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Case Report

# Posterior reversible encephalopathy syndrome in postpartum patients with gestational hypertension: A case report emphasizing early recognition and management ☆

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ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is a rare neuroradiological condition causing headaches, altered mental status, seizures, visual disturbances, and focal deficits. It is often associated with preeclampsia and eclampsia in pregnancy, but can also occur in patients with other medical conditions, such as hypertension, autoimmune diseases, renal dysfunction etc. This case report highlights the importance of recognizing PRES in postpartum patients with hypertension and the need for prompt diagnosis and management to prevent potential complications. A 30-year-old woman with gestational hypertension underwent scheduled induction of labor. After a successful delivery, she experienced a sudden headache but no other neurological symptoms. Imaging showed bilateral frontoparietal white matter edema, consistent with PRES. She was closely monitored, treated with analgesics, and improved within a week. The case highlights the rarity of PRES in postpartum patients without preeclampsia or eclampsia. It underscores the importance of considering PRES as a possible diagnosis in postpartum patients with hypertension, even in the absence of typical risk factors. Prompt control of blood pressure and careful monitoring are essential to ensure a positive outcome, as PRES can lead to life-threatening complications if not managed appropriately. The study highlights the importance of heightened awareness of PRES in postpartum patients with gestational hypertension. Early detection and timely manage-

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ment can improve prognosis, even in atypical cases. Healthcare professionals should be vigilant in assessing hypertension patients to diagnose and manage PRES, preventing neurological sequelae. Further research is needed to better understand PRES pathophysiology and risk factors in postpartum patients.



## Introduction and importance

Posterior reversible encephalopathy syndrome (PRES) is an acute neuroradiological entity in which the patients present with non-specific signs and symptoms such as headache, altered mental status, seizures, visual disturbances in the form of cortical blindness, and focal neurological deficits [1,2]. It is also known as reversible posterior cerebral edema syndrome (RPCS), hyper-perfusion encephalopathy, or brain capillary leak syndrome [3].

It is associated with other medical conditions such as hypertensive encephalopathy, preeclampsia, eclampsia, acute or chronic renal diseases, hemolytic uremic syndrome, use of cytotoxic and immunosuppressant drugs, blood transfusion, and electrolyte disturbances. However, the most common causes of PRES are preeclampsia and eclampsia [4]. The usual radiological finding of PRES is parietooccipital white matter edema [5,6]. PRES is usually reversible with a good prognosis. However, it may sometimes leave permanent neurological sequelae and can even lead to death [4]. Here we present a rare case of PRES diagnosed in a postpartum female without any evidence of preeclampsia and eclampsia. This case report has been reported in accordance with the CARE criteria [7].

### *Case details*

A 30-year-old lady was admitted to our center for a scheduled induction of labor and vaginal delivery for gestational hypertension. She was a $G_3P_2L_2$ at 38 weeks of gestation. The patient had attended all her antenatal visits at our center and had completed all her antenatal care recommendations. Her obstetrician had noted her blood pressure to be 135/90 mm Hg at 24 weeks of gestation without proteinuria which prompted a closer follow-up. She was advised for blood pressure monitoring at home, limiting her physical activities and she was explained about the alarming signs that should prompt any unscheduled visits to the hospital. Throughout her pregnancy, she consistently maintained a blood pressure range of 125-135 mm Hg systolic and 90-110 mm Hg diastolic. Additionally, her 24-hour urine protein and urine dipsticks for protein never showed any trace of proteinuria, and no antihypertensive medications were initiated. The fetal ultrasound reports showed no abnormalities, and the entire antenatal period was without any complications. Consequently, due to her existing pregnancy-induced hypertension, it was recommended that she undergo induction of labor at 38 weeks of gestation to ensure the best possible outcome for both the mother and the baby. The patient agreed to follow this advice. The patient had no known existing comorbidities, no regular medications besides her supplements, no smoking or alcohol consumption, and no use of any other substance of abuse. Her 2 previous pregnancies had resulted in spontaneous vaginal deliveries at term without any antepartum or postpartum complications. The current pregnancy was planned, and both she and her husband had Rh-positive blood groups with no family history of medical, surgical, or genetic conditions. The review of her systems at admission for the induction of labor was unremarkable. Hence the patient was admitted and subsequently, the procedure along with the risks and possible outcomes were discussed. After receiving the necessary blood investigations and fetal evaluation, induction was scheduled for the following day. Induction with Misoprostol was done which resulted in the ripening of the cervix and the onset of mild contractions which were augmented by Oxytocin. She had a successful vaginal delivery and gave birth to a healthy girl.

