

# CASE REPORT

## NEUROLOGICAL INVOLVEMENT IN ECLAMPSIA

Anita Pal<sup>1</sup>, Rama Thakur<sup>2</sup>, Yogita Dogra<sup>3</sup>, Vijay Thakur<sup>4</sup>, Suman Thakur<sup>5</sup>

### HOW TO CITE THIS ARTICLE:

Anita Pal, Rama Thakur, Yogita Dogra, Vijay Thakur, Suman Thakur. "Neurological Involvement in Eclampsia". Journal of Evolution of Medical and Dental Sciences 2014; Vol. 3, Issue 67, December 04; Page: 14537-14541, DOI: 10.14260/jemds/2014/3950

**ABSTRACT:** Eclampsia & severe preeclampsia have neurological manifestations. Serum Lactate dehydrogenase (LDH) levels are more in the group that later develops hypertensive encephalopathy. Posterior reversible leukoencephalopathy syndrome (PRES) is a clinical and radiological entity presented by headache, altered mental state, cortical visual disturbances & seizures with transient edematous changes of subcortical white matter. Neuroimaging is must in late postpartum eclampsia

**KEYWORDS:** Preeclampsia, eclampsia, posterior reversible encephalopathy syndrome, neuroimaging.

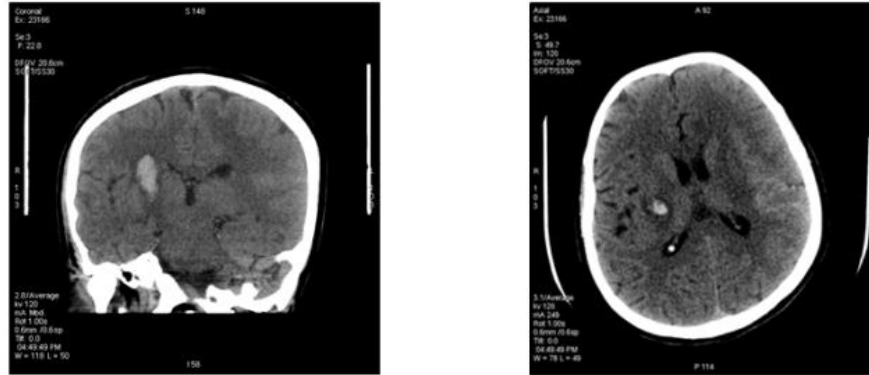
**INTRODUCTION:** Despite availability of intensive care unit & improved antenatal care still some women die of eclampsia. Incidence is 1.8% in developed countries & 14 % in developing countries. The most common cause of death is cerebral complications.<sup>1</sup> Neurological manifestations of pregnancy induced hypertension vary from diffuse symptoms such as headache and confusion to focal signs such as paralysis and visual loss. Computerized tomography (CT) Scan and magnetic resonance imaging (MRI) have greatly enhanced our understanding of the correlation between neurological complaints and neuroanatomic pathological changes characteristic of Preeclampsia (PE) & Eclampsia (E).<sup>2</sup> We report 2 cases of eclampsia with findings of PRES on neuroimaging.

### CASE REPORTS:

**PATIENT 1:** A 38 years old G3P1+1 attended the antenatal outpatient department at Kamla Nehru Hospital, Indira Gandhi Medical College, Shimla at 37+<sup>3</sup> weeks with breech presentation and her BP record was 144/100mm Hg with proteinuria (urine albumin 1+) for which she was admitted. She had no signs and symptoms of imminent eclampsia. Her antenatal period was supervised adequately and was apparently uncomplicated. Her blood sugars were normal. She had first increased BP record of 132/90mmHg at 35 weeks without proteinuria. Her ultrasounds at regular intervals were normal. She had previous one full term normal vaginal delivery 16 years back followed by one spontaneous abortion. There was no significant past and family history. Laboratory investigations revealed normal platelet count and renal function tests, SGOT-52, SGPT-63, LDH-403.

Elective caesarean section with bilateral abdominal tubectomy was done and a healthy baby was delivered. Post-operative BP records were 150/96-160/94 mmHg with adequate urine output. She threw a generalized tonic clonic seizure with frothing at 7 hours postoperatively. Her BP was 220/140mmHg with urine albumin 2+ at that time. Inj MgSO<sub>4</sub> was started and maximum (220mg) dose of Labetalol was given intravenously but she remained agitated, disoriented with no response to verbal commands. Inj Mannitol was also given. She threw another convulsion which was followed by loss of consciousness. She was continued on Inj MgSO<sub>4</sub> and Labetalol. MRI brain revealed Subacute hematoma posterior limb of right internal capsule and right basal ganglia with Subarachnoid hemorrhage in left fronto-temporo-parietal region with aneurysm of left Posterior Cerebral Artery. She was then managed conservatively with antihypertensives, Inj mannitol and tablet phenytoin.

