Case report

Posterior reversible encephalopathy syndrome (PRES) in a postpartum woman with late-onset preeclampsia

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Abstract

Posterior reversible encephalopathy syndrome (PRES) is a rare neuroradiological condition characterized by vasogenic oedema of subcortical white matter, primarily affecting the posterior cerebral hemispheres. We present a remarkable case of PRES in a 28-year-old primigravida with late-onset preeclampsia, following an elective caesarean section for major degree placenta previa. Despite immediate management, the patient experienced persistent symptoms, prompting neuroimaging that confirmed the diagnosis. Prompt action led to a favourable outcome in this patient, who remained asymptomatic with no neurological impairment during the six-month follow-up. Notably, postpartum preeclampsia itself is infrequent, with researchers estimating its occurrence in approximately 4% to 6% of women diagnosed with preeclampsia and eclampsia during the postpartum period. Within this subset, PRES is an exceedingly uncommon complication, further underscoring its rarity. Prompt recognition and management are crucial, given the uniqueness of this presentation, to ensure a favourable outcome.

Keywords: posterior reversible encephalopathy syndrome, PRES, preeclampsia, pregnancy induced hypertension, PIH

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a rare neuroradiological syndrome characterized by vasogenic oedema of subcortical white matter, commonly involving the posterior cerebral hemispheres. It typically presents with acute visual impairment, severe headaches, seizures, vomiting, altered mental status, and focal neurological deficits. Although PRES is known to be "reversible," it can occasionally progress to cytotoxic oedema, which may result in irreversible complications. Prompt diagnosis, immediate elimination of causative factors, and appropriate antihypertensive and anticonvulsant therapy are essential to prevent serious sequelae.

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Case presentation

A 28-year-old primigravida with no previous history of hypertension or risk factors for PRES underwent an elective caesarean section at 37 weeks due to major degree placenta previa. The procedure had no intra-operative complications, and she was discharged on postpartum Day 2 after an uneventful recovery.

On postpartum Day 8, the patient presented with a gradually worsening headache for 2 days, acute bilateral visual loss associated with nausea and vomiting since the morning of presentation. There were no symptoms of shortness of breath, epigastric pain, photophobia, phonophobia, limb weakness, or involuntary movements. On admission, her Glasgow Coma Scale (GCS) was 15/15, and her blood pressure was elevated at 190/100mmHg. Neurological examination revealed mild exaggeration of deep tendon reflexes with downward plantar reflexes, normal bilateral pupillary reflexes but only with light perception, and no papilledema. Extraocular movements were intact, and no neck stiffness or periorbital or lower limb oedema was observed.

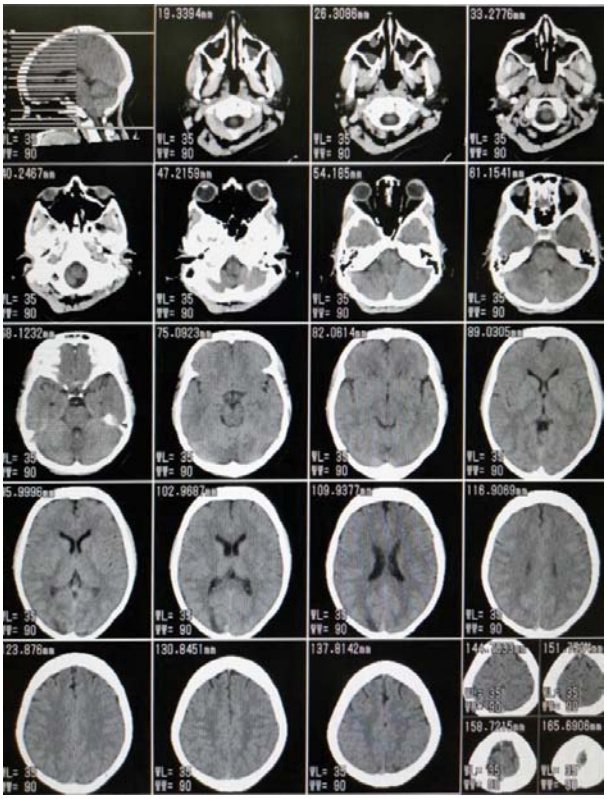
Investigations

The urine dipstick test revealed 3+ albumin, and urine analysis showed positive ketone bodies. All other basic investigations, including full blood count, liver function tests, renal function tests, coagulation profile, electrocardiogram, and prothrombin time/international normalized ratio (PT/INR), were normal.

