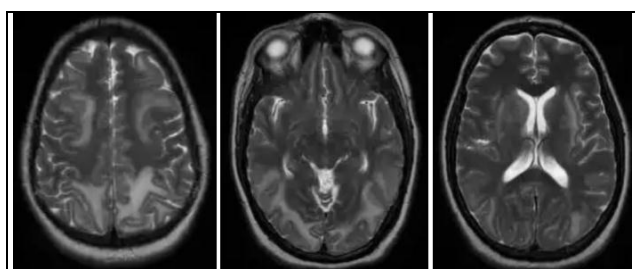
Central Nervous System examination revealed normal tendon reflexes in all four limbs, normal tone and power, pupils were 3 mm reacting to light with no evidence of any focal neural deficit.

Haemoglobin-12.7, White Blood Cells-16400/cumm<sup>3</sup>, Platelet count-2/08 lacs/mm<sup>3</sup>, ESR 80 mm in 1<sup>st</sup> hour, her urine albumin was 3+. Her antiphospholipid antibody profile was negative.

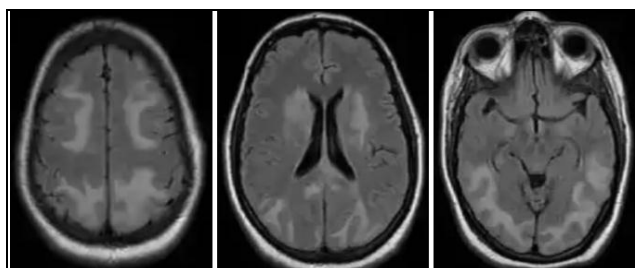
### DISCUSSION OF MANAGEMENT

After examination patient was administered inj. Mannitol 100 mg IV TDS, inj. Dexamethasone 4 mg I.V. TDS, inj. Ceftriaxone 1 gm I.V. BD, inj. Levipril 500 mg I.V. BD, tab. Telmisartan 40 mg po BD, tab. Prazosin 5 mg per oral BD. Patient was subjected for MRI which showed features of symmetrical areas of altered signal intensity in the fronto-parieto-temporal-occipital region involving the bilateral corona radiata and centrum semiovale appearing isodense on T1 W1, hyperintense on T2 and FLAIR and showing no diffusion restriction on DWI with no areas of blooming on GRE. Impression – posterior reversible encephalopathy syndrome. (Figure 1, 2, 3)

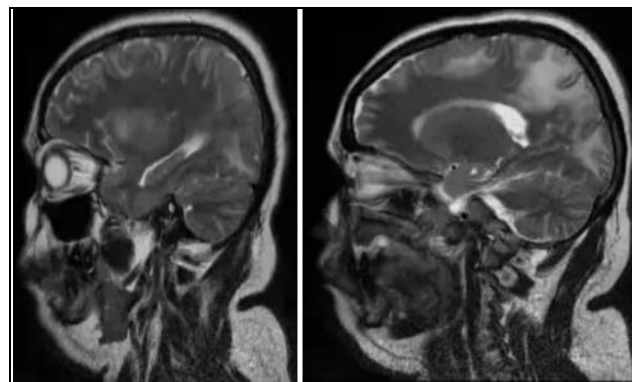
Patient was further continued with inj. Dexamethasone 4 mg I.V. TDS, inj. Levipril 500 mg BD, Tab. Prazosin 5 mg BD for 7 more days. There were no fresh GTCS episodes or any similar complaint. There was improvement in blurring of vision. Thus, patient had responded well to medical line of management. Her general condition gradually improved. She was shifted to general ward from medicine ICU. Her blood pressure was under control. Rest all vital signs were normal. No seizure activity was recorded during stay in hospital. Baby was kept in NICU in view of prematurity and low birth weight. Baby was managed with antibiotics, Orogastric feeds and other supportive measures. Patient was thus discharged and was advised follow up after 1 month for the same.



