Indian Journal of Medical Case Reports ISSN: 2319–3832(Online)
An Open Access, Online International Journal Available at http://www.cibtech.org/jcr.htm
2017 Vol.6 (3) July-September, pp. 16-19/Maheshwari
Case Report

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME WITHOUT SEIZURE IN A WOMAN WITH LATE POSTPARTUM ECLAMPSIA

***R. Uma Maheshwari**
No 20, MIG, Vallal orhi St, NH 1, Maraimalai Nagar, Kanchipuram District – 603209
**Author for Correspondence*

ABSTRACT
Posterior reversible encephalopathy syndrome (PRES) also known as Reversible Posterior Leukoencephalopathy Syndrome (RPLS), is a syndrome characterized by headache, confusion, visual disturbances and seizures. It may occur due to a number of causes predominantly malignant hypertension, eclampsia and some medical treatments. MRI is gold standard for diagnosing cerebral changes due to PRES. We report a 31-year-old primi with a no history of preeclampsia during pregnancy complicated by PRES without seizures at the postpartum period. Complete resolution without any complications was observed on the 8th day after delivery. Early recognition and proper treatment results in complete reversibility of this condition.

Keywords: *Posterior Reversible Encephalopathy Syndrome, Pre-Eclampsia, MRI*

INTRODUCTION
Posterior reversible encephalopathy syndrome (PRES) also known as Reversible Posterior Leukoencephalopathy Syndrome (RPLS), presents with neurologic signs and symptoms such as headache, altered consciousness, seizures, visual loss etc. is often associated with abrupt increase in blood pressure (BP) (Fugate *et al.*, 2010).
This clinico-neuro-radiologic entity described by Hinchey has unique neuroimaging findings of vasogenic edema that are often symmetric, affecting the parietal and occipital lobes. Other areas involved are basal ganglia, frontal lobes, the inferior temporal-occipital junction and the cerebellum (Bartynski, 2008). Here, we present a case of primi with uneventful antenatal course who developed PRES in the postpartum period.

CASES
Ms. V S is a 31-year-old female, P1L1 who had a vaginal delivery at 36 weeks due to PPROM (preterm premature rupture of membrane). She didn't have pre-eclampsia and gestational diabetes during her antenatal period.
Her intrapartum and immediate postpartum periods were uneventful. Her BP during labor and ring hospital stay was 100-120/70-80mmHg. Patient was discharged on the 4th postnatal day. On the 5th postnatal day she came with complaints of palpitation and swelling both legs.
On examination her heartrate was 43 per minute and BP (blood pressure) 140/90 mmHg. Bilateral pedal edema was present.
Her ECG showed sinus bradycardia of 43 per minute and BP 140/90 mmHg. Cardiologist opinion was obtained and ECHO showed ejection fraction (EF) of 60%, with trivial MR and TR. Patient was treated with T. Oxeprenalin 10 mg BD. 2 hours later she complained of headache and neck stiffness. Her BP was 180/90 mmHg with persisting bradycardia of 45/min. She was hospitalized and was given Tab. Amlodipine 5 mg stat for blood pressure. Baseline investigations were done which included liver function tests, renal function test, serum uric acid, coagulation profile and urine analysis. No significant abnormalities were detected (Table 1, 2).
MRI brain was done which showed focal subcortical T2 flare hyper-intensity in left posteroparietal region. No significant abnormality was found in MR angiography of neck and MR venogram of brain (image 1).

Table 1: Blood Investigations

Blood Parameters	Value	Blood Parameters	Value
Uric Acid	4.2 mg/dl	**SGOT**	32 U/L
Creatinine	0.5 mg/dl	**SGPT**	29 U/L
BUN	6 mg/dl	**ALP**	142 U/L
Sodium	142 mmol/l	**Total Protein**	6.3 g/dl
Potassium	4 mmol/l	**Albumin**	3.8 g/dl
Chloride	111 mmol/l	**Globulin**	2.5 g/dl
Bicarbonate	22 mmol/l	**Total Bilirubin**	0.37 mg/dl
Partial Thromboplastin Time	29.4 Seconds	**Direct Bilirubin**	0.13 mg/dl
LDH	298.20 U/L	**Total count**	12300 cells/cumm
Fibrinogen	377.1 mg/dl	**RBC count**	4.26 million/cu mm
Hemoglobin	11.2 gms/dl	**Platelet count**	3.09 lac/cu mm

Table 2: Urine Analysis

PH	8	Bilirubin	Negative
Specific gravity	1.010	Ketone	Negative
Urine protein day 6 post-partum	Negative	Pus cells	2-3 /HPF
Urine protein day 7 post-partum	Negative	RBCs	nil
Urine protein day 8 post-partum	Negative	Epithelial cells	2-3 /HPF