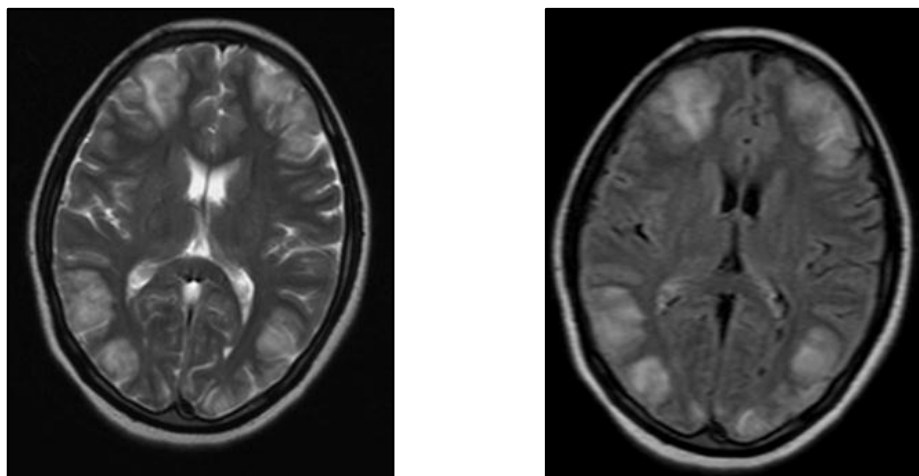
## CASE REPORT



Axial (Left) and Coronal (Right) non-contrast CT Scan brain shows hyperdensity s/o haematoma in Right basal- ganglia region with sub- arachnoid hemorrhage in Left fronto-temporo-parietal regions.

**PATIENT 2:** A 20 years old unbooked primigravida reported to labour room at Kamla Nehru Hospital, Shimla with 8 months amenorrhea and chief complaints of labour pains for 4 hours, H/O headache, vomiting x 2 days and 8-9 episodes of seizures with up rolling of eyeballs and urinary incontinence. On examination she was semi-conscious, agitated, not responding to verbal commands but to painful stimuli; PR-104/min; BP-150/120mmHg; RR- 24/min and Urine albumin- 1+. Per abdomen examination revealed cephalic presentation, uterus contracting with no fetal heart sound. Inj MgSO<sub>4</sub> was started and Labetalol was given intravenously.

She delivered a macerated still born baby. Laboratory investigations revealed normal platelets and renal function tests, Total bilirubin-1.76, SGOT-192, SGPT-168, ALP-912, LDH-710. She had persistent comatose state postpartum. Dilantinization was done. MRI brain showed Posterior Reversible Encephalopathy Syndrome. Fundus examination showed evidence of retinopathy. Inj unfractionated heparin (UFH 5000 IU s/c BD) was started. She was then managed conservatively with inj. Mannitol, Inj Phenytoin & anti hypertensives.



Axial T2 weighted (Left) and FLAIR weighted (Right) MR Images show hyperintense signals in bilateral frontal and parieto-occipital regions suggestive of PRES.

## CASE REPORT

---

**DISCUSSION:** Preeclampsia is a syndrome unique to human pregnancy featuring hypertension and proteinuria, usually occurring after 20 weeks. Eclampsia is defined as seizures before, during pregnancy or post-partum in patients with PE.<sup>3</sup> Placental disorders like poor placentation and hyperplacentosis is the main aetiological factor.<sup>4</sup> In pathophysiology there are three primary problems:

- a) Endothelial cell injury.
- b) Systemic vasospasm.
- c) Progressive developmental of systemic oedema.<sup>5</sup>

Cerebral microcirculation is the major target. Disordered cerebral auto regulation due to acute fluctuation in blood pressure and endothelial dysfunction is the main cause of neurological symptoms.<sup>6</sup> Cerebral lesions are in the posterior circulation and watershed zones which are sparsely innervated by sympathetic nerves.<sup>7</sup> Hypertensive encephalopathy is characterized by rapidly progressive signs and symptoms such as headache, nausea, vomiting, visual symptoms, confusion, focal neurological signs and seizures. Magnetic Resonance Imaging has been useful in demonstrating parieto-occipital high signal intensities involving the cortex and the subcortical white matter.<sup>3</sup>

Reversible posterior encephalopathy syndrome (PRES), or as the previous name, parieto-occipital encephalopathy or reversible posterior leuko encephalopathy refers to a clinical and radiological entity presented by headache, altered mental state such as confusion, lethargy, cortical visual disturbances and seizures with transient edematous changes of subcortical white matter on neuroimaging.<sup>8</sup> A CT /MRI Imaging of brain typically demonstrates focal regions of symmetric hemispheric oedema, parietal and occipital lobes are commonly affected followed by the frontal lobes, inferior temporal occipital junction and the cerebellum.<sup>9</sup>

Other cases in which neuroimaging is must is late post-partum eclampsia which is, eclampsia occurring >48 hrs but <4 weeks after delivery. These patients may not present with all the classical symptoms of intrapartum PE. They usually have lower BP, minimal proteinuria and significantly higher incidence of neurologic deficits than those with early onset eclampsia.<sup>10</sup>

MRI is recommended not only for symptomatic patients with suspected PRES but also for asymptomatic patients with severe PE. If cerebral oedema is demonstrated on MRI, then it is important to consider immediate delivery before eclamptic seizures or exacerbation of neurological symptoms occur.<sup>11</sup>