**Figure 1. Axial T2 Weighted MR Images Showing Symmetrical Areas of Increased Signal Intensity in Bilateral Corona Radiata, Centrum Semiovale and Fronto Parieto Occipital Region**



**Figure 2. MRI Flair Images Showing Increased Intensity in Bilateral Corona Radiata, Centrum Semiovale and Fronto Parieto Occipital Region**



**Figure 3. T2 Weighted MR Images Demonstrating Increased Signal Intensity in Bilateral Corona Radiata, Centrum Semiovale and Fronto Parieto Occipital Regions. There was No Diffusion Restriction on DWI, No Blooming on GRE s/o PRES**

### DISCUSSION

There are multiple factors associated with PRES which include pre-eclampsia/eclampsia, hypertension, immunosuppression agents, cancer chemotherapy, autoimmune disorders, infection, sepsis.<sup>(9)</sup> Pre-eclampsia and eclampsia are most commonly reported aetiologies. The exact pathology behind PRES is yet to be known. There exist two opposing pathophysiological mechanisms. First hypothesis being the vasospasm/hypoperfusion doctrine and the second hypothesis, is the hypertension/hyperperfusion hypothesis.<sup>(10)</sup>

This dysfunction is set to occur when Mean Arterial pressure rises above 150 mmHg and even higher in chronic hypertensive patients.<sup>(6)</sup> Irreversible damage occurs when mean arterial pressures rises above 200 mmHg <sup>(6)</sup>. Advanced imaging techniques including catheter angiography and MR angiography have been used for the assessment of PRES, that supports vasospasm/hypoperfusion hypothesis and/or string-of-bead appearance.<sup>(10),(11),(12)</sup>

The placenta plays an important role in the pathogenesis of preeclampsia however specific exact mechanism is unknown.<sup>(13)</sup> With the formation of the placenta, the placental ischemia causes more syncytial surface tissue apoptosis and necrosis and dropping off, and together with activated T-helper cells release large amounts of inflammatory cytokines, including tumour necrosis factor (TNF- $\alpha$ ), interleukin (IL)-1, interferon (IFN)- $\gamma$  and IL-6 into the maternal blood circulation causing severe maternal systemic immune response (toxaemia of pregnancy), thereby leading to systemic endothelial cell activation and injury. Excess of inflammatory mediators and vasoconstrictor substances are secreted by activated endothelial cells, induce diffuse systemic vascular contraction, that leads to vasogenic oedema in brain <sup>(13),(14)</sup> Cerebrospinal fluid is usually normal, though mildly elevated protein has been occasionally reported. <sup>(9)</sup>

Clinical features of PRES are highly non-specific that includes headache, vomiting, visual disturbances, altered mental status, seizures, and unconsciousness, most common ones being seizure and headache<sup>(9),(15),(16),(17)</sup>. Seizure are usually of generalized tonic clonic type<sup>(9),(18),(19)</sup> Visual manifestations includes blurred vision, visual neglect, hemianopia and cortical blindness.<sup>(9),(15)</sup>

MRI brain imaging findings of PRES include reversible vasogenic subcortical oedema at bilateral occipital lobes and parietal lobes. Other sites involved are Frontal lobes, temporal lobes and cerebellar hemisphere. Basal ganglia, brainstem and deep white matter can get involved but is relatively uncommon. Unilateral oedema can be seen rarely.<sup>(7),(10)</sup>

The diagnosis of PRES requires continuous observation of the clinical condition. cerebral infarction closely resembles it and hence wrong diagnosis can be made leading to delayed treatment.<sup>(15)</sup> When blood pressure is high, the vasoconstriction that occurs during autoregulation could exacerbate such a pre-existing condition, causing hypoxia leading to vasogenic oedema. This forms the basis of reversal of PRES by controlling blood pressure.

Early diagnosis and treatment hold key to the prognosis of PRES as delay in diagnosis can lead to permanent neurological deficits and sometimes can even lead to death. In cases where early and prompt treatment is given, patients usually recover within 1-2 weeks without any residual neurological deficits.<sup>(15),(20),(21)</sup> Treatment of the cause remains mainstay in treatment of PRES. For pre-eclampsia/eclampsia induced PRES, the prime treatment is to control blood pressure and prevention or treatment of seizure and termination of pregnancy when necessary.

### CONCLUSIONS

In antenatal patients with history of seizure disorder, apart from common causes of eclampsia other causes should be sought for. Diagnosis of PRES requires a high index of suspicion and should be promptly diagnosed and treated.

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