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Case Report

# Posterior Reversible Encephalopathy Syndrome - A Case Report

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### Abstract

Posterior reversible encephalopathy syndrome (PRES) is a clinico-radiological syndrome characterized by a headache, seizures, altered mental status and visual loss and characterized by white matter vasogenic edema affecting the posterior occipital and parietal lobes of the brain predominantly [1].

We present here a young woman with altered sensorium, generalised tonic-clonic seizures in postpartum stage after massive blood transfusion. Reversibility of the symptoms and characteristic imaging findings led us to a diagnosis of PRES in our patient.

**Keywords:** Posterior Reversible Encephalopathy Syndrome; Seizures

### Introduction

Posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological entity characterised by the acute or subacute onset of headache, altered level of consciousness, visual alterations, seizures, nausea, and vomiting; it also causes neuroimaging alterations, which are generalised, reversible, and predominantly posterior in occipital and parietal lobe [2-4].

Brain magnetic resonance imaging (MRI) is essential for diagnosis as it identifies the presence of oedema surrounding the white matter bilaterally, mainly in the posterior area (parietal and occipital lobes) [3-6]. The pathophysiology of PRES is unknown; several mechanisms have been suggested, and probably coexist in some cases: loss of autoregulatory vascular tone causing hyper perfusion, systemic vasoconstriction with hypoperfusion, and dysfunction or endothelial injury with lesion to the blood-brain barrier [6, 7]. Symptoms fully resolve when the underlying cause is corrected early; otherwise, however, the condition may result in such irreversible damage as cortical blindness or death. MRI abnormalities disappear in follow-up examinations performed after the proper treatment is administered [8-10].

The typical MRI findings of PRES are most apparent as hyperintensity on FLAIR image in the parieto-occipital and posterior frontal cortical and sub cortical white matter, less commonly the brain stem, basal ganglia and cerebellum.

# **Case Report**

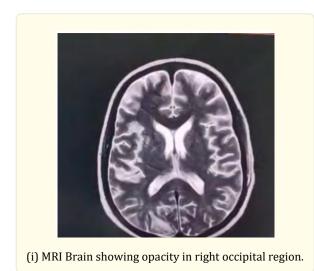
We present here a young woman of 19 years age. Patient relative gave the history and history seems to be reliable. He stated that patient had normal vaginal delivery 1 month ago and later developed severe fatigue, shortness of breath and pedal edema for which she was diagnosed as a case of severe anemia and blood transfusion was advised. During blood transfusion she developed one episode of tonic clonic seizure, headache, vomiting followed by altered sensorium for which she was referred to our hospital as a case of

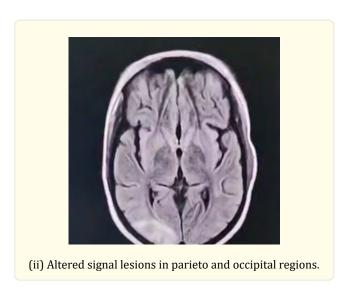
"Altered sensorium cause under evaluation" for which MRI (Brain) was done and blood investigations sent.

MRI brain revealed multiple focal asymmetrical altered signal lesions distributed in supratentorial compartment, predominantly involving sub cortical white matter in bilateral parieto-occipital regions suggestive of PRES syndrome.

Laboratory investigations revealed haemoglobin to be 3 g/dl. Due to severe anemia. Cerebrospinal fluid analysis revealed a normal picture. No history of hypertension was found as per medical records. No other significant history was available. On examination vital signs of the patient were normal. There were no signs of meningeal irritation.

Laboratory examination revealed decreased hemoglobin (3.0 g/dl), neutrophilic leukocytosis (11,000 cells/dl), thrombocytopenia (20,000 cells/cmm), urinary tract infection (8-10 pus cells/high-power field). Urine (Culture and Sensitivity) revealed E. Coli infection sensitive to colchicine and fosfomycin and resistant to all other antibiotics. Liver function tests and renal function tests were normal. Peripheral smear showed macrocytic anemia. Other blood tests, coagulation profile were normal. Malaria Parasite, Widal and Dengue antigen were negative.







(iii) Altered signal lesions distributed in supratentorial compartment and bilateral parieto-occipital regions.