The day after delivery, the patient reported experiencing a sudden, throbbing, and persistent headache, which she rated as 7/10 on a pain scale. She also felt nauseated but did not experience any vomiting. However, she did not have any blurring of vision, abnormal body movements, confusion, drowsiness, loss of consciousness, neck rigidity, deficits in memory and concentration, or any motor symptoms. She did not receive any epidural analgesia during labor and delivery. Additionally, she did not complain of abdominal pain, excessive vaginal bleeding, or abdominal distension. During the examination, the patient displayed mild distress but remained oriented to time, place, and person. Her postpartum temperature was normal, and her blood pressure ranged from 125 to 130 mm Hg systolic and 90-100 mm Hg diastolic. Her pulse was 105 beats per minute, her respiratory rate was 18 per minute, and she maintained oxygen saturation at 95% on room air. Her urine output was adequate, and there were no signs of edema. The systemic examination did not reveal any abnormalities. A complete blood count (CBC), liver function test (LFT), renal function test (RFT), Urine analysis, serum electrolytes, Ultrasonography of the abdomen, and noncontrast computerized tomography (NCCT) scan of the head were ordered. Blood tests, ultrasonography of the abdomen, and evaluation of urine suggested no abnormalities. The NCCT revealed diffuse white matter hypodensity in bilateral frontoparietal white matter and posterior limb of the right internal capsule without significant mass effect (Fig. 1). These findings were followed by a MRI head which also showed high signal intensity in the bilateral frontoparietal white matter (right>left) without obvious mass effect (Figs. 2A and B). The findings were suggestive of PRES which was unusual since PRES usually presents along with preeclampsia or eclampsia but the patient did not have either of these conditions. To assess the other possible causes a thyroid function test (TFT) was or-

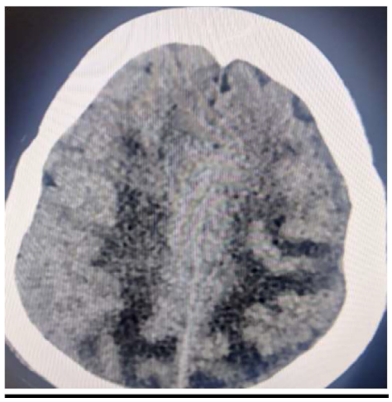

**Fig. 1 – Noncontrast axial CT image at the fronto-parietal lobe region shows hypodensity involving the white matter with finger like extension without obvious mass effect.**

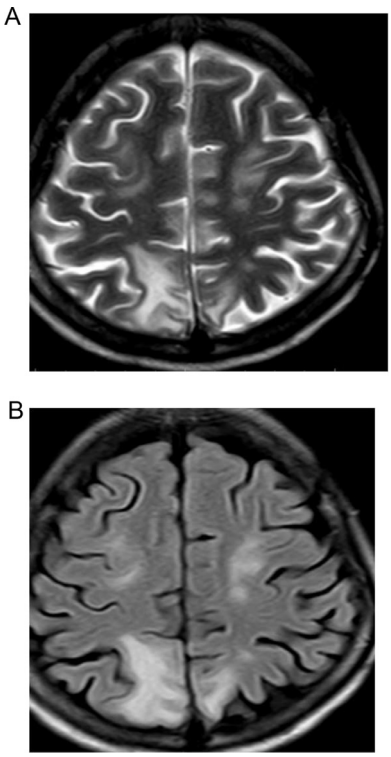


**Fig. 2 – (A) Axial T2 weighted MRI image showing T2 high signal intensity in bilateral frontoparietal white matter. (B) Axial FLAIR weighted MRI image showing high signal intensity in the bilateral frontoparietal white matter (right>left) without obvious mass effect.**

