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CASE REPORT

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# Posterior Reversible Encephalopathy Syndrome (PRES) in Two Patients with Severe Preeclampsia

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## Introduction

Posterior reversible encephalopathy syndrome (PRES) is first described by Hinchey et al in 1996 [1]. PRES has become a more recognized medical term due to alerting neurologic symptoms it has and also readily available imaging techniques that enable doctors to make the diagnosis. It is characterized by headaches, seizures, nausea or vomiting, vision problems due to posterior cerebral white matter edema but the syndrome is not always reversible and it is often not confined to either the white matter or the posterior regions of the brain [2,3]. The incidence of PRES is not known. The etiology of those disturbances may vary in a wide range including hypertension, kidney failure, preeclampsia/eclampsia, collagen vascular diseases, sepsis, consumption of immunosuppressive agents, etc. It is more common in women even when patients with eclampsia are excluded [1-3]. As definition, preeclampsia is a systemic vascular disorder characterized by new-onset hypertension and proteinuria after 20 weeks of gestation and eclampsia is defined as a seizure occurring in association with preeclampsia.

Prompt recognition and treatment of the syndrome are important in preventing of the permanent damage that can occur in this otherwise typically reversible condition. The management of PRES mostly based on the investigation and resolution of the underlying cause. If the underlying cause is diagnosed and treated effectively, clinical symptoms may subside in a week [1,2].

There are a few theories to explain the pathophysiology of PRES. The most popular theory is that the autoregulation of the blood perfusion in brain vessels disrupted due to uncontrolled hypertension [2]. Sudden elevations in blood pressure cause extravasation of proteins to the intercellular area and this results in vasogenic edema . However, this theory does not explain the cerebral hypoperfusion which sometimes can be seen in PRES and why PRES may occur in normotensive patients as well . In this perspective, the association of PRES with systemic inflammatory conditions such as autoimmune diseases, sepsis or preeclampsia revealed a theory claims that PRES might be associated with endothelial dysfunction [4]. Compatible with both theories, the pathogenesis of preeclampsia or eclampsia make a predisposition to developing PRES syndrome. Therefore, it is a potentially devastating disease that obstetricians should be prepared in preeclamptic pregnant women.

In these case presentations, we aimed to present two preeclamptic women who recovered without any sequele in intensive care unit after developing PRES.With this article, we would like to show the importance of early diagnosis and optimal management of entity.