Diagnosis and management

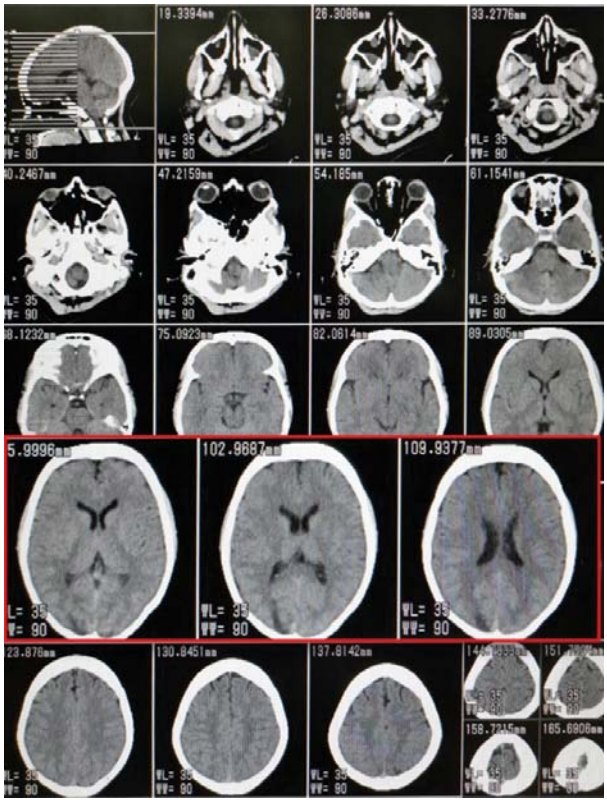
Immediate administration of magnesium sulphate (MgSO4) and a loading dose of 4g in 20 minutes, followed by a maintenance dose (intravenous 1g per hour), along with a stat dose of labetalol 200 mg, was given due to initial suspicion of severe preeclampsia. Blood pressure was gradually controlled, but headache and bilateral visual impairment persisted despite treatment.

A non-contrast CT scan of the brain was performed, revealing hypoattenuation in the subcortical white matter of bilateral occipital arcs with slight midline shift, suggestive of vasogenic oedema consistent with PRES syndrome.



1 (a)

Figure 1a. **Non-contrast CT of brain on Day 1.**



1 (b)

Figure 1b. **Enlarged images with hypodense lesion in left occipital region (Red Box).**

In view of the clinical and radiological features indicative of PRES, the patient was kept under observation and prescribed amlodipine 2.5 mg twice daily and subcutaneous Enoxaparin 40 mg daily. Her blood pressure gradually normalized, and her headache and vomiting improved simultaneously. Within 12 hours, her normal visual acuity was restored without any neurological impairment. A repeat NCCT brain scan on the following day showed improvement of the left occipital lobe hypoattenuation.

Further evaluation

On Day 6, a cerebral MRI was performed, which revealed high signal intensity in the right occipital lobe, predominantly in the white matter, associated with small haemorrhagic foci. Mild changes were noted in the left occipital lobe. No abnormalities were observed on diffusion weighted images (DWI) or MRA. These findings confirmed the diagnosis of PRES.

Follow-up and recovery

Repeat investigations were normal, and urine albumin remained positive for 2 consecutive days, then became trace. The patient remained clinically stable with no neurological impairment and was discharged after 10 days of admission with amlodipine 2.5 mg twice daily. Blood pressure was monitored twice weekly for another one-month duration and was found to be consistently normal. A clinical evaluation after one month showed the patient was symptom-free and had no impairment. After six months, we re-assessed the patient and found her to be clinically asymptomatic with no neurological impairment.

