

# Posterior Reversible Encephalopathy Syndrome Presenting in Late Postpartum Eclampsia

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## Abstract

*Posterior reversible encephalopathy syndrome (PRES) is a severe neurological condition that presents with seizure, headache, visual impairment, accelerated hypertension, altered consciousness, and other neurological symptoms along with abnormal characteristic neuroimaging. Most of the cases are reversible. We described a case of a 31-year-old female with PRES secondary to delayed maternal postpartum eclampsia. The diagnosis was established after magnetic resonance imaging.*

*Background: Posterior reversible encephalopathy syndrome (PRES) was first described in 1996 in a group of patients having hypertension, renal impairment, and immunosuppression(1). There was a constellation of symptoms, including headache, seizure, visual disturbances, and altered conscious level along with characteristic*

*neuroimaging findings. The exact cause is not known but abrupt swinging blood pressure disrupting the blood-brain barrier and endothelial injury seems to be a common denominator(2). In most of the cases, PRES resolves spontaneously and patients show both clinical and radiological improvements.*

*The global incidence of PRES is unknown. It has been reported in patients ranging from 4 to 9 years of age, mostly occurring in young to middle-aged adults with female preponderance(3).*

*We report a case of 28 years 28-year-old woman who developed late postpartum eclampsia complicated by the development of PRES.*

*(J Bangladesh Coll Phys Surg 2024; 42: 297-300)  
DOI: <https://doi.org/10.3329/jbcps.v42i3.74067>*

## Case report

A 31-year-old woman para 2 gravida 2 was admitted through the ER with severe headache and respiratory distress for 3 days. 4 days back she underwent LUCS and gave birth to a healthy baby. She is non-diabetic and has no previous history of hypertension and all through her pregnancy she was well. Due to perioperative blood loss, she was transfused 2 units of red cell concentrate. On 1st POD she developed a headache which was blunt and bilateral with some visual blurring. She also developed shortness of breath which

was initially mild but gradually worsened over 2 days then she was admitted to the general cabin for further management. Then on the 3rd POD, she developed a generalized tonic-clonic seizure for which she was transferred to ICU. On admission to ICU, she was conscious but drowsy GCS E4V3M6, Pupil 3 mm reacting with no other focal neurological features nor papilloedema. Pulse was 100/minute regular, blood pressure was 210/110 mm of Hg, and there was bilateral pitting oedema. Mild basal crackles with tachypnea, no jaundice, and no anemia.

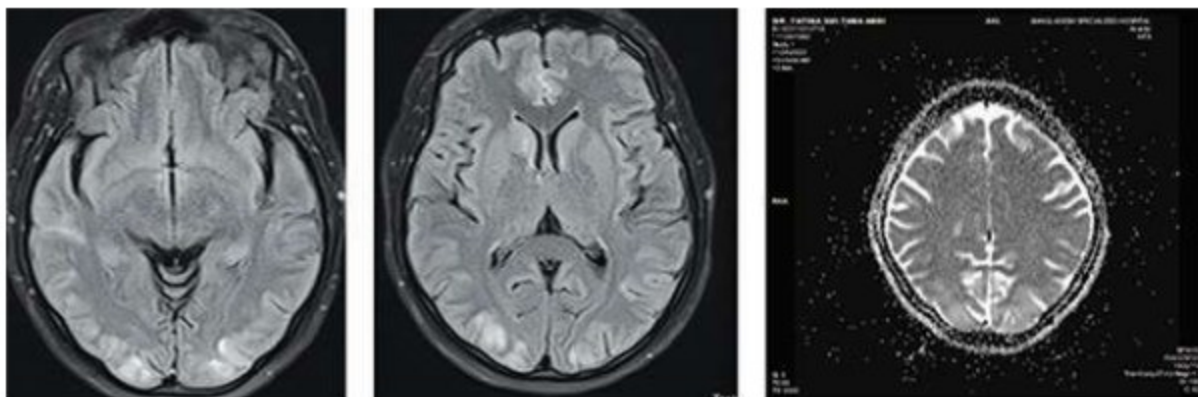
Her ECG and echocardiogram were unremarkable and normal platelet count and derangement of liver enzymes. Urine showing trace albumin and plenty of pus cells. As her headache and drowsiness did not improve along with visual complaints, an MRI of the brain was done which revealed hyperintensity in cortical, subcortical, and bilateral basal ganglia both occipito-parietal lobe in Fluid Attenuated Inversion Recovery with no diffusion restrictions in diffusion weighted imaging thus ruling out cerebral infarction. The patient was treated as a case PRES gradually she improved and was shifted to the ward after 2 days.

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Received: 4 February 2024

Accepted: 2 May 2024



### Differential diagnosis

With the sudden development of headache and bilateral vision loss with elevated blood pressure, we considered an initial diagnosis of ischaemic stroke, cerebral haemorrhage and cerebral venous thrombosis, post-partum Eclampsia, PRES, and hypertensive emergency with retinal haemorrhage. A normal fundus examination essentially ruled out retinal haemorrhage, while the reversibility of symptoms with characteristic MRI findings led us to a diagnosis of PRES.