## Case Presentation

### Case 1

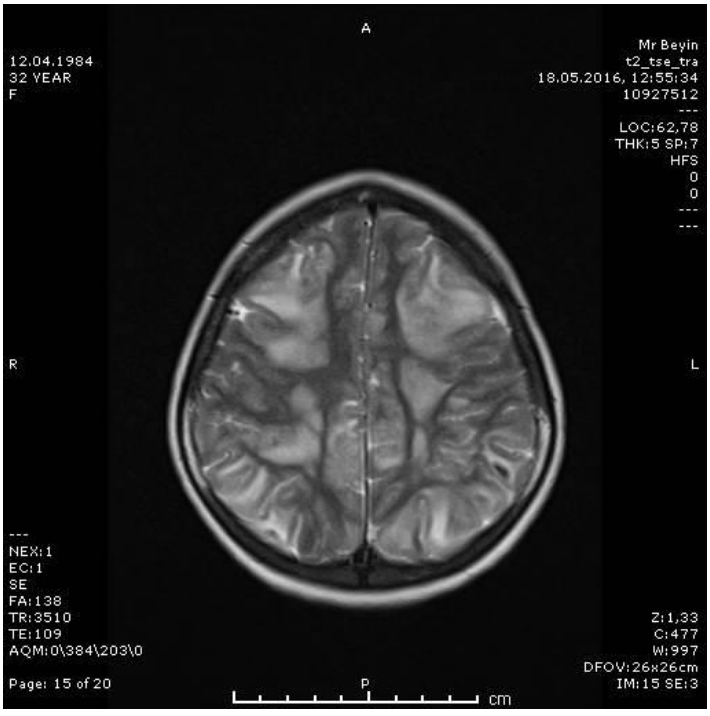


**Figure 1.** Hypointense lesions on T2- weighted sections

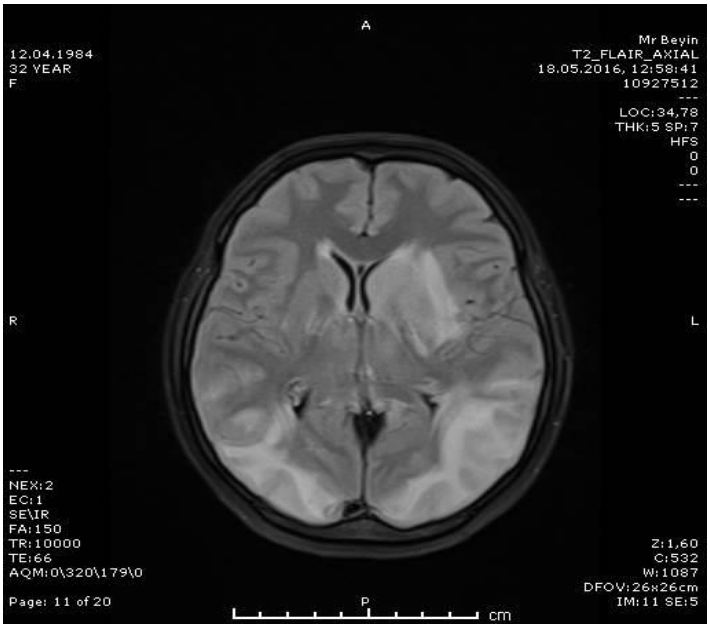


**Figure 2.** Hypointense lesions on FLAIR sections

32 years old primigravida woman has been admitted at 35 weeks of gestation due to hypertension of 170/100.Her hemoglobin level, hematocrite,white blood cell count and platelet level were 9.6 g/dL, 32%, $9.25x10^3/\mu L$ and 155 $10^3/\mu L$ respectively.Her liver function enzymes, namely ALT and AST were being 35 and 47 U/L. She had +1 proteinuria in urine analysis and her fundoscopic exam was in normal limits without having any sign of intracranial hypertension. Her high blood pressure was taken under control with 250 mg of alpha methyl dopa per 6 hours. The intrauterin growth retardation is detected in fetus according to ultrasonographic measurements which were consistently indicating 31 weeks of gestation.Betametazon injections were applied in order to support the pulmonary function. During her prenatal care, late decelerations resistant to resuscitations occurred and she underwent an urgent cesarean section due to the diagnosis of fetal distress. She did not have any complications in postoperative 48 hours. At day 3, her blood pressure raised to 180/110 and subsequent generalized seizure with urinary incontinence occurred. Parenteral magnesium and perlinganit infusion were promptly started. Urgent neurology, internal medicine, and anesthesia consultations were done. 4 mg steroid injection two times per day, metabolic panel, fibrinogen, and peripheral blood smear were ordered. Liver enzymes came up to be elevated 10 times (AST: 562 U/L, ALT: 199 U/L), fibrinogen level was found to be slightly low (198 mg/dl) and platelet counts suddenly decreased to 44.000. In the peripheral blood smear, hematologist found acanthocytes too many to count and the actual platelet number was 34.000. HELLP syndrome is diagnosed and the patient was transferred to intensive care unit (ICU). 1 volume of plasmapheresis was planned. Meanwhile, she became sluggish and her consciousness gradually deteriorated. She was not opening eye nor to verbal command either to pain. Her Glasgow Coma Score was 6 (1 for no eye-opening, 1 for no verbal response and 4 for withdrawal from pain). Urgent cranial MRI and MRI venography were carried out. Diffuse high signal intensity on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images were interpreted to be edematous lesions located in the cerebellum and through the temporoparietal junction to the cortex of the brain. The lesions were found to be compatible with posterior reversible encephalopathy syndrome. 200 cc Mannitol IV push is administered to decrease extracellular fluid retention in the brain. Close observation is undertaken with an intermittent metabolic panel and coagulation markers. After the completion of plasmapheresis, prednol 1 mg/day per kilogram is ordered. Her liver enzymes and coagulation markers were gradually normalized. With the anti-edematous and supportive treatment patient clinically improved in 3 days and she was out of ICU on postoperative day 8. After spending 3 days in inpatient service for full recovery, she was discharged to home (Figures 1,2).

