LATE POSTPARTUM ECLAMPSIA COMPLICATED WITH POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME: A RARE CASE REPORT

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Abstract

Posterior reversible encephalopathy syndrome (PRES) is a rare but serious clinical-neuroradiological entity characterized by headache, vomiting, visual disturbances, altered mental status, seizures, and unconsciousness associated with the characteristic imaging findings, including subcortical vasogenic oedema at the bilateral parietal and occipital lobes. We describe a case of a 26-year-old female PRES patient in the late postpartum period without antenatal pre-eclampsia or eclampsia. This patient was not initially diagnosed with pre-eclampsia and PRES. The diagnosis was established after magnetic resonance imaging (MRI). After treatment, the patient's PRES resolved. Early diagnosis and treatment are key to reversing PRES.

Keywords: Posterior reversible encephalopathy syndrome (PRES); late postpartum eclampsia; reversible posterior leukoencephalopathy syndrome (RPLS); Vasogenic oedema; Magnetic resonance imaging.

Introduction

Posterior Reversible Encephalopathy Syndrome (PRES) is a rare and serious entity, with manifestations of multiple non-specific clinical signs and symptoms, which include headache, vomiting, visual disturbances, altered mental status, seizures, and unconsciousness. The characteristic findings on CT include subcortical vasogenic oedema at the bilateral parietal and occipital lobes.[1,2] PRES occurs mostly in cases of pre-eclampsia (PET) and eclampsia.[1,3] Most of the studies have shown that PERS occur after eclampsia.[4] Preeclampsia during pregnancy is characterized by hypertension and proteinuria. When a PET seizure occurs for which no other causative factor is present, then the diagnosis of eclampsia is established.

Most of the cases of PET and eclampsia occur between 24 weeks of pregnancy and 48 hours after delivery. However, a few cases occur between 48 hours and 4 weeks postpartum. Late postpartum eclampsia accounts for only 16% of postpartum eclampsia.[5-7] Late postpartum eclampsia with PRES syndrome is a very rare occurrence, and most of the gynaecologists are unaware of this clinical condition. Usually, the clinician and the pregnant female tend to pay sufficient attention during pregnancy. Still, most of the time, the postnatal period is neglected, and often, the postpartum eclampsia occurs after discharge from the hospital. Then the patient is usually brought to the hospital in critical condition.[8,9] Most of the late postpartum eclampsia cases report to the hospital on the 6th or 7th day after delivery.[10-11]

More than 90% of late postpartum eclampsia patients have headache, restlessness, and visual symptoms before actual seizure, but they do not visit the hospital for these early symptoms. Although the risk of eclampsia is reduced, PRES is increased

Case report

The present case is of a 26-year-old female primigravida with a full-term pregnancy. She had no history of hypertension or neurological or mental illness. Blood pressure (BP) was normal throughout the antenatal period. Other investigations during the antenatal period were normal, including the urine albumin level (*Table 1*). A lower segment caesarean section (LSCS) was performed for a breech presentation with oligohydramnios. The surgery was successful. There was no other obvious discomfort, and no treatment was administered. Patient was discharged on day 4th with an abdominal incision that was healthy. On the 6th day, the

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patient had severe headache in the morning, for which she took a mild analgesic. Suddenly, the patient had a convulsion in the evening at home, and then she was brought to the hospital at 6 pm in a state of semiconsciousness. Blood pressure at the time of admission was 200/110 mm Hg, pulse 110/min, PO2- 92%, suddenly patient again had typical tonic-clonic convulsions with frothing from the mouth. The patient was immediately managed with IV diazepam followed by MgSO4 IV 4g stat with 5gm in each buttock with oxygen inhalation. Catheterization was done.

After stabilizing the patient's condition, the patient was referred to the neurological center, where MRI was done, which showed non-enhancing symmetrical abnormal signals in the bilateral frontal, parietal, occipital, and temporal close-based ganglia and pons, predominantly involving the parietal and occipital lobes, suggesting PRES as shown in (*Figure 1*).

On the second day after admission, the patient's condition improved markedly, with normal consciousness and no headache, though the patient's spirits remained poor. The blood pressure was 130/80 mmHg. MgSO4 was given for 24 hours after the last seizure.

