Case Report

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Posterior Reversible Encephalopathy Syndrome (PRES), not Unusual with Eclampsia

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Abstract

Posterior reversible encephalopathy syndrome (PRES) is a reversible syndrome characterized by headache, seizures, altered sensorium, and loss of vision associated with white matter changes on imaging. We report here a 23 year-old lady third postpartum day, presenting with high blood pressure, seizures and loss of vision and diagnosed as posterior reversible encephalopathy syndrome. She was treated successfully with antihypertensives and showed dramatic improvement. This condition is important to recognize and needs to be treated promptly to prevent morbidity and mortality in pregnancy and postpartum.

Keywords: Posterior Reversible Encephalopathy Syndrome; Postpartum; Eclampsia; Oligohydramnios.

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Introduction

Posterior reversible encephalopathy syndrome (PRES) is a clinical as well as neuro-radiological diagnosis, first described by Hinchey et al in 1996 [1]. Predominent clinical features are headache, vomiting, visual abnormalities, mental confusion, seizures and radiological features are white matter changes, indicative of edema mainly in the posterior regions of the cerebral hemispheres, but sometimes also involving the brainstem, cerebellum, and other areas of cerebral hemisphere. PRES is associated with numerous underlying conditions like preeclampsia, hypertensive encephalopathy, thrombotic thrombocytopenic purpura, systemic lupus erythematosus, renal failure and central nervous system infections and patients on immunosuppressants. Most Importantly, PRES appears to be completely reversible if the underlying cause is treated or the precipitating drug timely withdrawn [3]. Postpartum eclampsia is an under-recognized condition occurring in 5.7% of all cases of pregnancy-induced hypertension [2]. We report here a case of PRES in association with postpartum eclampsia, emphasizing the fact that early diagnosis and treatment can prevent complications.

Case Report

A 23 year old female patient, primigravida, booked case was admitted at 38 weeks 2 days period of gestation with oligohydramnios (amniotic fluid index 5.4) for safe confinement. On admission, patient had no complaints. Her general and systemic examination was within normal limits. Her hematology and biochemistry investigations were within normal limits. She underwent emergency LSCS with delivery of male baby of 2.9 kg (indication-failed induction witholigohydramnios) and was given routine postoperative care.

After 2 hours of caesarean section, she developed high BP records of 170/96 mm Hg, for which she was given inj Labetalol 20 mg and inj Furosemide 10 mg and her BP dropped down gradually to normal. Her

hematology and biochemistry investigations were within normal limits except serum uric acid (9.1mg/dl). On 1st and 2nd post op day, patient was comfortable and her BP remained normal. On 3rd post op day, she complained of headache and blurring of vision (Rt>Lt) and her BP was found to be180/112 mm Hg. She was given inj Labetalol 20 mg (2 doses). After 20 min, she had generalized tonic clonic seizure causing tongue bite. She was managed with antiseizure therapy (inj MgSO4), antihypertensives (inj Labetalol, tab Amlodipine), antibiotics and shifted to ICU. Next day, her BP returned to normal on antihypertensives but still complained of severe occipital headache and blurring of vision (Rt>Lt). Her general examination was unremarkable. Central nervous system examination revealed diminution of vision in both eyes with regard to perception of hand movement. Pupillary reactions and fundoscopic examination were normal and plantars were flexor. Abdominal, cardiovascular, and respiratory system examinations were unremarkable. Her complete blood picture, kidney function test, liver function test, clotting parameters, and electrocardiogram were within normal limits except serum uric acid (8.9 mg/dl) and serum LDH (683 mg/dl). Urine examination revealed 2+ proteinuria. MRI brain (dated 13/09/15) showed symmetrical hyperintense signals in the white matter of the bilateral occipital lobes in T2-weighted and FLAIR sequences (Figure 1). Magnetic resonance venogram (MRV) was done to examine the deep venous system which was found to be normal. Her vision improved to 6/6 after 5 days following commencement of the treatment. She was followed up and an MRI (dated 21/09/2015) after 2 weeks showed disappearance of the hyperintensities (Figure 2). This confirmed the diagnosis of PRES. Gradually patient's symptoms recovered and patient was discharged to home with a healthy baby.

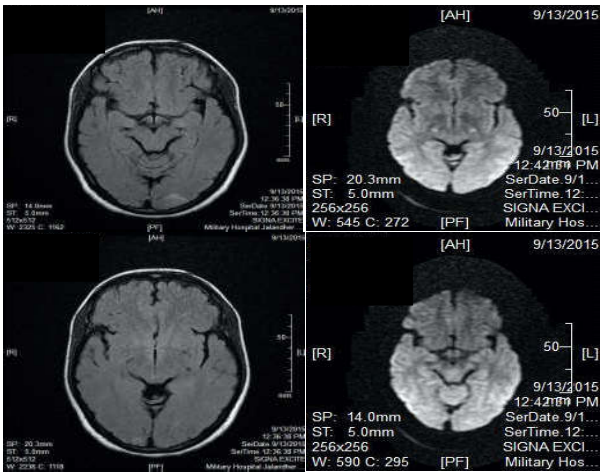

Fig. 1: MRI Brain (dated 13/09/15) Bilateral hyperintensties seen in parieto-occipital lobes on FLAIR images, no mass effect. No restriction on DW images suggestive of possibility of PRES

