



## POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

Dr Seema Dande

Dande hospital, Ravinagar, Nagpur

**ABSTRACT** Posterior reversible encephalopathy is a reversible neurological entity characterised by seizure, headache, visual symptoms, impaired consciousness and other focal neurological deficit. It happens when the auto regulatory mechanism fails to maintain the cerebral blood flow. There is partial lack of sympathetic innervations of the vasculature that emerges from the basilar artery. Edema occurs in the posterior regions of the brain with compression of brain and causing headache, nausea, vomiting and seizures. I present a young woman who was referred to my hospital as a case of sudden loss of vision, severe headache and vomiting. It was her 5th postoperative day after a lower segment caesarean section. The intraoperative and immediate postoperative periods were uneventful. She had no history of seizures. Her blood pressure was 150/100 with albuminuria. She was managed with magnesium sulfate therapy. The reversibility of the symptoms and characteristic imaging findings led us to a diagnosis of PRES syndrome

**KEYWORDS :** Posterior reversible encephalopathy, Seizure, Sudden loss of vision

**INTRODUCTION-****Case history**

A 28 year old woman para 1 who delivered 5 days back by cesarean delivery at a private hospital was referred to my hospital as a case of thromboembolism with acute onset breathlessness and sudden loss of vision. She had no history of convulsions or unconsciousness. She was breathless but conscious oriented and responding to oral commands. She was unable to see us but answering the questions very well. She had severe headache and vomited in the morning. Her antenatal and intrapartum period was uneventful. On examination she was conscious oriented and answering to all the questions. She was unable to see. She was afebrile with pulse rate of 110/min. She was breathless with respiratory rate of 36/min. Oxygen saturation was 84%. She was admitted in ICU, given propped up position and nasal oxygen. Chest was full of basal crepitations. Radiograph of chest showed pulmonary oedema. Blood pressure was 140/100 and urine was positive for albumin. Cardiovascular examination was normal. Neurological examination was normal. Ocular examination revealed diminution of vision in both the eyes. All these findings made me think of PRES syndrome. It was confirmed on MRI.

**Investigations**

Her haemoglobin was 9.5gm/dl. TLC was 14200. neutrophil count was 90%. platelets were 2 lac/cumm  
Urine albumin was 2+  
Liver enzymes were normal  
Serum sodium 135 and potassium was 3.4.  
Coagulation profile was also normal  
MRI revealed bilateral symmetrical T2W/flair hyperintensity in bilateral occipital lobe.

**MANAGEMENT**

She was managed in ICU with nasal oxygen at 2 litres per minute, propped up position and intravenous Furosemide 40 mg immediately after setting an intravenous line. She was started with injection cefoperazone with sulbactam combination. Tablet Nifedipine 10 mg was given orally. She was given 4 grams of magnesium sulfate intravenously slowly as the loading dose. This was followed by 4 gram intramuscularly in each buttock. Magnesium sulfate was given as a maintenance dose of 1 gram per hour for 24 hours. The patient was monitored for respiratory rate, patellar reflex and haemodynamic stability.

She showed improvement. Her tachypnoea settled and her vision started improving gradually. Oxygen saturation started rising. Magnesium sulfate was continued for 24 hours.

**DIFFERENTIAL DIAGNOSIS**

Immunosuppressive therapy  
Nephrotic syndrome  
Sepsis  
SLE

**DISCUSSION-**

PRES is a reversible neurological entity characterised by presence of

white matter oedema affecting the occipital and parietal lobes. The exact incidence is unknown. It can occur at any age. It causes occipital lobe related symptoms like headache, seizures and cortical blindness of acute or subacute onset. In pregnancy it is usually related to pre eclampsia and eclampsia. There is severe impairment of vision limited to distinguishing light and dark, normal optic fundi, normal pupillary reflex. It is preceded by blurring of vision, photophobia, nausea and vomiting.

The symptoms and signs recover rapidly. Neurological impairment or death occurs in a minority of cases. A variety of clinical conditions are associated with development of PRES. The common causes are renal disease, immunosuppressive drugs. Other causes are sepsis, SLE, systemic sclerosis, guillain barre syndrome and AIDS.

The differential diagnosis of PRES includes meningoencephalitis, demyelinating lesions of the brain and cerebral venous thrombosis. Early imaging is crucial in making this distinction. MRI is the imaging modality of choice. PRES appears as a high signal intensity in the posterior regions of the brain. My patient presented with headache, vomiting, breathlessness and cortical blindness on 5th postoperative day. Symptoms showing abnormality at two system – respiratory and neurological system caused a diagnostic dilemma for us.

The management of PRES syndrome requires early diagnosis, treatment of symptoms and correction of causative factor. Timely and appropriate treatment ensures a complete recovery.

**Follow up-**

My patient was observed in ICU for 48 hours. Her blood pressure was controlled. Her headache resolved rapidly. Her vision improved to 20/20. Her urine output was normal. She had her food and fed her baby well.

She was discharged from the hospital after staying for 3 more days outside ICU.

At the time of discharge her vision was normal. She did not have headache or vomiting. Her blood pressure was normal and there was no need of antihypertensive treatment.

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