

Storm After the Calm: A Case of Atypical Posterior Reversible Encephalopathy Syndrome (PRES) in a Postpartum Patient

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Abstract: ***Background:** Posterior Reversible Encephalopathy Syndrome (PRES) is a rare, potentially reversible neurological disorder characterised by headache, seizures, visual disturbances, and altered mental status. It is often associated with hypertension, eclampsia, renal failure, or immunosuppressive therapy. While PRES predominantly affects the occipital and parietal lobes, atypical cases involving the frontal lobes and basal ganglia have been reported. **Case Presentation:** We report a case of a 28-year-old postpartum woman who presented with multiple episodes of seizures, headache, and neck pain following lower segment caesarean section for twin delivery. After excluding pre-eclampsia and eclampsia through clinical and laboratory evaluation, further assessment including brain MRI confirmed the diagnosis of atypical posterior reversible encephalopathy syndrome (PRES), with vasogenic oedema involving the frontal, parietal, and occipital lobes along with the basal ganglia. The patient was managed conservatively with appropriate supportive care, showed rapid clinical improvement, and was discharged on day 9 with complete neurological recovery. **Conclusion:** In postpartum patients, even in the absence of eclampsia, this case emphasises the importance of early recognition and management of PRES. Atypical MRI findings involving the frontal, parietal, occipital lobes and basal ganglia should be considered in the diagnosis of PRES. Early intervention leads to favourable outcomes and complete recovery.*

Keywords: Posterior Reversible Encephalopathy Syndrome, PRES, postpartum

1. Introduction

Posterior Reversible Encephalopathy Syndrome (PRES) is a neurological condition characterised by vasogenic oedema that predominantly affects the parieto-occipital lobes¹. It is commonly associated with conditions such as immunosuppressive therapy, pre-eclampsia/eclampsia, renal failure, and hypertensive crises². Although the exact pathophysiology remains unclear, endothelial dysfunction and impaired cerebral autoregulation are believed to contribute to blood-brain barrier disruption and vasogenic oedema³. While PRES is often reversible with appropriate management, delayed diagnosis or persistent hypertension can result in infarction, haemorrhage, or irreversible neurological deficits⁴.

PRES is frequently observed in pregnant and postpartum women, particularly in the context of pre-eclampsia and eclampsia, but it can also occur in patients without overt hypertension⁵. Haemodynamic fluctuations, endothelial activation, and immunological changes during the postpartum period may contribute to PRES even without overt hypertensive emergency³. Neuroimaging, particularly MRI with FLAIR sequences, is crucial for diagnosis, typically revealing bilateral symmetrical vasogenic oedema in posterior cerebral regions². Most patients achieve full recovery with treatment focused on seizure control, blood pressure management, and addressing the underlying cause.

This case report describes a postpartum patient who developed PRES without overt hypertension. It highlights the importance of recognising atypical presentations, especially in postpartum women, to facilitate early diagnosis and prevent complications.

