

Posterior Reversible Encephalopathy Syndrome (PRES) as a Differential of Sepsis and Isolated Pons Hyperintensity: A Case Report

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Abstract

Posterior reversible encephalopathy (PRES) is a clinico-neuroradiological transient entity with atypical clinical presentation and hyper-intensities in posterior regions of brain on T2 weighted sequence in magnetic resonance imaging. A well-known condition among neuro-radiologists, PRES is still unfamiliar to many intensive care clinicians. Delay in diagnosis may lead to additional morbidity and prolonged ICU stay. So the intensivists should be well aware of this syndrome since prompt recognition and precocious treatment have prognostic implications. We present an unusual case of PRES in association with sepsis and an atypical MRI finding of isolated pontine lesion.

Keywords: Posterior reversible encephalopathy syndrome (PRES); Sepsis; Pontine hyperintensity

Introduction

Posterior reversible encephalopathy (PRES) is a clinico-neuroradiological transient entity characterized by headache, vomiting, vision changes, altered mental status, seizures and magnetic resonance imaging (MRI) findings of parieto-occipital T2 hyperintensity [1]. Although a well-known condition among neuroradiologists, PRES is still unfamiliar to many clinicians working in critical areas, especially because of vague atypical clinical presentations. Though commonly associated with hypertension, pre-eclampsia and immunosuppressive drugs, we present an unusual case of PRES in association with sepsis and an atypical MRI finding of isolated pontine lesion.

Case Report

A 45 years old female presented with chief complaints of fever, headache, vomitings for 2 weeks and loss of consciousness for one day. Her past medical history was unremarkable. She presented in the emergency department in a gasping state and low sensorium, in view of which she was intubated. At the time of admission, patient's vitals were: heart rate 120/minute, blood pressure 114/70 mmHg (MAP=84 mmHg), respiratory rate 36/min, and oxygen saturation 98%. The laboratory findings on admission were: total leukocyte count (TLC) 42400/cu.mm (91% neutrophils with band forms), serum sodium 157 mEq/L, serum potassium 2.2 mEq/L and serum creatinine 2.17 mg/dl. A presumptive diagnosis of metabolic/septic encephalopathy was made and supportive treatment was started in the form of ventilator support, intravenous fluids, electrolytes correction and ICU care. Free water replacement was done with Dextrose 5%. Patient was prescribed empirical antibiotic injection ceftriaxone (100 mg/kg/day). Blood and urine cultures were sterile. Cerebrospinal fluid analysis and fundus examination were normal. She underwent neuro-imaging with 1.5 Tesla GE MRI scanner and T1, T2, ADC, FLAIR, DWI scans were

performed. MRI brain revealed expanded pons with T2 hyperintense signals and central area of diffusion restriction (Figure 1).

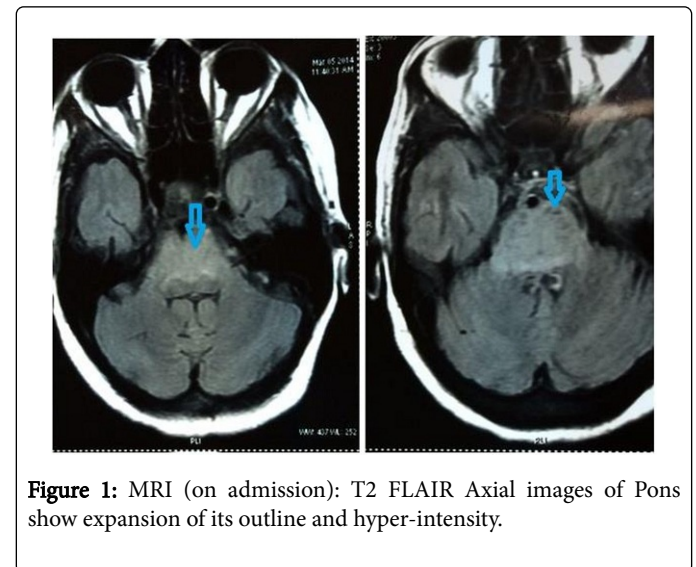


Figure 1: MRI (on admission): T2 FLAIR Axial images of Pons show expansion of its outline and hyper-intensity.

There was hyperintensity on DWI & hypointensity on ADC, showing true diffusion restriction. Post contrast MR showed minimal patchy enhancement. The possibility of osmotic demyelination, pontine glioma or PRES was kept. Over next 24 hours there was improvement in tachycardia and tachypnea. There were no major fluctuations in patient's blood pressure values during treatment. The neurological status improved within 48 hours, TLC started improving and she was weaned off ventilator. The empirical antibiotic was continued for 7 days as there was good clinical response. She was shifted to step down unit on day 4 and discharged on day 10. Follow up scan at 3 months showed interval resolution with small ill-defined T2 hyperintensities in central pons (Figure 2). The rapid improvement in

clinical status along with radiological resolution favours the diagnosis of PRES secondary to sepsis in our case.



