

**Archana B. Patel,
Samiuddin Shaikh,**
*Department of Pediatrics and
Clinical Epidemiology Unit,
Indira Gandhi Medical College,
Nagpur, India.*

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Posterior Reversible Encephalopathy Syndrome (PRES)

We describe here a case of PRES in a boy with nephrotic syndrome who presented with loss of vision which showed complete reversal following normalization of his blood pressure.

A 5-year-old boy presented with sudden onset headache, diminished vision and loss of consciousness of 8 hours duration. Initially he had loss of vision in bitemporal field which progressed to complete loss in next 6 hours. On admission his Glasgow coma scale score was 2 and blood pressure was 150/100 mm Hg. His blood urea and serum creatinine levels were 80 mg and 3.4 mg % respectively. He received sublingual nifedipine following which his blood pressure became normal and dramatic return of his vision was observed in next 48 hours. Noncontrast CT (NCCT) of head showed geographical areas of hypodensity in the bilateral parietal lobe white matter (*Fig.1a*). A repeat NCCT of head performed after 6 months revealed complete resolution of the white matter abnormality in the parietal lobes (*Fig.1b*). Based on the

clinical presentation, imaging appearances and complete restoration of visual function following normalization of blood pressure a diagnosis of PRES was made.

Posterior reversible encephalopathy syndrome (PRES) refers to a clinico-radiological entity characterized by headache, confusion, visual disturbances, seizures and posterior transient changes on neuroimaging(1). Hinchey, *et al.*(2) in 1996 described this condition and suggested that PRES represented a localized manifestation of hypertensive encephalopathy occurring secondary to hypertensive crisis. Lethargy and somnolence are often the first signs noted. Visual perception abnormalities are invariably observed(1). PRES has been described in several conditions including hypertensive encephalopathy, pre-eclampsia, eclampsia, infections, electrolyte imbalance, hypercalcemia and use of several drugs(1-4). It occurs due to elevated blood pressure which exceeds the autoregulatory capacity of brain vasculature. The posterior circulation supplied by vertebro-basilar system has poor sympathetic innervation and, therefore, is frequently involved(4).

The role of neuroimaging is to establish

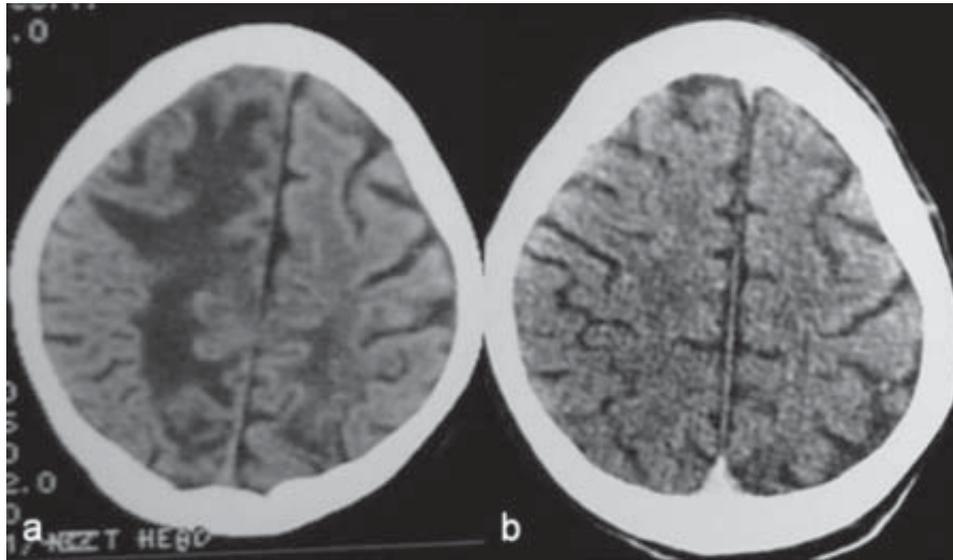


Fig.1. NCCT axial section showing geographical areas of hypodensity in the bilateral parietal lobes white matter more marked in right side (a). Repeat NCCT after 6 months showing complete resolution of the white matter abnormality in bilateral parietal lobes (b).

the initial diagnosis and to exclude other causes of neurological symptoms and signs. NCCT is sufficient to make the diagnosis in a proper clinical setting. MRI features are characteristic and has diagnostic and prognostic value. Diffusion weighted imaging (DWI) can differentiate this condition from ischemia/cytotoxic edema(1,5). Differential diagnosis of PRES includes PCA territory infarcts, venous thrombosis, demyelinating disorders, vasculitis and encephalitis(1). The diagnosis has important implications because the reversibility of the clinico-radiological abnormalities is contingent on the prompt control of blood pressure and/or withdrawing of the offending drug.

**Chandan Jyoti Das,
Ashu Seith,**

*Department of Radiodiagnosis,
All India Institute of Medical Sciences,
New Delhi 110 029, India.
E-mail: ashubhalla1@yahoo.com*

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