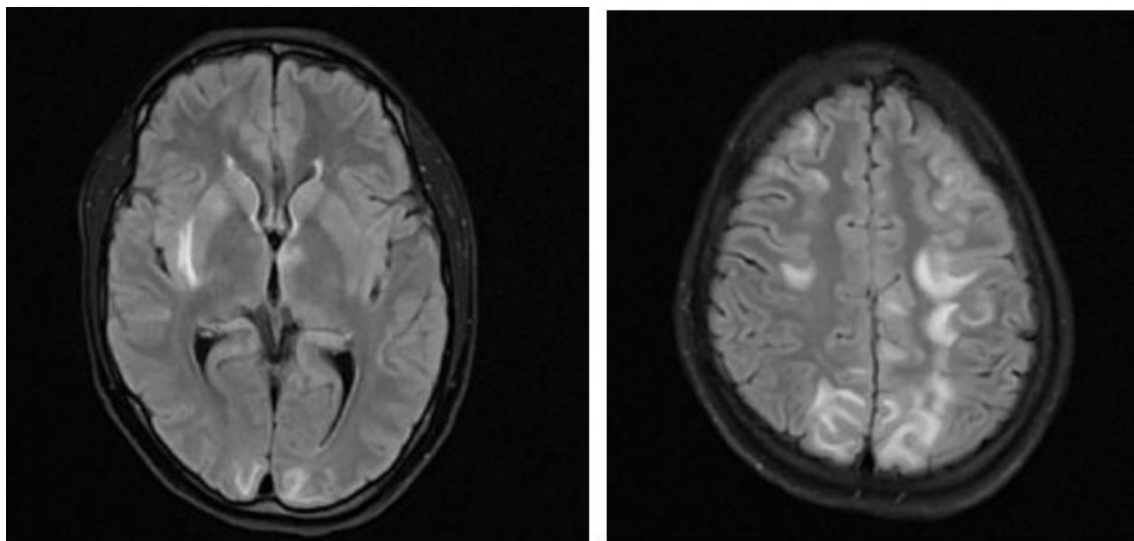
2. Case Details

A 28-year-old woman presented to the emergency department five days after delivering twins via lower segment caesarean section, complaining of four episodes of generalised tonic-clonic seizures over the previous two days. She had also experienced persistent headache and neck pain for three days. One seizure episode was terminated in the emergency department with 4 mg intravenous lorazepam. Magnesium sulphate was considered initially until further evaluation made overt eclampsia less likely. She had no history of fever, visual disturbances, prior seizures, limb weakness, or loss of consciousness between episodes. Her obstetric history was notable for a twin pregnancy delivered five days earlier via caesarean section. There was no history of hypertension, pre-eclampsia, or eclampsia, although the pregnancy had been complicated by severe anaemia in the sixth month, requiring transfusion of three units of packed red blood cells.

On initial assessment, her Glasgow Coma Scale score was E4V5M6. Vital signs included a random blood sugar of 83 mg/dL, temperature 98.9°F, pulse rate 76 beats per minute, respiratory rate 18 breaths per minute, and blood pressure 130/90 mmHg. Cardiovascular and respiratory examinations were unremarkable. Neurological examination showed bilateral extensor plantar reflexes (positive Babinski sign), reactive pupils (3 mm bilaterally), reduced motor power in the right upper limb (4/5), and normal power in the other limbs (5/5). There were no sensory deficits, cranial nerve abnormalities, or signs of meningeal irritation.

A non-contrast CT scan of the brain was performed promptly due to new-onset seizures in the postpartum period and was normal. However, owing to persistent neurological symptoms and seizures, an MRI of the brain was undertaken. It revealed features of atypical PRES with vasogenic oedema affecting

the frontal lobes, parietal lobes, basal ganglia, and occipital lobes.



Axial T2/FLAIR MRI images showing hyperintense lesions involving bilateral frontal and parietal lobes.

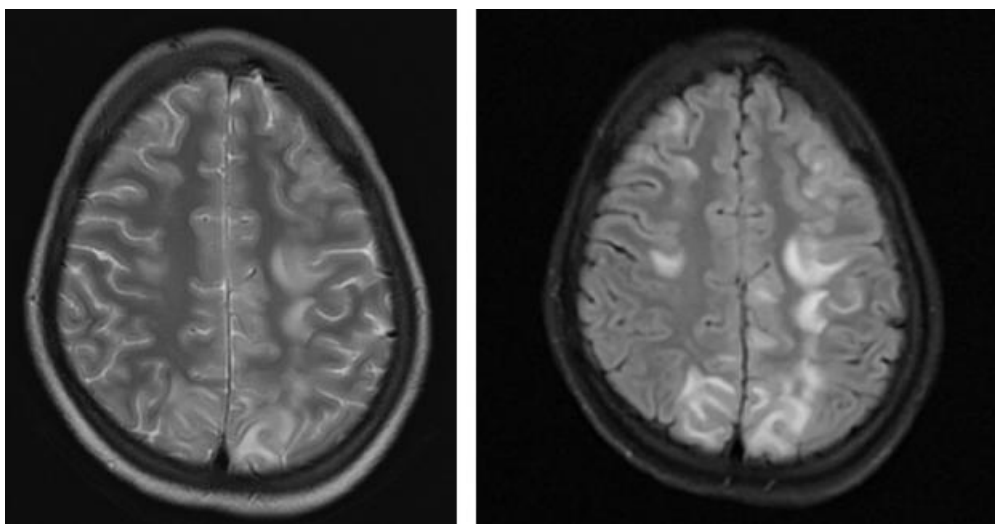
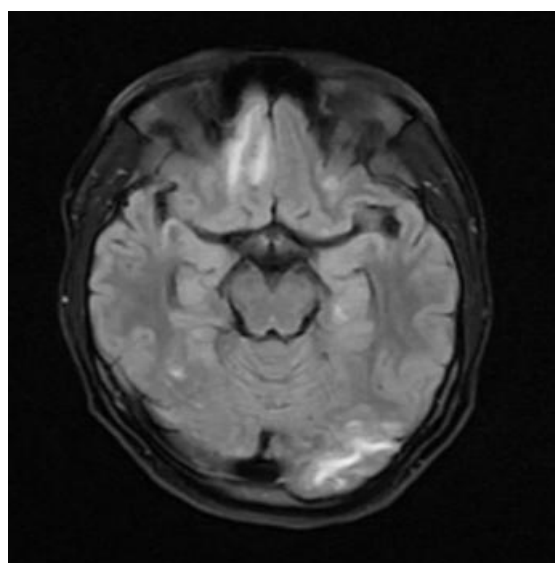


Figure 1

Figure 2

Axial T2/FLAIR MRI images demonstrating hyperintense lesions in the occipital lobes and basal ganglia, consistent with atypical PRES.



