



## A Rare Case of Hypertension-Hyponatremia with Posterior Reversible Encephalopathy Syndrome

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### Abstract

*PRES is a clinico-radiological syndrome first described in 1996 in an individual receiving immunosuppression with cyclosporine. Hypertension, occipital blindness, lethargy, transient motor deficits, confusion, visual hallucination and convulsion (GTCS >Focal) constitute PRES<sup>(9)</sup>.*

*PRES is a neurotoxic state associated with unique MRI changes*

**Keywords:** *PRES, Hyponatremia, HTN, convulsion, MRI.*

### Introduction

Posterior Reversible Encephalopathy Syndrome is characterised by headache nausea, vomiting, convulsion, transient motor deficit. It has certain radiological features which allow diagnosis in appropriate clinical setting and enable right therapy to be instituted.

### Case Study

A 13 year female child product of second degree consanguineous marriage from lower socio economic status family presented to casualty with chief complaints of headache for 30 days not associated with fever. Vomiting intermittently for 3-4 days, double vision for 4-5days. Child was neuro-developmenatly normal and not immunised properly but BCG scar was present. Family and sibling history was normal.

O/E-child was conscious, oriented, afebrile, HR-100/min,RR-22/min spo2-99%in room air BP-180/142mmhg (right arm in supine)

Anthropometric measurements were with-in normal limit. Wt=27kg ht= 132cm. On head to toe examination no abnormality was detected some pallor was present and icterus, clubbing, oedema and lymphadenopathy were absent.

On systemic examination of CNS higher mental functions were intact, left lateral rectus palsy with other cranial nerve normally functioning. Fundoscopy shows bilateral papilloedema On motor systemic examination power of both lower limbs 4/5,b/l planter extensor, ataxia, dysdiadochokinesia present. All other systemic examination were normal. On investigation normal serum urea & creatinine value MPICT-negative CRP-negative CBC- within normal limit. Urine routine & microscopic - albumin ++, Serum sodium - 86 meq/l, Serum potassium -1.3 meq/L. Normal 2D echo study of heart. usg abdomen and pelvis- right renal artery stenosis with multiple calculi or calcification in right kidney and compensatory hypertrophy of left

kidney with normal colour flow waveform in left main renal artery.

MRI- Ill defined hyperintense areas in bilateral frontal and parieto -occipital temporal region, predominantly temporal in white matter ,bilateral cerebellar hemisphere body and splenium of corpus callosum. On FLAIR sequence - no restricted diffusion on enhancement. no finding of acute intra cranial hemmorrhage or ICSOL possibly suggestive of PRES SYNDROME.



(MRI-ill defined hyperintense areas in b/l frontal and parieto-occipital temporal region, predominantantly temporal in white matter,b/l cerebellar hemisphere body and splenium of corpus callosum possibly suggestive of PRES)

### Discussion

PRES occurs most commonly in children or adults who develop acute hypertension in the setting of either acute or chronic nephrotic conditions.<sup>(9)</sup> .Most common presenting symptoms in PRES - seizure GTC type, visual disturbance, headache,

transient motor deficits<sup>(9)</sup>. More than 70% patients with PRES are hypertensive though significant proportion have normal or mildly elevated blood pressure.<sup>(3,5)</sup> Seizure and status epilepticus are common and non-convulsive status epilepticus may be more frequent than generalised status epilepticus.<sup>(6)</sup>

Non convulsive status should be suspected in patients with prolonged state of altered consciousness and may be mistaken for post ictal confusion.<sup>(6)</sup> Sign of non -convulsive seizure include stereotypic movements such as staring, eye blinking, or head turning.<sup>(7)</sup> Post ictal confusion last for hours and convulsive status can both persist several days and mistaken for psychosis.<sup>(7)</sup>

Comorbidities associated with PRES include chronic HTN in 50%,chronic kidney disease in 38%, Autoimmune diseases like ITP, SLE and some have exposure to immunosuppressive drug such as cyclosporine, tacrolimus.<sup>(4)</sup> The cause of PRES remains controversial. Auto regulatory compromise of central blood flow plays a vital role.<sup>(2,8)</sup> Un controlled HTN leads to hyper-perfusion and cerebral blood vessels are damaged leading to vasogenic oedema.<sup>(9)</sup> But this theory does not explain why some person with normal BP do develop PRES. An alternate mechanism is systemic inflammatory state causing endothelial dysfunction.<sup>(8)</sup> Brain imaging in PRES usually illustrate fairly symmetric and extensive abnormalities within 24 hours of clinical onset.<sup>(9)</sup> MRI brain is the investigation of choice.

Typical MRI findings is bilateral white matter abnormalities<sup>(9)</sup> in vascular watershed areas in the posterior regions of cerebral hemispheres affecting mostly the occipital and parietal lobes.<sup>(1,4)</sup>

Atypical features include haemorrhage, asymmetrical changes involving frontal lobe and cortex.<sup>(1,4)</sup> PRES may involve both gray and white matter. Lumbar puncture can diagnose infection but may be normal early in the illness or alter antibiotic treatment. No specific treatment is available for PRES .Management of hypertension,

seizure, withdrawal of the offending drug, symptomatic management usually help<sup>(2)</sup> Corticosteroids should theoretically improve vasogenic edema but there is no evidence for their use in PRES<sup>(2)</sup>. Few cases of PRES have been reported till date where hyponatremia is a significant finding as in our case.

### Conclusion

PRES is a rare disorder in children. Radiological features and clinical features correlates for diagnosis. If recognised and treated promptly, the rapid onset symptoms and radiological features usually resolve completely within days to weeks.

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