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CASE REPORT

Late Postpartum Eclampsia with Reversible Posterior Leucoencephalopathy Syndrome: a Case Report and review of Literature

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ABSTRACT
Postpartum eclampsia occurs in 10-45% of women with eclampsia. Posterior reversible leucoencephalopathy syndrome (PRES) may develop in a patient with preclampsia, eclampsia or late postpartum eclampsia. PRES is characterised by headache, seizures, visual loss, altered mental status in conjugation with radiological findings of posterior cerebral white matter edema/hypodensties. We present a case of 30 year previous healthy postpartum woman suffering from severe persistent headache, nausea and generalized tonic-clonic convulsions diagnosed to be a case of PRES.
Key Words : *Posterior reversible encephalopathy syndrome (PRES); Late postpartum eclampsia; Hypertension*

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INTRODUCTION

PRES was first described by Hinchey et al in 1996 (Hinchey *et al*, 1996). It typically presents as headache, seizures, altered mental status such as confusion, lethargy, cortical visual disturbances with transient edematous changes of subcortical white matter on neuroimaging (Kwon *et al*, 2001). Predisposing factors for PRES include hypertensive encephalopathy, chronic renal insufficiency, poststreptococcal glomerulonephritis, thrombitic thrombocytopenic purpurea, systemic lupus erythematous on exposure to immunosuppressants like cyclosporine, tacrolimus, interferon alpha, cisplatin, cyclophosphamide (Hinchey *et al*, 1996, Sanders *et al*, 1991). In the neuroimaging studies of PRES, computed tomography (CT) and magnetic resonance imaging (MRI) show bilateral changes in the posterior regions of cerebral hemispheres and posterior fossa structures. Early recognition of PRES is very important because immediate control of blood pressure or withdrawl of immunosppresive drugs will cause reversal of neurological symptoms and neuroimaging abnormality (Schwartz *et al* 1998). Delay in diagnosis and treatment can cause permanent damage to affected brain tissues resulting in stroke and death (Antunes *et al.* 1999) . We report a case of PRES occuring in the late postpartum and managed successfully.

CASE REPORT

A 30 year old woman gravida 2, para 2, presented to the emergency department 7 days postpartum with complaints of severe persistent headache, nausea and generalised tonic- clonic convulsions. She had normal pregnancy, uncomplicated vaginal delivery and labour period. She delivered healthy baby. She denied history of fever, blurred vision, previous head injury. On examination she was conscious, Glasgow coma scale (GCS)13/15. Vitals included BP 180/110 mmHg, Pulse 76/min regular, respiratory rate 20/min. There was no edema, neck rigidity. Kernig sign was negative. Examination of nervous system, cardiovascular, respiratory and abdominal system revealed no abnormality. Laboratory investigations including hemogram,ESR, renal, lipid, hepatic , electrolytes (Sodium, Pottasium, Calcium, Magnesium), coagulation profile, serum uric acid were within normal limits. ECG was normal. Urine examination did not show proteinuria. Cerebrospinal fluid analysis revealed slightly elevated pressure (225 mm H_2o) and mild elevation of protein (90 mg/dl). Serum serological study including VDRL and autoimmune profile (ANA, dsDNA, antiphospholipid antibody) revealed no abnormality. C3, C4 profile could not be done due to

financial constraints. Fundus examination did not reveal papilledema. CT and MRI venogram was normal. Brain MRI showed T_2 weighted axial image thus revealing hyperintense areas within the cerebellar hemispheres, grey and subcortical white matter of bilateral frontal, parietal, occipital and temporal regions, left lentiform nucleus and right caudate nucleus suggesting the diagnosis of PRES. She was started on IV magnesium sulfate (2gm per hour for 48 hours with close monitoring of magnesium level) , IV 1 gm Phenytoin, 2 mg I/V diazepam, IV labetalol and glycerol. She became stable on 4th day. Her neurological symptoms subsided completely by 8th day. Repeat MRI brain on 8th day showed complete resolution of brain lesions. She was discharged on ramipril 2.5 mg once daily with 300 mg per day of phenytoin. On follow up at 6 months, she was free of neurological symptoms.