Axial T2/FLAIR hyperintense lesions in frontal and occipital regions

MR venography and MR angiography were normal, excluding ischaemic stroke and cerebral venous sinus thrombosis. Laboratory investigations showed haemoglobin 11.3 g/dL, platelet count 210,000/mm³, and total leukocyte count 12,490/mm³. Coagulation profile, thyroid function, liver function, and renal function tests were normal. Urine protein was assessed via dipstick, which was negative; no further quantitative assessment was performed. Absence of proteinuria and lack of severe hypertension made pre-eclampsia/eclampsia less likely clinically.

Differential diagnoses considered included cerebral venous sinus thrombosis, postpartum eclampsia, encephalitis, and acute ischaemic stroke. Normal MR venography and angiography, absence of fever or infectious features, and characteristic MRI findings supported the diagnosis of atypical PRES.

Following diagnosis of atypical PRES, the patient received conservative management. Blood pressure was closely

monitored, and conservative antihypertensive management was provided, and levetiracetam was continued for seizure prophylaxis. Supportive care included intravenous fluids and oxygen as required, with vigilant neurological monitoring.

The patient's condition improved steadily over the following days. Seizures ceased, and motor deficits resolved completely. She achieved full neurological recovery and was discharged on day 9. Follow-up MRI to confirm resolution of the oedema could not be performed owing to financial constraints. Ongoing neurology review was advised, with recommendations for home blood pressure monitoring and gradual tapering of antiepileptics based on clinical progress.

At outpatient follow-up after two weeks, the patient remained seizure-free with no residual neurological deficits.

This case underscores the need for early recognition of atypical PRES, particularly in postpartum patients without classic risk factors for eclampsia or pre-eclampsia. Prompt neuroimaging and timely intervention are essential for good prognosis and complete neurological recovery.

3. Discussion

Posterior Reversible Encephalopathy Syndrome (PRES) results from failure of cerebral autoregulation, leading to transient vasogenic oedema¹. Although commonly linked to pre-eclampsia, severe hypertension, and renal failure, it can occur in patients with only mild blood pressure elevation or fluctuations, as in this case³. The underlying mechanism is thought to involve endothelial dysfunction, impaired autoregulation, and blood-brain barrier disruption, resulting in oedema predominantly in parieto-occipital white matter². However, atypical radiological patterns can involve the cerebellum, brainstem, and basal ganglia⁴.

Postpartum patients may be vulnerable to PRES due to vascular and immunological changes following delivery, even without severe hypertension. In our patient, mildly elevated blood pressure (130/90 mmHg) and possible fluctuations may have contributed to cerebral endothelial dysfunction and vasogenic oedema. Prior studies suggest that blood pressure variability, rather than absolute severe hypertension, can precipitate PRES⁶.

Diagnosis relies on neuroimaging, with MRI superior to CT for detecting early changes. Classic findings are bilateral symmetrical T2/FLAIR hyperintensities in posterior regions; however, in atypical cases, involvement extends to deep white matter, brainstem, or basal ganglia⁴. Here, MRI confirmed atypical PRES with vasogenic oedema in the frontal lobes, parietal lobes, basal ganglia, and occipital lobes. A normal CT does not exclude the diagnosis, making MRI essential in suspected cases².

Management involves seizure prevention, blood pressure stabilisation, and addressing precipitating factors². In our patient, lorazepam aborted acute seizures, followed by levetiracetam maintenance. Aggressive blood pressure reduction was not required given the absence of severe hypertension. Most patients recover fully within days to weeks, and long-term anticonvulsants are usually

unnecessary once imaging abnormalities resolve³. This case highlights the importance of considering PRES in postpartum seizures, even without overt hypertension. It also stresses the value of MRI in detecting atypical variants, particularly with deep grey matter involvement and atypical cortical distribution⁴. Early intervention prevents complications such as ischaemic infarction, haemorrhage, or permanent neurological deficits².

4. Conclusion

Posterior Reversible Encephalopathy Syndrome (PRES) is an important yet under-recognised neurological condition, particularly in postpartum women without overt hypertension⁵. As illustrated by this case with atypical frontal, parietal, occipital lobes and basal ganglia involvement, a high index of suspicion is required even without traditional risk factors such as pre-eclampsia or eclampsia⁴.

Complete recovery depends on early MRI diagnosis and prompt management, including blood pressure control, seizure prophylaxis, and supportive care². Given its reversible nature, timely recognition of PRES is critical, as delayed treatment may lead to permanent neurological sequelae³. This case further supports the need for multidisciplinary care to achieve optimal outcomes in patients presenting with postpartum seizures.

References

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