### Treatment

Blood pressure was controlled with intravenous labetalol and seizure by diazepam. Magnesium sulfate was given in a loading dose of 6 g intravenously over 15 min followed by a maintenance dose of 2 g/h with monitoring of respiratory rate and patellar reflex. The patient was continuously monitored for haemodynamic stability. After an MRI of brain findings consistent with PRES, magnesium was discontinued.

### Follow-up and outcome

The patient's headache rapidly resolved and her vision improved to 20/20 after about 3 h of the onset of symptoms. After a couple of days in the ICU, She continued to be symptom-free and was later transferred to a regular floor. The patient continued to improve clinically and was discharged home symptom-free on the sixth day of hospitalization

### Discussion

PRES has been found increasingly in clinical practice, probably due to advanced neuroimaging facilities' availability. The condition is potentially reversible if diagnosed and treated without delay. It occurs in all age groups and in both genders especially in females of reproductive age(4). The pathophysiology of PRES is elusive and strongly associated with sepsis, hypertension, renal impairment, eclampsia, cytotoxic

drugs, sickle cell anaemia, connective tissue diseases, organ transplantation, etc (5).

There are two leading theories regarding the pathophysiology of PRES(6). The first hypothesis proposes a rapid increase of arterial blood pressure up to a hypertensive crisis or emergency. According to this hypothesis, elevation of blood pressure levels above the upper auto-regulatory limit leads to cerebral hyperperfusion, which may cause vascular leakage and vasogenic edema (7). Increased cerebral perfusion pressure contributes to additional blood-brain barrier dysfunction causing extravasation of plasma and macromolecules through tight-junction proteins(6). The second theory regarding the cause of PRES is that the syndrome is triggered by endothelial dysfunction caused by circulating endogenous or exogenous toxins (6).

The common factor in these diverse conditions is the presence of endogenic (preeclampsia, sepsis) or exogenic (chemotherapy, immunosuppressive agents) toxins causing endothelial dysfunction(8).

PRES may present with non-specific symptoms and manifest acutely or sub-acutely over

several hours or days (9). Encephalopathy and seizures are the most common symptoms followed by visual disturbances, headache, and focal neurological deficits (10, 11)Encephalopathy of varying grades has been reported in 28–94% of patients with PRES. Seizures commonly occur early in the disease course and are observed in 74–87% of patients (9,10). Sometimes status epilepticus may be the presenting symptom of PRES (12). Headache has been reported in 50% of patients (13). It is usually dull, diffuse, and gradual in onset. Varying degrees of visual symptoms have been reported in 39% of patients (9, 13). Fundoscopic examination is often unremarkable but papilledema with flame-shaped retinal hemorrhages

and exudates have been observed in the setting of hypertension. Focal neurological deficits like aphasia and hemiparesis have been observed in 19% of patients (13). On rare occasions, myelopathic symptoms and signs of spinal cord involvement have been demonstrated (14). Other uncommon presentations include abulia, agitation, delusions, opisthotonus, optic ataxia, ocular apraxia, and simultagnosia (15). There is no specific gold standard test for diagnosing PRES. Cerebrospinal fluid (CSF) protein levels are elevated at 70%, but CSF pleocytosis is rare and its presence is a marker of infarction or hemorrhage (16) (17). EEG can help identify patients with ictal or epileptiform activity. Brain imaging is the cornerstone in confirming a diagnosis of PRES. Although vasogenic edema can be visualized on non-contrast computed tomography (CT) in some patients, brain MRI, especially the T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences are much more sensitive (18). FLAIR helps in detecting cortical and a subcortical lesion related to PRES and is an important sequence in establishing its diagnosis (19). Diffusion-weighted imaging combined with apparent diffusion coefficient (ADC) mapping sequences helps differentiate cytotoxic from vasogenic edema and thus may aid in differentiating PRES from ischemic lesions.

#### Common features

Vasogenic cerebral edema  
 Parieto-occipital pattern  
 Holohemispherical watershed distribution  
 Frontal and temporal lobe involvement  
 Subcortical white matter  
 Bilateral, frequently symmetric pattern  
 Hyperintense T2 weighted and FLAIR sequences  
 Isointense, hypointense, or hyperintense lesions on DWI  
 Increased ADC values reflective of vasogenic cerebral edema

#### Uncommon

Brainstem (Central) variant  
 Unilateral PRES  
 Contrast enhancement  
 Microhemorrhages  
 Intracerebral hemorrhages  
 Sulcal SAH  
 Decreased ADC values indicative of ischemia

#### Grades of cerebral edema

Mild  
 Moderate  
 Severe

Imaging findings in PRES

Courtesy: Archana Hinduja

Diagnosis is made by an astute clinician with characteristic clinical symptoms and brain imaging findings. Early diagnosis and management is crucial. Recently the PRES early warning scoring (PEWS) scale which consisted of [1] risk factors, [2] clinical features, and [3] EEG features has improved the prediction of PRES early in suspected cases, with a high index of suspicion in patients with a score of 10 points or higher (20).

#### Conclusion:

PRES is a serious condition that needs to be addressed early to diagnose and treat symptoms and finally remove the possible associated causes. Permanent complications and fatalities have been reported (21). Recurrence of symptoms has been observed in 8% of the cases (22) (22).

#### Conflict of interest:

We have no conflict of interest to declare

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