Eclampsia patients who are refractory to MgSo4 & antihypertensive therapy have significant CNS pathology.<sup>1</sup>

RBC morphology is the strongest predictor of abnormal radiographic findings. The only laboratory parameter that has been found to be abnormal a week prior to the development of neurological symptoms is serum LDH level which is higher in the group that later developed hypertensive encephalopathy related brain oedema.<sup>12</sup>

**CONCLUSION:** Various neuroimaging techniques have been utilized to evaluate eclamptic patients. Cerebral angiography is capable of demonstrating large haemorrhages, arterio-venous malformations & mass lesions, but is a potentially risky procedure in severely hypertensive gravida. CT can be performed with minimal risk, although it has a limited capacity. MRI has been shown to have better sensitivity & specificity in diagnosing lesions in deep white matter & basal ganglia, it correlates better with the clinical findings & it is safe in pregnant women.

## CASE REPORT

---

### REFERENCES:

1. Patil Mithil M. Role of Neuroimaging in Patients with Atypical Eclampsia. *J of Obstet & Gynecol of India* 2012; 62 (5): 526-30
2. Roybart M, Seidman DS, Serr DM, Mashiach S. Review. Neurological Involvement in Hypertensive Disease of Pregnancy. *Obstet & gynaecol Survey* 1991; 46(13): 656-64.
3. Mehmet A, Osmanagaoglu, Gulseren DINC, Selen OSMANAGAOLU, Hason DINC, Hason DINC, Hason BOZKAYA Comparison of cerebral magnetic resonance and electroencephalogram findings in pre-eclamptic and eclamptic women. *Australian & New Zealand J of Obstet & Gynecol* 2005; 45: 384-90.
4. Chakravorty A, Chakrabarti S. D., The Neurology of Enclamsia: Some Observations. *Neurol India*, 2002; 50: 128-35.
5. Bartynski WS, Sanghvi A, Neuroimaging of Delayed Eclampsia. *J Comput Assist Tomogr* 2003; 27: 699-713.
6. Diwan AG, Agrawal PH, Panchanadikar TM, Yadav SV, Joshi P. Late post-partum eclampsia with reversible encephalopathy syndrome: an interesting case. *Indian J of Medical Specialities* 2012
7. Demirtas O, Gelal F, Vidinli BD, Demirtas LO, Uluc E, Baloglu A. Cranial MR Imaging with clinical correlation in preeclampsia and eclampsia. *Diagn Interv Radiol* 2005; 11: 189-94.
8. Chiou YH, Chen PH, Reversible Posterior Encephalopathy Syndrome as the Presentation of Late Post Partam Eclampsia: A case Report. *Acta Neurologica Taiwanica* 2007; 16 (3):158-62.
9. Bartynski WS, Posterior Reversible Encephalopathy Syndrome, Part I: Fundamental Imaging and Clinical Features. *Am J Neuroradiol* 2008; 29: 1036-42.
10. Vertkamp R, Kupsch A, Polasek J. Late Onset Post partam eclampsia without pre-eclamptic prodromi: Clinical and neuroradiological presentation in two patients. *J Neurol Neurosurg Psychiatry*. 2000; 69:824-27
11. Ekawa Y, Shiota M, Tobiume T, Shimaoka M, Tsuritani M, Kotani Y etal. Reversible Posterior Leukoencephalopathy Syndrome Accompanying Eclampsia: Correct Diagnosis using Preoperative MRI. *Tohoku J.Exp. Med* 2012; 226: 55-58.
12. Richard BS, Steven KF, Joseph FP, De Girolami U, Lalia A, Beckner KM. Preeclampsia-Eclamsia: Clinical and Neuroradiographic Correlates and Insights into the Pathogenesis of Hypertensive Encephalopathy. *Radiology* 2000; 217: 37.

## CASE REPORT

---

### **AUTHORS:**

1. Anita Pal
2. Rama Thakur
3. Yogita Dogra
4. Vijay Thakur
5. Suman Thakur

### **PARTICULARS OF CONTRIBUTORS:**

1. Professor, Department of Obstetrics and Gynaecology, Kamla Nehru State Hospital, IGMC, Shimla.
2. Assistant Professor, Department of Obstetrics and Gynaecology, Kamla Nehru State Hospital, IGMC, Shimla.
3. Senior Resident, Department of Obstetrics and Gynaecology, Kamla Nehru State Hospital, IGMC, Shimla.
4. Associate Professor, Department of Radiodiagnosis, IGMC, Shimla.

5. Assistant Professor, Department of Obstetrics and Gynaecology, Kamla Nehru State Hospital, IGMC, Shimla.

### **NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:**

Dr. Rama Thakur,  
Assistant Professor,  
Department of Obstetrics and Gynaecology,  
Kamla Nehru State Hospital,  
IGMC, Shimla, H. P, India.  
Email: ramanonu@gmail.com

Date of Submission: 20/11/2014.  
Date of Peer Review: 21/11/2014.  
Date of Acceptance: 28/11/2014.  
Date of Publishing: 04/12/2014.