dered with antinuclear antibody (ANA) which did not reveal any abnormal findings. Hence a diagnosis of PRES in a postpartum lady with pregnancy-induced hypertension was made. The patient was explained about the diagnosis and its potential reversibility. As no other predisposing factor for PRES was identified, she was treated with analgesics for the headache. She was kept under continuous blood pressure monitoring and a close watch for seizures. She was advised to continue breastfeeding.

After spending 1 week receiving routine postpartum care, analgesics, and close monitoring, the patient showed improvement in her symptoms. Her blood pressure averaged 120/90 mm Hg, and she had no issues with breastfeeding. Upon discharge, the patient was briefed about any possible alarming signs and was advised to monitor her blood pressure regularly. A follow-up appointment was scheduled for a week later. During the follow-up, a repeat CT scan was performed, which showed normal findings. The patient reported feeling much better and expressed joy in welcoming her new child into the family.

## Clinical discussion

PRES is a rare and serious entity of the central nervous system, characterized by headaches, seizures, altered mental status, and visual impairment. Seizures, which are usually generalized and tonic-clonic, are often the presenting manifestation [8]. Our case was unique in the sense that, it occurred without seizures in a woman in the postpartum period that was diagnosed with gestational hypertension but with no evidence of preeclampsia or eclampsia.

PRES was first described by Hinchey in 1996 as a reversible syndrome due to edema mainly in the posterior regions of the cerebral hemispheres mostly the occipitoparietal region, but in some atypical cases, also involving the brainstem, cerebellum, and other cerebral areas [9].

There are various medical causes or clinical entities associated with the causation of PRES which include hypertensive encephalopathy, pre-eclampsia, eclampsia, acute or chronic renal diseases, hemolytic uremic syndrome, use of cytotoxic and immunosuppressant drugs, blood transfusion, and electrolyte disturbances [4]. However, the ones which predominate in the causation of PRES are pre-eclampsia and eclampsia [8]. Presentation of PRES with gestational hypertension without associated proteinuria and seizures like in our case are rare incidences.

Pre-eclampsia and eclampsia come under hypertension complicating pregnancies, affecting around 10% of all pregnancies with significant maternal and fetal morbidity and mortality [10]. Pre-eclampsia is characterized by hypertension after 20 weeks of gestation in a previously normotensive individual with signs and symptoms of target organ injury. Eclampsia is the abrupt development of seizures or coma during the gestational period or postpartum not attributable to any other cause [11]. Usually, both these above entities occur between 20 weeks of pregnancy and 48 hours postpartum. In 5% of the cases, it can also develop during the puerperium [12]. Few cases have been reported, where they occur from 48 hours to 4 weeks postpartum, termed late postpartum eclampsia or preeclampsia [13]. PRES with late-onset eclampsia is a rarely encountered entity, causing diagnostic dilemmas in most physicians [14].

The pathophysiology behind the development of PRES is still not well defined. However, it is believed to be due to endothelial dysfunction and disorder in cerebral auto-

regulation. The combination of acute hypertension and endothelial damage can lead to vasogenic edema causing the leakage of fluid from the capillary walls and into the brain interstitium [15].