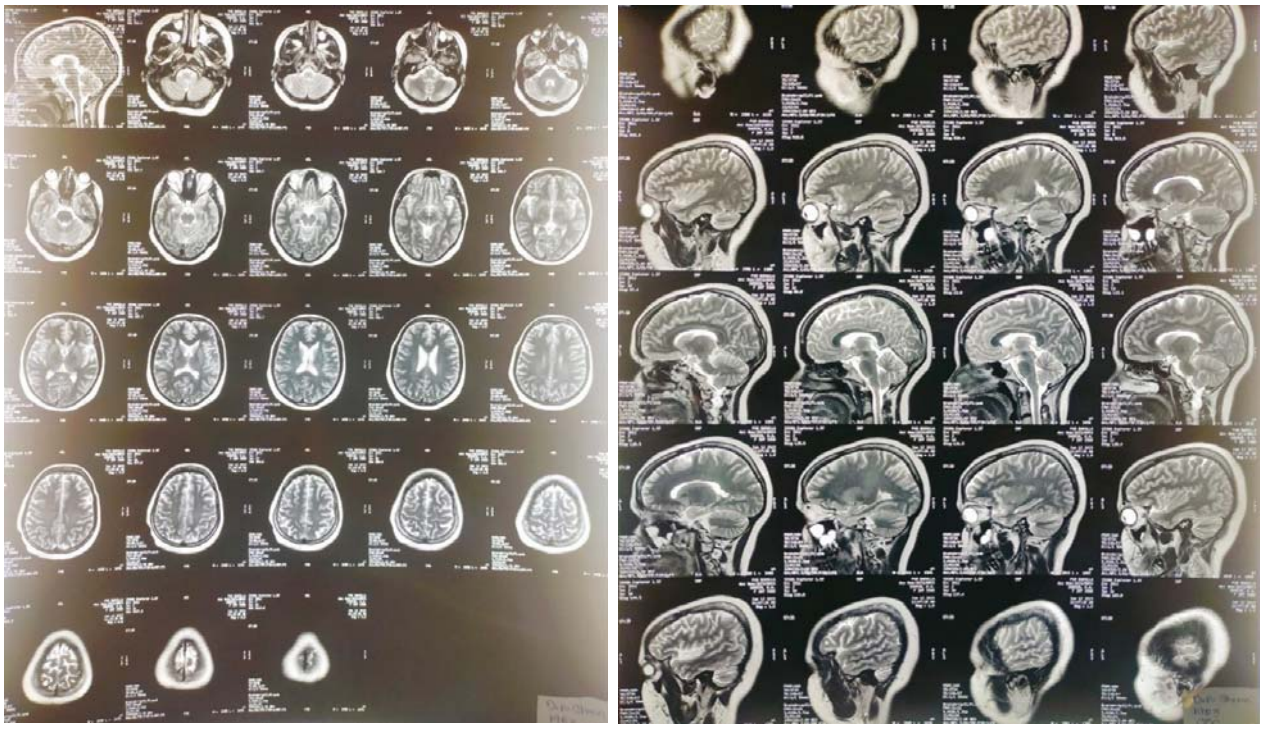


Figure 2. **T2-weighted MRI with foci of hyperintensity in occipital region.**

Discussion

PRES is a rare condition characterized by reversible vasogenic oedema of subcortical white matter, often affecting the posterior cerebral hemispheres. It can be challenging to differentiate from other conditions, such as cerebral venous sinus thrombosis (CVST) or severe preeclampsia, especially in pregnant or postpartum women.

Pathogenesis of PRES is not fully understood, and two proposed hypotheses are contradictory. One suggests severe hypertension leading to hyperperfusion and vasogenic oedema, while the other suggests endothelial dysfunction causing vasoconstriction, hypoperfusion, and ischemic oedema. Recent evidence indicates that vascular endothelial growth factor (VEGF) may play a crucial role in the pathogenesis of PRES, particularly during pregnancy.

In this case, we believe that late-onset preeclampsia, possibly due to endothelial dysfunction triggered by the effects of pregnancy, was the underlying cause of PRES in this postpartum woman.

Conclusion

PRES can manifest in the postpartum period with a late-onset preeclamptic background, even without prior evidence of pregnancy-induced hypertension during the antenatal period. It is essential for healthcare providers to be aware of PRES and take prompt action to reduce mortality and morbidity in affected patients.

Limitations

Angiographic studies were not conducted to rule out vascular pathology, and follow-up MRI was not performed due to the lack of facility.

Acknowledgments

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The successful outcome of this case study would not have been possible without the dedication and expertise of everyone mentioned above, especially the invaluable contribution of my Sir Dr Keerthi Bandara, MD, the Consultant Obstetrician and Gynaecologist. Their contributions are sincerely appreciated.

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