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## “PRES” sing on an infectious cause

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### Abstract

Posterior reversible encephalopathy syndrome (PRES) is characterized by headache, confusion, seizures and visual loss with different triggers and associated conditions, such as acute hypertension, acute kidney injury, eclampsia, sepsis, multi-organ failure, and autoimmune disease [1]. Infectious cause of PRES is a rare entity. We report a case of left 3<sup>rd</sup> nerve palsy and visual loss with headache, diagnosed to have Atypical PRES due to Leptospirosis.

**Keywords:** Posterior reversible encephalopathy syndrome (PRES), leptospirosis

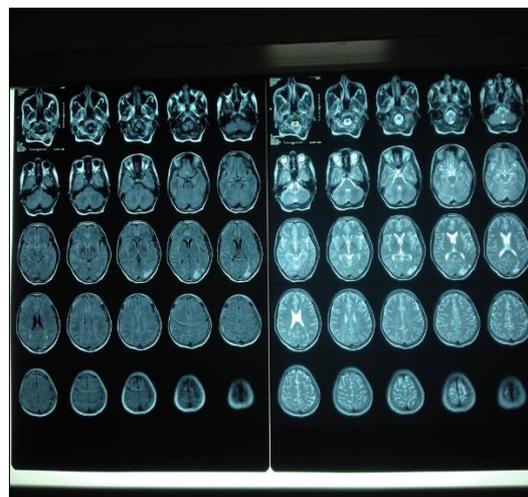
### Introduction

Posterior Reversible Encephalopathy Syndrome (PRES) is a clinico-neuroradiological entity, demonstrated by white-gray matter oedema involving predominantly the posterior region of the brain. PRES can affect any locations in central nervous system including brainstem [2]. Atypical presentations of PRES are being increasingly described. Authors have demonstrated cases of PRES with frontal lobe, cerebellum, basal ganglia or brain stem involvement [3-6]. Unilateral cases of PRES have also been demonstrated [7].

Infection/sepsis may be a cause of PRES, particularly in relation to infection with gram-positive organisms [8]. In infective cause of PRES, immune-mediated capillary endothelial damage, producing vasogenic oedema is implicated [9].

### Case Report

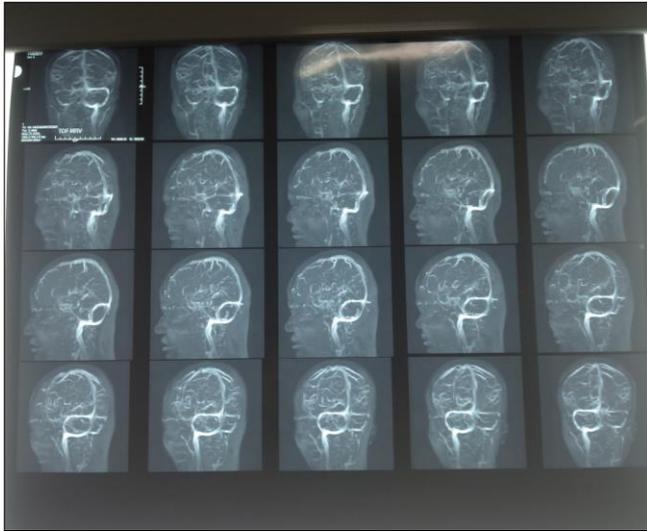
42 year old female patient with no comorbidities presented with the history of acute onset of painless loss of vision in both the eyes with headache since 3 days with background history of fever since 5 days. On examination she had bilateral visual loss with counting finger at 1m with funduscopy showing changes of papilloedema. Next day she developed left complete 3<sup>rd</sup> nerve palsy with diplopia to right gaze. There were no long tract symptoms/signs. Her MRI brain revealed asymmetric areas of T1 hypointense and T2 & FLAIR hyperintense lesions in parietoccipital areas, and in midbrain. MR Venogram was normal.



**Fig 1:** MRI Brain.

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**Fig 2:** MRV.

Her renal parameters were normal. Vasculitic work up was negative.

With background history of fever, her complete haemogram revealed Pancytopenia with Haemoglobin of 10gm%, Total count of 2570/cumm, Platelet count of 20,000. Peripheral smear revealed normocytic RBCs with elliptocytes and anisocytosis with pancytopenia.

Liver function tests were abnormal with SGOT of 153, SGPT of 152 and raised ALP of 483 with normal bilirubin. In view of infectious cause to explain pancytopenia, hepatic dysfunction and PRES, leptospira IgM antibody was sent which was positive.

Patient was started on crystalline penicillin and doxycycline for 14 days. Her counts got normalized in a week, with visual loss and 3<sup>rd</sup> nerve palsy started to improve on 3<sup>rd</sup> day of the treatment with complete resolution in 10days.

### Clinical photographs of the patient



**At admission**



**At discharge**



**Fig 3:** (At admission) left complete and right partial ptosis due to 3<sup>rd</sup> nerve palsy (At discharge) complete improvement

### Discussion

Pres is a clinico-radiological syndrome which is reversible, had we diagnose it at early stages even when symptoms/radiological findings are atypical. Concept of

symmetrical radiological findings and typical location needs a warrant of caution as 28% of PRES can be asymmetrical<sup>[10]</sup> and 18.3% can involve brainstem<sup>2</sup>. Hence, according to few authors “multifocal,” “posterior dominant,” or simply “reversible encephalopathy syndrome” may be a better terminology. The pathophysiology of PRES in infective aetiology involves a complex, integrated response that includes the activation of lymphocytes, inflammatory mediators, and the haemostatic system. Central to this process is an alteration of the endothelial cell function. The cytokine response (TNF- $\alpha$ , IL-1) is believed to play a critical role in the development of the endothelial dysfunction<sup>[11, 12]</sup>.

### Conclusion

It's imperative to search for the cause of PRES with high index of clinical suspicion especially in atypical PRES with definitive etiology. Infections need to be ruled out especially in the presence of sepsis/pancytopenia.

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