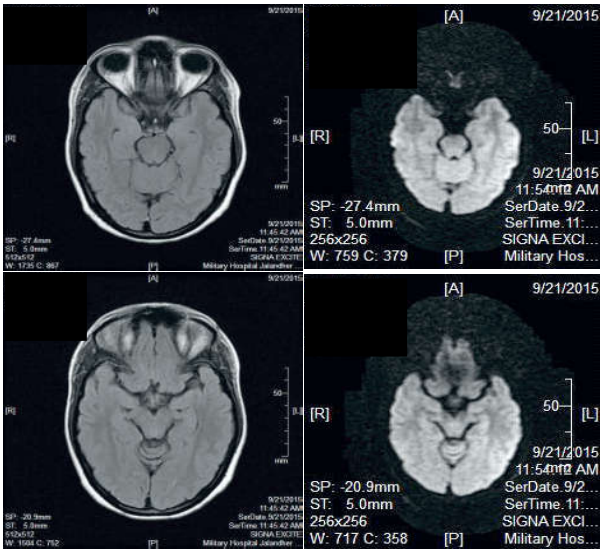

Fig. 2: MRI Brain (dated 21/09/2015) Disappearance of hyperintensities in parieto-occipital lobes with no restriction on DW as compared to old MRI dated 13/09/15, Suggestive of confirmation of PRES

Discussion

Incidence of eclampsia is 8.6 of every 10,000 live births [1]. Late postpartum eclampsia (LPE) accounts for 5% to 26% of eclampsia cases. LPE is seen mostly between 48 hours and 1 month post delivery with clinical picture different from classic eclampsia.In classic eclampsia, hypertension and proteinuria occur days before onset of seizures. However, in LPE, the pregnancy and delivery are often uneventful and without signs of any preeclamptic syndrome. LPE presents initially like hypertensive encephalopathy and mostly begins with headache of abrupt onset, with increasing severity days to weeks after delivery (50%–75% of cases). Other common symptoms are visual disturbances (19%–32% of cases), nausea/vomiting, and focal or generalized neurologic deficits. Convulsions develop within hours to days of onset. T2-weighted MRI often shows findings consistent with PRES [2]. In our case, the patient had a warning symptom of headache. Her blood pressure was just elevated on presentation without significant pedal edema. Her antenatal period was devoid of any clues for possibility of development of eclampsia, but still she presented with all the features in late postpartum stage.

Eclampsia is well known cause of PRES [3]. PRES results from loss of autoregulation of cerebral perfusion due to sudden high blood pressure, which results in dilatation of cerebral arterioles with leakage of endothelial tight junction leading to cerebral vasogenic edema. Posterior circulation is more affected due to relatively less sympathetic innervation

of vertebrobasilar vessels, which in turn causes sudden marked increase in arterial pressure.The patient becomes symptom free within days or weeks of treatment. In T2-weighted and FLAIR images of MRI brain, high signal intensity is seen mainly in the posterior regions due to subcortical white matter vasogenic edema [4]. Furthermore diffuse weighted imaging (DWI) and apparent diffusion coefficient (ADC) map images are helpful to distinguish vasogenic from more harmful cytotoxic edema, which causes irreversible ischemia. Clinical improvement precedes radiologic improvement.

Our patient developed features suggestive of eclampsia with severe headache and visual disturbances. The main differential diagnoses considered were cerebral venous sinus thrombosis, pregnancy-related stroke, and postpartum eclampsia with PRES [5]. An MRV done in my setup was normal, so, venous sinus thrombosis was ruled out. DWI and ADC imaging were done to rule out cytotoxic edema, further confirming vasogenic edema, which went in favour of PRES. Pregnancy-related stroke was an unlikely diagnosis as the patient recovered dramatically and the MRI lesion disappeared on follow-up. Therefore, a diagnosis of PRES was made. RCVS is a differential diagnosis which was considered but we thought PRES is due to failure of autoregulation and vasogenic edema. MR arteriogram could not be done to rule it out. The difficulty in diagnosis of late postpartum preeclampsia is that there are no preceding symptoms during or after delivery. Most cases of postpartum eclampsia begin abruptly with headache days to weeks following delivery [6]. They also have associated visual disturbances and can progress to eclampsia. Other complications can include aspiration pneumonia, pulmonary edema, disseminated intravascular coagulation, permanent neurological deficit, encephalopathy, and death.

Conclusion

PRES is reversible after appropriate treatment, which makes it important to recognize and treat the etiology to prevent its progress to irreversible damage [6]. This case report demonstrates that early treatment with control of blood pressure can reverse this condition and also prevent progression to eclampsia, thus emphasizing the need for early diagnosis and treatment. Thus, patients should be warned of preeclampsia symptoms, not only in the antenatal period but also in the postpartum period so that this condition can be recognized early. Prompt recognition and treatment with antihypertensive and anticonvulsant prevent severe maternal complications.

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