On the third day, the patient was generally in good condition, with no obvious symptoms; neurological examination revealed no abnormal findings. Vital signs were normal. With improvement in symptoms and signs after treatment, together with MRI findings, cerebral vasospasm and PRES were considered. Further treatment aimed to relieve cerebral vasospasm while continuing anti-inflammatory therapy, human serum albumin supplementation, and other supportive treatments, including antihypertensives. The patient was hospitalized for a total of five days, two days in the ICU and three days in the ward. The final diagnosis of this patient was late postpartum eclampsia complicated with PRES.

Table 1. Laboratory investigations.

Blood urea	25.76 mg/dl
Serum creatinine	0.7 mg/dL
Blood glucose	104mg/dl
Sodium	138mg/dl
Potassium	2.7mg/dl
Chloride	1.4mg/dl
Urine protein	+

Discussion

In 1996, Hinchey et al. reported 15 cases of reversible posterior leukoencephalopathy syndrome (RPLS).[11] Since then,

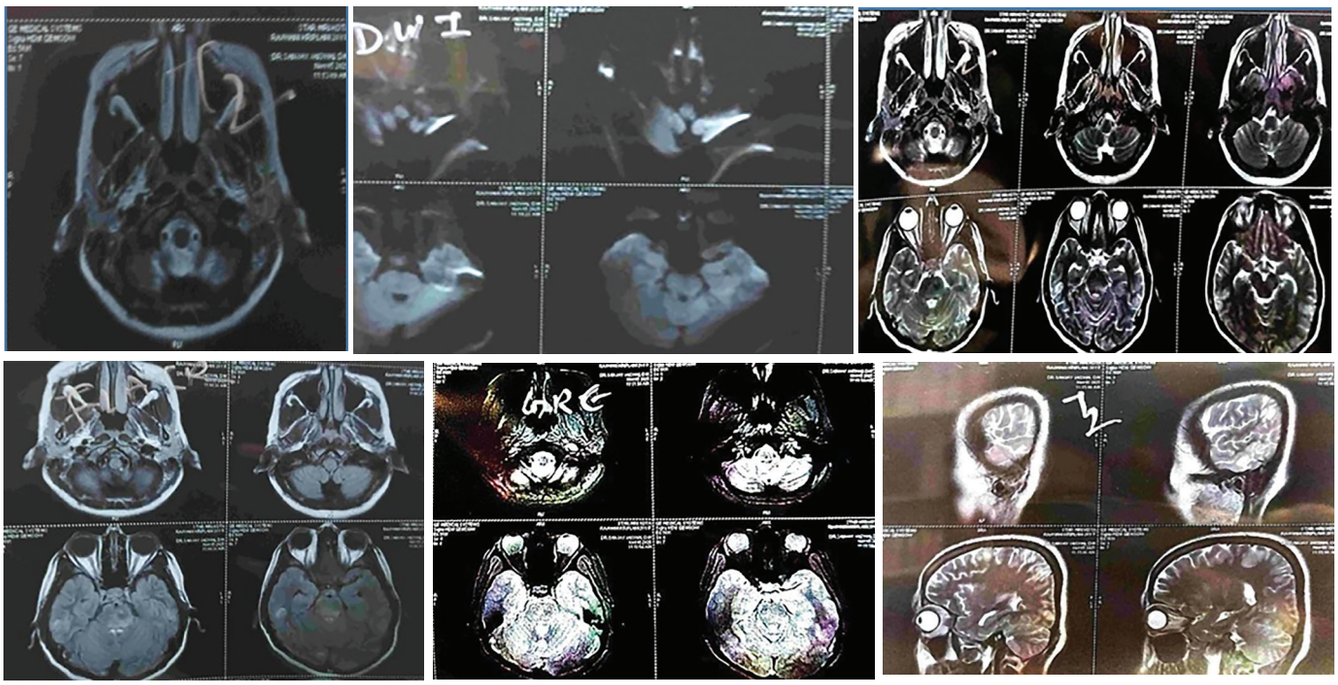

Figure 1. Brain MRI showing symmetrical bilateral parieto-occipital predominant hyperintense lesions, consistent with posterior reversible encephalopathy syndrome (PRES).