**Case 2**

28 years old nulliparous woman was admitted for the diagnosis of preeclampsia and in-utero growth retardation (IUGR) at the gestational age of 28 weeks and 1 day pregnancy.Her initial blood pressure was 150/110 mmHg and there was +3 protein in urine analysis. Abdominal circumference of fetus was in 3rd percentile and there were notches in the bilateral uterine arteries. Her initial liver function enzymes were in the normal limits (ALT:21,AST:20 U/L),platelet count was 175.000 and proteinuria in 24 hour urine was 9149 mg. 2 consequent doses of 12 mg betametazon injections were applied for pulmonary maturation. As severe preeclampsia is diagnosed and the blood pressure increased up to 160/90 mmHg, 2 gr/h of magnesium infusion was started. She is taken to the close observartion of perinatalogist. A day later, her blood pressure peaked to 170/90 mmHg and it was controlled by the administration of 5 mg intravenous Hydralazine. At the 4th day of hospitalization, the doppler examination of umblical arteries were normal but there was minimal ascites in the maternal abdomen. By day 5, she experienced prodrome findings such as generalized headache and scotomas in the visuel field and the blood pressure elevated once again to 170/90 mmHg. Since she developed prodromal symptoms due to severe preeclampsia, she underwent urgent C-section. There was no complications during or the first 24 hours of surgery. 2 gr/h of magnesium infusion was continued when the blood pressure elevated to 170/90 and she reported diplopia. Diplopia was regressed and her blood pressure was stabilized after alpha-methyldopa 4x250 mg regimen. However, at postoperative day 2, she developed a generealized tonic-clonic seizure, her blood pressure was 200/110 mmHg, pulse was 98 and oxygen saturation was 91%. Airway is applied immediately, oxygen flow was started at the rate of 10 lt/min. She was protected from the sharp edges at the surrounding and bilateral IV routes were inserted. Loading dose of magnesium and 5 mg of hydralazine were pushed. The blood pressure regressed to 180/90 mmHg and the seizure came to the end. Then she developed post-ictal agitation and diazem 5 mg was administered intramuscularly.15 minutes later, the blood pressure was 170/90 mmHg. Another 5 mg of hydralazine was administered.10 minutes later, her blood pressure was 150/90 mmHg and she regained orientation and cooperation. Her antihypertensive regimen was changed from alpha-methyl dopa to amlodipine 5 mg 2x1 and the highest blood pressure was 150/90 mmHg during the consequent 20 hours. In postoperative day 3, she was consulted to neurology as she developed generalized headaches once again. BT scan was performed and her fundoscopic examination found to be in normal limits with no signs of

papiledema or findings of hypertensive retinopathy. Since her cranial BT showed a hypodens lesion at the site of Posterior Cerebral Artery (PCA), diffusion MRI was ordered immediately in order to investigate PRES syndrome related to eclampsia or other differential diagnoses. MRI showed hyperintensity in the perfusion area of PCA which is found to be compatible with PRES syndrome. Mannitol 100 mg 4x1 and dexamethasone 2x 8 mg infusions were started per neurology consultation. As her neurologic symptoms continued during the succeeding 5 hours, she was consulted to neurology again. Lumbar puncture (LP) and empiric antibiotherapy were ordered for the possibility of meningitis. Because of persistent hypertension, lethargy and diplopia for more than 6 hours, she was transferred to ICU for monitorization of mental status and vitals. During her stay in ICU, her meningeal irritation findings were regressed and it was decided to withold LP procedure. The persistant hyperintensive lesions in the left occipital and bilateral frontal lobes in the second cranial MRI confirmed the vasogenic edema due to PRES. After her consciousness got improved and neurologic examination became normal, she was transferred back to inpatient service in the postoperative day 8 (Figure 3).