There are various modes of imaging for diagnosing PRES. But the brain MRI remains the most suitable diagnostic tool [1]. CT, EEG, and other diagnostic tests can be used to exclude other disorders [8]. The most consistent imaging pattern of PRES in MRI is the presence of edema involving the white matter of the posterior portions of both cerebral hemispheres, especially the occipitoparietal regions, in a relatively symmetric pattern sparing the calcarine and paramedian parts of the occipital lobes. Other structures may also be involved like the brain stem, cerebellum, and frontal and temporal lobes. Although PRES usually involves the subcortical white matter, the cortex and the basal ganglia may also be involved. In complicated cases, gyriform signal enhancement or parenchymal hemorrhage can also occur [15]. Accordingly, eclampsia and seizures will occur if this takes place at the motor cortex, whereas lesions at the level of the occipital cortex are related to PRES [16]. PRES can also be manifested by diffuse asymmetric partial or extensive cerebral edema, an ischemic or hemorrhagic stroke causing a mass effect on the cerebral structures, or even hydrocephalus, subarachnoid hemorrhage, or a focal hematoma in 15% of cases. These features may cause difficulty in the diagnosis of PRES [17]. Vasogenic edema extending to the thalamus and annular protuberance, cerebral hemorrhage, and cytotoxic edema may also be encountered [17]. The parieto-occipital region is thought to be more susceptible to vasogenic edema, possibly due to the absence of a sympathetic tone in the vasculature of the basilar artery [15]. Cerebral venous sinus thrombosis may also share the same clinical presentation as that of PRES. So, it must be excluded via imaging tools since it is the most frequent cerebrovascular disorder in the puerperium [14].

Due to relatively diverse clinical presentations and a lack of sufficient data on PRES, the treatment recommendations are somewhat limited. However, the most obvious response is to address pre-eclampsia or eclampsia, the most common cause of PRES. The initial goal in malignant hypertension is to rapidly lower the diastolic pressure to about 100-105 mm Hg. Aggressive blood pressure control is not advised, because this may reduce the blood pressure below the autoregulatory range which may lead to ischemic events like stroke and coronary disease. It has been found that, in most cases of PRES, the neurological symptoms and cerebral lesions disappear completely within days to weeks after control of blood pressure [18]. Nicardipine and Labetalol are the first-line drugs for lowing blood pressure in PRES patients [19]. Nimodipine can reduce the infarction rate caused by cerebral vasospasm [20]. Nitroglycerine is not recommended for blood pressure control as it can aggravate brain edema [21]. Magnesium therapy should be initiated as soon as eclampsia or PRES in pregnancy is suspected, as it treats both seizures and hypertension [2].

The prognosis of PRES is good and 75%-90% of the patients fully recover [18]. Complications may arise from PRES like status epilepticus, intracranial hemorrhage, and cerebral ischemia. Although these complications are rare, they have to be taken seriously as they are life-threatening events. The mortality rate amounts to 3%-6% of PRES patients [20]. The recurrence of PRES is rare [10].

## Conclusion

This study presents a rare case of PRES in a postpartum woman with gestational hypertension but no pre-eclampsia or eclampsia. It is very important to note the possibility of PRES in pregnancy and postpartum period even in the absence of its typical risk factors. Early recognition and appropriate management of PRES are crucial because it is potentially reversible but it also has potential for serious complications. Hence PRES should be one of the differential diagnoses even if a pregnant and postpartum patient presents with only headache as a predominant symptom.

## Registration of research studies

Not applicable

## Provenance and peer review

Noncommissioned, externally peer-reviewed

## Ethical approval

Ethical approval is not required for case reports in my institution (Patan Academy of Health Sciences, Bagmati Lalitpur) so ethical approval was exempted.

## Author contribution

Shailendra Katwal: Conceptualization, mentor and reviewer for this case report and for data interpretation. Aastha Ghimire: Contributed in performing literature review, writing the paper and editing. Amrit Bhusal: Contributed in writing the paper. Abhisek Bajracharya: Contributed in writing the paper. All authors have read and approved the manuscript

## Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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