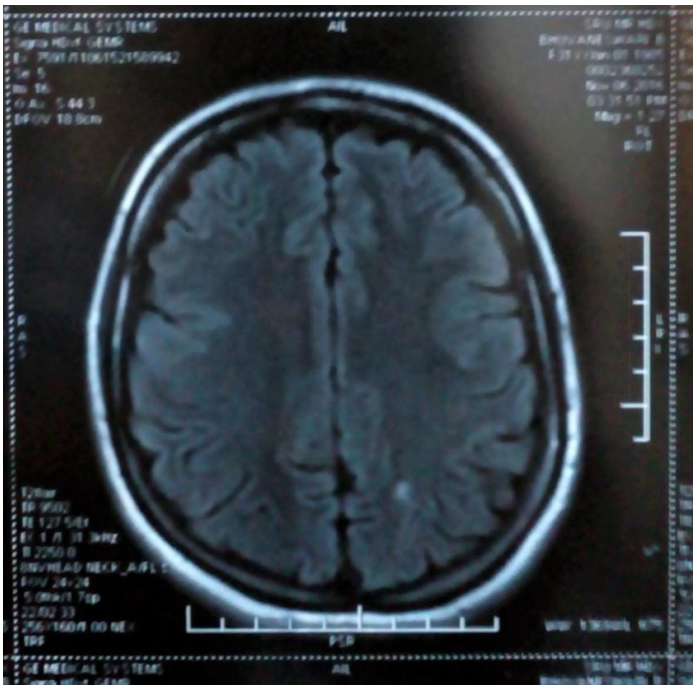

Image 1: MRI Brain

Focal subcortical T2 flare hyper-intensity in left posteroparietal region present.
Neurologist's and ophthalmologist's opinion were obtained. Patient was started on T.amlodipine 5 mg BD. Blood pressure reduced to 130/80 mmHg and heart rate was 60 / min after 6 hours of observation. Patient being symptomatically better with no complaints of palpitation or headache was discharged on 8th postpartum day (table 3). She was advised to continue T. amlodipine 2.5 mg BD till the end of puerperal period and come for review after one week.

Table 3: Blood Pressure (BP)

BP during pregnancy	110-100/70-80 mmHg
BP during labor	110/80 mmHg
BP on 1 - 3 postpartum days	120-100/80-70 mmHg
BP on day 5 postpartum(with bradycardia and headache)	180-140/100-90mmHg
BP on day 6 & 7 postpartum (after treatment)	130-110/90-70 mmHg
BP on day 8 postpartum(discharge)	110/70 mmHg

Follow-up

The patient was advised home monitoring of BP and was reviewed weekly for 2 weeks, followed by biweekly till 6 weeks period. Her BP had normalized (100–120/70– 80mmHg). She didn't complain of headache or neck stiffness.

DISCUSSION

PRES is seen, not only in relation to preeclampsia, but in a variety of conditions like post-transplant, immunosuppressant therapy, cancer chemotherapy, infection/sepsis and autoimmune diseases. Global incidence of PRES is unknown. It has been reported inpatients aged 4-90 years, although the most cases occur in age group of 39-47 years with a marked female predominance.

In PRES associated with pregnancy-induced hypertension, it is thought that pregnancy itself predisposes to cerebral edema, particularly in late-pregnancy. It is suggested that in pregnancy increased BP, neurotransmitters, increased vascular endothelial growth factor (VEGF) receptor, placental growth factor and endothelial dysfunction may have a role in PRES. While as general rule preeclampsia-eclampsia treatment is achieved with delivery, for unknown reasons, PRES might be developed in the postpartum period. The typical MRI findings in PRES is T2-weighted and FLAIR sequences showing hyperintense focilocated bilaterally at the grey-white junctions, involving subcortical white matter of most parts of brain; although it was earlier believed to mainly affect the posterior parietal and occipitallobes.

The pathophysiology of PRES remains controversial. There are two theories – vasogenic theory and cytotoxic theory. The vasogenic theory is considered to be the most likely and accepted cause of PRES. Vasogenic theory states that impaired cerebral autoregulation responsible for an increase in cerebral blood flow (CBF) and this leads to damage of the vascular endothelium causing blood-brain barrier dysfunction and cerebral vasogenic edema (Cipolla, 2007). The involvement of posterior circulation is attributed to the lack of sympathetic innervation.

But some patients with PRES donot have hypertension which supports cytotoxic theory which says endothelial dysfunction with cerebral hypoperfusion is the main pathophysiology. But irrespective of the cause of PRES, hypertension is a feature in up to 80 % of cases. Inflammatory response and multiorgan involvement in PRES is attributed to increased levels of cytokines, renal dysfunction, vasoconstriction, coagulation system alterations and endothelial dysfunction.

Studies advocate routine MRI screening of all patients with preeclampsia. Since high BP was undetected in antenatal period MRI was not done in our patient (Ekawa *et al.,* 2012). Increased cerebral pressure causes bradycardia and palpitation due to Cushing's Effect. This complaint led to early diagnosis and seizure prevention in our patient.

Conclusion

Early diagnosis and management of blood pressure can prevent seizures and neurological damage in patients with a reversible condition like PRES.

Conflict of Interest: None

REFERENCES

Bartynski WS (2008). Posterior Reversible Encephalopathy Syndrome, Part 1: Fundamental Imaging and Clinical Features. *American Journal of Neuroradiology* **29**(6) 1036–1042. https://doi.org/10.3174/ajnr.A0928

Cipolla MJ (2007). Cerebrovascular Function in Pregnancy and Eclampsia. *Hypertension* **50**(1) 14–24. https://doi.org/10.1161/HYPERTENSIONAHA.106.079442.

Ekawa Y, Shiota M, Tobiume T, Shimaoka M, Tsuritani M, Kotani Y and Hoshiai H (2012). Reversible Posterior Leukoencephalopathy Syndrome Accompanying Eclampsia: Correct Diagnosis Using Preoperative MRI. *The Tohoku Journal of Experimental Medicine* **226**(1) 55–58. https://doi.org/10.1620/tjem.226.55

Fugate JE, Claassen DO, Cloft HJ, Kallmes DF, Kozak OS & Rabinstein AA (2010). Posterior Reversible Encephalopathy Syndrome: Associated Clinical and Radiologic Findings. *Mayo Clinic Proceedings* **85(**5) 427–432. https://doi.org/10.4065/mcp.2009.0590