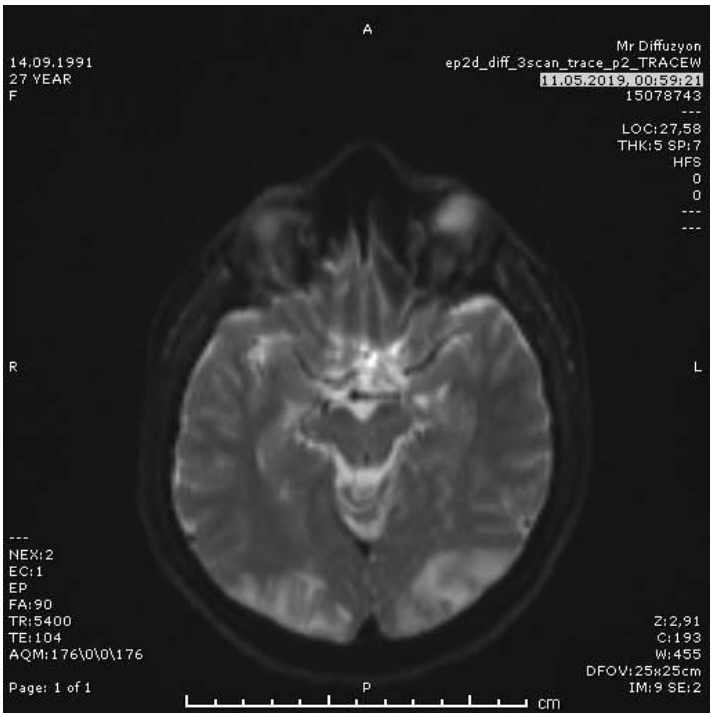


**Figure 3.** Hyperintense lesions on diffusion-weighted MRI

## Discussion

In our cases, PRES syndrome most possibly occurred secondary to late postpartum eclampsia (LPE) . In LPE, the seizures are seen 2 days to 4 weeks after the delivery. LPE can be challenging to diagnose since those pregnant women may not have obvious findings of preeclampsia such as high blood pressure or proteinuria but both of those patients had an obvious preeclampsia presentation.

Preeclampsia is attributed to generalized endothelial dysfunction of the vessels due to the cytotoxic trophoblastic factors that originated from the placenta. It is still unknown that if this damage in the endothelial lining is the cause or the result of hypertension. In both situations, the blood-brain barrier disruption causes fluid and protein transudation to the intercellular area and this may lead to brain edema. Also, the cerebral autoregulation of blood perfusion cannot be sustained after repetitive abrupt hypertensive changes in brain blood vessels and this situation further increases the interstitial fluid and exacerbates vasogenic edema [5,6]. This mechanism also explains the possible pathogenesis of seizures seen in our patients.

As the name indicates, the lesions of PRES are generally located in the posterior territory of the brain. This is thought to be secondary to decreased sympathetic innervations around the basilar artery and its branches. The classic response of arterioles to hypertension is vasoconstriction in order to limit hyperperfusion and possible extravasation of plasma. This response is regulated by sympathetic nerves and the posterior regions that have relatively scarce sympathetic innervations become more prone to develop vasogenic edema [7,8].

Although the relationship between Press and preeclampsia/eclampsia has not been clearly explained, we believe that the trophoblastic cytokines caused generalized endothelial dysfunction and played a major role in the development of PRES similar to our cases with preeclampsia and eclampsia [4].

The management of PRES should be based on the underlying pathology. In our cases, the definitive treatment of preeclampsia which is the delivery of the baby was already carried out. Prompt blood pressure control and regulation of electrolyte disturbances are the mainstays of treatment. To decrease the cerebral edema and prevent convulsions, we administered anti-edematous medications such as mannitol and steroids (methylprednisolone/dexamethasone). Anti-convulsive agents are also suggested for symptomatic treatment.

In conclusion, PRES should always be considered as one of the differential diagnosis in preeclamptic or eclamptic women who present with altered mental status, convulsions or headaches. Prompt diagnosis and treatment of underlying cause will likely expedite the resolution of cerebral edema and full recovery is expected in days to weeks.

## References


1. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, et al. A reversible posterior leukoencephalopathy syndrome. *N Engl J Med*. 1996; 334: 494–500.
2. Roth C, Ferbert A. The posterior reversible encephalopathy syndrome: what's certain, what's new? *Pract Neurol*. 2011; 11: 136–144.
3. McKinney AM, Short J, Truwit CL, McKinney ZJ, Kozak OS, et al. Posterior reversible encephalopathy syndrome: incidence of atypical regions of involvement and imaging findings. *AJR Am J Roentgenol*. 2007; 189: 904–912.
4. Bartynski W. Posterior reversible encephalopathy syndrome, part 2: controversies surrounding pathophysiology of vasogenic edema. *AJNR Am J Neuroradiol*. 2008; 29: 1043–1049.
5. Schwartz RB, Feske SK, Polak JF, DeGirolami U, Iaia A, et al. Preeclampsia-eclampsia: clinical and neuroradiographic correlates and insights into the pathogenesis of hypertensive encephalopathy. *Radiology*. 2000; 217: 371-376.

6. Savvidou MD, Hingorani AD, Tsikas D, Frölich JC, Vallance P, Nicolaides KH. Endothelial dysfunction and raised plasma concentrations of asymmetric dimethylarginine in pregnant women who subsequently develop pre-eclampsia. *Lancet*. 2003; 361: 1511-1517.
7. Bartynski WS. Posterior reversible encephalopathy syndrome. Part 1. Fundamental imaging and clinical features. *Am J Neuroradio*. 2008; 29: 1036–1042.
8. The Eclampsia Trial Collaborative Group.Which anticonvulsant for women with eclampsia? Evidence from the collaborative eclampsia trial. *Lancet*. 1995; 345: 1455-1463.

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