DISCUSSION

PRES, the term first coined by Hinchey et al is a recently recognised brain disorder, mostly associated with various conditions in which blood pressure rises acutely or with the use of immunosuppressive and cytotoxic drugs[1]. It is characterised predominantly by white matter edema affecting the occipital and posterior parietal lobes of the brain (Hinchey *et al*, 1996). It is known by various names like hypertensive encephalopathy, extensive brain stem hyperintensity, pontine reversible edema, posterior reversible edema syndrome and reversible occipital-parietal ecephalopathy syndrome Pavlakis *et al,* 1999). Eclamptic convulsions can occur before, during or after delivery. Postpartum eclampsia (PPE) occurs in 10-45% of women with eclampsia (Matthys, *et al* 2004). About half of the cases of PPE occur within 48 hours delivery, while the rest occur between 2 days and 4 weeks after delivery as studied in our patient (late postpartum eclampsia - LPPE). In LPPE, the pregnancy and delivery often are completely normal without signs of a preeclamptic syndrome as in our case (Lubarsky, *et al* 1994). Blood pressure in eclamptic patients varies from 22-54% in severe hypertension, 30-60% in mild hypertension while 16% have no hypertension. Diagnosis of PRES mostly secondary to LPPE in our patient was suggested by the characteristic features of severe headache, vomiting without proteinurea mostly secondary to hypertensive encephalopathy itself. PRES is recently described under recognised clinico radiologic syndrome. The symptoms are often identical to antepartum eclampsia and include headache, blurred vision, photophobia, altered mental status,scotomas, dyspnoea, seizures. MRI brain findings include hyperintense areas on T_2 weighted and FLAIR. MRI imaging studies mainly occur in the white matter of parieto-occipital regions but also seen in the brain stem, cerebellum and basal ganglia (Katsumata *et al.* 1993). PRES is usually reversible with appropriate treatment and early recognition Striano *et al.* 2005). The management of PRES includes early recognition and withdrawl of immunosuppressive and cytotoxic drugs, antihypertensive drug management and appropriate anticonvulsant treatment with phenytoin, fosphenytoin, benzodiazepines, sorbitrates or combination (Schwartz *et al.* 1998). In patients with PRES due to hypertensive encephalopathy, the mean arterial pressure should be reduced by about 20% to diastolic blood pressure of 100 mmHg within the first hour (Vaughan and Delanty, 2000). Delayed treatment can result in irreversible neurological sequele like epilepsy, stroke and death (Antunes *et al.* 1999, Striano *et al.* 2005). The pathophysiologic hypothesis of PRES involves a breakdown of cerebral autoregulation and endothelial dysfunction due to increase in blood pressure above the patient's baseline. It is believed that the risk of autoregulation breakdown is markedly great in posterior brain because of lesser innervation and great inability to adjust to blood pressure fluctuations (Finocchi, *et al* 2005). This autoregulation failure leads to vasogenic edema (Finocchi, 2005). The presence of endothelial dysfunction decreases the threshold blood pressure at which vasogenic edema appears (Finocchi, *et al* 2005). For this reason vasogenic edema may occur with mild elevated or normal blood pressure. T_2 weighted MRI is the test choice for LPPE with PRES (Finocchi, 2005). Magnetic resonance diffusion weighted images (DWI) and apparent diffusion coefficients (ADC) can differentiate between vasogenic and cytotoxic edema (Schaefer , 1997).

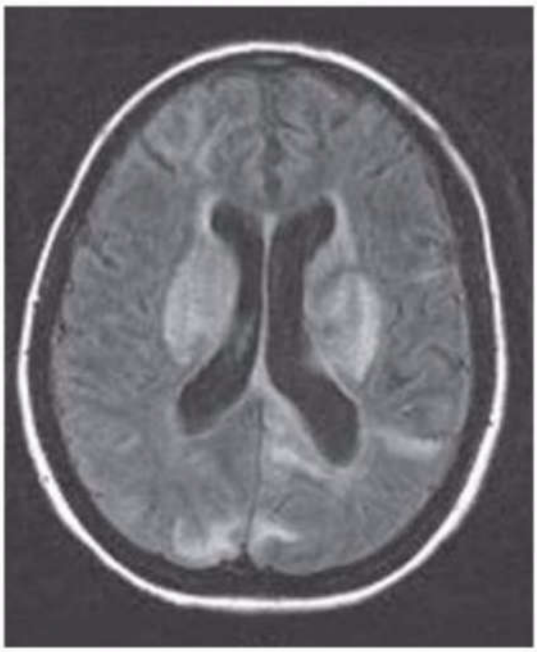

Figure 2
Brain MRI-T2/FLAIR showing T2 subcortical hyperintensity without enhancement in parieto-occipital lobes before treatment.

CONCLUSION

The rare presentation of late postpartum eclampsia as PRES should be considered in the differential diagnosis of postpartum seizure with brainstem dysfunction. Early recognition, prompt treatment and stopping the offending immunosupppresives as well as cytotoxic agents, can prevent permanent neurologic sequele and cerebral infarction and associated morbidity. Multidisciplinary care forms the corner stone to achieve a safe motherhood in these women. Before discharging any postpartum woman from the hospital, she should be made aware of the symptoms of postpartum eclampsia syndrome in the form of severe headache, nausea, vomiting, visual disturbances, generalised or focal neurological deficits.

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