During the admission in SVBP Hospital, patient's consciousness improved after infusion of 2 units of PRBCs. Patient was administered IV antibiotics and antiepileptics and supportive treatment during hospital stay. Gradually the patient gained complete consciousness and was fully oriented to time, place and person at the time of discharge.

Investigations		29/06/2022	02/07/2022
Hematological	НВ	3.0 gm/dl	9.0 gm/dl
	TLC	11000 cells/ micrL	11000 cells/ micrL
	DLC		
	NUETROPHILS	65%	85%
	LYMPHOCYTE	30%	10%
	EOSINOPHIL	03%	03%
	MONOCYTE	02%	02%
	BASOPHILS	00%	00%
	PLATELET COUNT	0.20 Lac cells/mm3	0.20 Lac cells/mm3
	PCV	9%	27%
COAGULATION	P TIME	19 sec	
	INR	1.36	
		29/06/2022	02/07/2022
BIOCHEMISTRY	KFT		
	S.UREA	91 mg/dl	110 mg/dl
	S.CREATININE	1.8 mg/dl	2.42 mg/dl
	TOTAL S. BILIRUBIN	2.8mg/dl	1.9mg/dl
	SGOT	28 IU/L	182 IU/L

ELECTROLYTE	S.SODIUM	139.6 mmol/l	144.1 mmol/l
	S.POTASSIUM	4.26 mmol/l	3.56 mmol/l
	S.CALCIUM	2.36 mmol/l	2.84 mmol/l
TOTAL PROTIEN	S.PROTEIN	7.3 gm/dl	5.3 gm/dl
	T.ALBUMIN	3.5 gm/dl	3.2 gm/dl
	S.GLOBULIN	3.8 gm/dl	2.1 gm/dl
	AG RATIO	0.92:1	1.52:1
LIPID PROFILE	S.CHOLESTROL	97 mg/dl	
	S.TG	171 mg/dl	
SUGAR PROFILE	HbA1C	5.3 %	
VIROLOGY	Anti HCV	NON REACTIVE	
	HBsAg	NEGATIVE	
	HIV1	NON REACTIVE	
	HIV2	NON REACTIVE	

Investigations		04/07/2022	12/07/2022
Hematological	НВ	12.0 gm/dl	11.0 gm/dl
	TLC	17000 cells/ micrL	10800 cells/ micrL
	DLC		
	NUETROPHILS	88%	87%
	LYMPHOCYTE	10%	10%
	EOSINOPHIL	01%	02%
	MONOCYTE	01%	01%
	BASOPHILS	00%	00%
	PLATELET COUNT	0.40 Lac cells/mm3	1.70 Lac cells/mm3
	PCV	36%	33%
BIOCHEMISTRY	KFT		
	T.UREA	76 mg/dl	55 mg/dl
	T.CREATININE	1.8 mg/dl	1.03 mg/dl
	TOTAL S. BILIRUBIN	1.3mg/dl	0.8mg/dl
	SGOT	74 IU/L	27 IU/L
ELECTROLYTE	T.SODIUM	136.1 mmol/l	134.5 mmol/l
	T.POTASSIUM	3.41 mmol/l	4.26 mmol/l
	T.CALCIUM	2.14 mmol/l	2.15 mmol/l

Table 1: Blood and other routine investigations during Hospital stay.

### Discussion

The term PRES has been used based on the similarity in the appearance on imaging, the common location of the parietal-occipital lobe or 'posterior' location of the lesions. The exact pathophysiological mechanism of PRES is still unclear. Three hypotheses have been proposed till now, which include (i) Cerebral vasoconstriction causing subsequent infarcts in the brain, (ii) Failure of cerebral autoregulation with vasogenic edema, and (iii) Endothelial damage with blood-brain barrier disruption further leading to fluid and protein transudation in the brain [6, 7].

In our case report the female patient developed PRES in post partum period during blood transfusion though the mean onset of neurological symptoms after blood transfusion is usually between 7-10 days [14].

Also in our patient blood pressure was normal, in contrast to available data which shows 80-85% cases may exhibit hypertension [15].

## PRES-associated clinical conditions [11-13]

Preeclampsia, Eclampsia, Infection/Sepsis/Shock, Autoimmune disease, Cancer chemotherapy, Transplantation including bone marrow or stem cell transplantation and Massive blood transfusion.

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