RPLS has gradually gained recognition among clinicians. Using MRI FLAIR, Casey et al. found that in 94% of RPLS patients, the cerebral cortex was also involved, and cortical lesions accounted for 46% of all lesions.[12] Therefore, in the year 2000, a new name was proposed, and PRES was used to replace RPLS, and this has been widely recognized.[11]

The aetiology of PRES is not clear. A variety of conditions and predisposing factors are associated with PRES, including hypertension, pre-eclampsia/eclampsia, immunosuppressive agents used in organ transplantation (especially cyclosporine and tacrolimus), cancer chemotherapy, autoimmune diseases (e.g., systemic lupus erythematosus, Wegener's granulomatosis), and infection or sepsis.[1] Among these factors, pre-eclampsia and eclampsia are most commonly reported in the literature.[13] Pre-eclampsia has an incidence of between about 3–8.7%, and the incidence of eclampsia is less than 10/10,000 births in most countries.[5] In addition to the typical pre-eclampsia/eclampsia, individual atypical cases may occur before 20 weeks of pregnancy or 48 hours after giving birth. There are a few atypical cases without hypertension or proteinuria.[6] PRES needs to be differentiated from many diseases, mainly cerebral venous sinus thrombosis (CVST) and ischaemic stroke, but also including acute or sub-acute cerebrovascular diseases, central nervous system infections,autoimmune diseases, and mitochondrial diseases.[14-16] CVST is the most common puerperal cerebrovascular disease and sometimes presents with clinical manifestations similar to PRES.[17-19] The differential diagnosis of PRES and CVST is through MRI and MRA/MRV.[14,19] The differentiation of PRES and ischaemic stroke is of vital importance for the treatment and prognosis.[17] In ischemic stroke, mild to moderate hypertension is not treated. While for PRES, aggressive treatment of hypertension should be taken to prevent the development of irreversible brain damage.[14,20] In addition, early diagnosis may also provide for the opportunity of thrombolytic therapy for ischaemic stroke.[21,22]

With correct, timely diagnosis and effective early treatment, the patient's prognosis is usually good. Neurological symptoms, signs, and radiographic changes usually disappear completely in 1–2 weeks, while delayed diagnosis and/or incorrect treatment likely cause irreversible neurological damage or even death.[11,13,15] Treatment includes management of the primary disease, control of neurological symptoms, and control of hypertension.

Our patient was a rare case of late postpartum eclampsia complicated with PRES. In the morning of the seizure event, the patient had blurred vision, headache, and these symptoms gradually worsened. The patient had no history of hypertension in the antenatal and immediate postpartum period, while there was late postpartum high blood pressure as well as neurological and psychiatric symptoms.

The diagnosis of PRES was established on MRI brain and the obstetricians' treatment for eclampsia was timely and effective. Neurologists' measures to reduce cerebral oedema and cerebral vasospasm, along with other "infarction" treatment measures, were beneficial for PRES; therefore, the patient recovered quickly.

Conclusion

Health care workers and mothers should pay attention to physical changes in the postpartum period, with close monitoring of blood pressure and other neuropsychiatric symptoms. Obstetricians, neurologists, ophthalmologists and radiologists should be familiar with the clinical and imaging features of PRES. Radiological changes provide key characteristics for early diagnosis of PRES. When suspected, a brain MRI should be performed, with particular attention to Diffusion-Weighted Imaging (DWI) and Apparent Diffusion Coefficient (ADC) maps. The key to full recovery of PRES is early diagnosis and treatment. Misdiagnosis and delayed treatment may cause permanent neurological damage.

Declaration section

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Editor's comments

This case involves a 26-year-old patient presenting with late postpartum eclampsia complicated by Posterior Reversible Encephalopathy Syndrome. PRES is a rare but serious clinical-neuroradiological entity characterized by headache, vomiting, visual disturbances, altered mental status, seizures, and unconsciousness associated with the characteristic imaging findings that include subcortical vasogenic oedema at the bilateral parietal and occipital lobes. With a correct, timely diagnosis and effective early treatment, the patient's prognosis is usually favorable. While neurological symptoms, signs, and radiographic changes usually disappear completely in 1–2 weeks, delayed diagnosis and/or incorrect treatment are likely to cause irreversible neurological damage or even death. Treatment includes management of the primary disease and control of neurological symptoms and hypertension.

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