Figure 2: Follow-up MRI: Interval resolution with small hyperintensities in central Pons.

Discussion

The case under discussion highlights the unusual isolated pontine distribution of PRES and its association with sepsis. The differential diagnosis included central pontine myelinolysis which is characterised by irreversible demyelinating lesions in central pons typically occurring after the rapid correction of hyponatremia. But our patient classically presented with hypernatremia itself. Other differentials included ischaemic and radiation changes which are generally irreversible and pontine glioma that usually exhibits expansion and mass effect and is also irreversible [2]. The characteristic feature of PRES is reversibility once treatment is initiated, as was observed in our patient.

Hinchey in 1996 first described a reversible neurological syndrome characterized by headache, visual disturbances, seizures and altered mental status associated with neuroimaging findings of predominantly white matter oedema involving the posterior regions of the central nervous system and coined the term posterior reversible leukoencephalopathy [3]. As both white and grey matter can be affected, later the term posterior reversible encephalopathy syndrome (PRES) was introduced [4]. The clinical spectrum of PRES is characterized by acute encephalopathy with headache as the most common symptom, usually encountered in association with acute hypertension, toxemia of pregnancy and exposure to immunosuppressive therapy and less frequently with sepsis, infection and autoimmune diseases [1]. Association of sepsis and PRES is of recent interest, first reported by Bartynski et al. [5]. In a review of 120 patients with PRES, hypertension was the commonest etiology, while sepsis was involved in 7% cases [6]. Our patient also had clinical presentation suggestive of sepsis although no obvious source of infection could be identified.

Computed tomography can be used to detect hypodense lesions of posterior encephalopathy, but magnetic resonance is the gold standard. During acute phase, MRI reveals hyperintense signals on both T2 weighted and flair sequences, including both grey and white matter, typical locations being parieto-occipital region, frontal lobes, temporal lobe and cerebellum [7]. Our case had atypical MRI finding with

mainly pontine involvement and focal area of diffusion restriction. Isolated pontine involvement in a patient with PRES secondary to hypertensive encephalopathy has been reported [2]. There are also recent reviews which have demonstrated brain stem lesions and focal restricted diffusion on diffusion weighted imaging [5,6].

The exact pathogenesis of PRES remains incompletely understood. The favoured pathogenic theory suggests rapidly developing hypertension leading to breakdown in cerebral autoregulation and resulting vasogenic edema. However PRES is seen in increasing frequency without hypertension, as in our patient. In a retrospective review of 25 septic patients diagnosed with PRES, 40% had normal blood pressure and greater brain edema compared with severe hypertension group [5]. Current research suggests that endothelial dysfunction seen in sepsis can explain pathogenesis of PRES. The systemic cytokine mediated inflammatory response in sepsis might result in increased leukocyte adherence, altered vascular tone and vascular permeability culminating in vasogenic edema [5,8].

	Normal values	Day 0	Day 3	Day 7	Day 10
TLC	4000-11000/cu mm	42400	17000	12200	6900
DLC (Differential leukocyte count)	Neutrophils 40-75% Lymphocytes 20-40% Monocytes 2-10% Eosinophils 1-6%	Neutrophils 91% lymphocytes 07% Monocytes 01% Eosinophils 01%			
Serum sodium	135-145 mEq/L	157	153	151	146
Serum potassium	3.5-5.1 mEq/L	2.2	3.5	4.0	3.8
Serum creatinine	0.50-1.30 mg/dl	2.17	1.19	1.18	1.05
Serum AST	5-40 U/L	34			
Serum ALT	5-40 U/L	15			

Table 1: Blood investigations.

Management of patients with PRES should focus on rapid correction of mean arterial blood pressure, hydration using intravenous crystalloid fluids, adequate oxygenation, and correction of coagulopathies and electrolyte disturbances [7].

In conclusion, PRES may complicate the course of sepsis, even in normotensive patients and may be more frequent than reported. This syndrome has broad clinical spectrum and MRI features. The rapid resolution of clinical status with supportive treatment helps in differentiation from other causes of encephalopathy. Delay in diagnosis of this condition may lead to additional morbidity and prolonged ICU stay. So the intensivists should have a high index of suspicion in septic patients with neurological features, since prompt recognition and precocious treatment have prognostic implications (Table 1).

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