

Posterior Reversible Encephalopathy Syndrome (PRES) – Neurocritical Care Emergency

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Dear Editor,

Posterior reversible encephalopathy syndrome (PRES) is a transient clinic radiological syndrome. It was first described in 1996 in a case series by Hinchey et al as a ‘reversible posterior encephalopathy syndrome’ [1]. However in many cases, it is not always reversible. The word “posterior” in its name indicates the signature posterior cerebral imaging findings, usually in the occipital lobe. It is seen in patients with hypertensive encephalopathy linked to emergency conditions like eclampsia, thrombotic thrombocytopenic purpura, porphyrias’ and hemolytic uremic syndrome [2]. This syndrome is also associated with patients with autoimmune conditions like systemic lupus erythematosus (SLE), those taking immunosuppressive therapeutic agents particularly cyclosporine, tacrolimus, methotrexate and interferon; patients with mineral deficiencies, and human immunodeficiency virus (HIV) infection [3]. Typical clinical features of PRES are elevated blood pressure, headache, altered mental functioning, vomiting, seizures, visual loss including cortical blindness, focal neurologic deficits, and altered consciousness [1].

PRES is diagnosed, in addition to the above mentioned clinical features, on magnetic resonance imaging (MRI). The hallmark findings on radio imaging of the brain, located mostly in the parietal, occipital, and the temporal lobes, are the diffuse bilateral hyperintensities on T2 weighted MRI images of the white matter, usually sparing the calcarine and the paramedian occipital lobe structures [4]. The exact pathogenesis of PRES remains unknown [5], however, three popular theories have been postulated. One theory is regarding the failure of the autoregulation system of the cerebral circulation, which may lead to a vasogenic edema in the brain. According to another theory, the elevated blood pressure activates the autoregulatory system in the brain and leads to vasoconstriction of vessels inside the brain, in turn causing hypoperfusion and the development of PRES symptoms. A third theory postulates the immune system activity leading to endothelial

cell-led triggering of a molecular cascade altering normal homeostasis and disruption of the blood-brain barrier leading to leakage of fluid from capillaries and consequent edema [6]. PRES is a condition not readily thought of by medical personnel. A sound knowledge and a good clinical eye can help in early diagnosis and subsequently a definitive diagnosis after excluding other causes of symptoms [7].

PRES is often a missed diagnosis since the radiological signs usually disappear within a day of symptom onset. Early initiation of symptomatic treatment; hyperosmolar therapy to reduce brain edema, lowering of blood pressure, seizure control by anticonvulsant drugs and stopping the offending medications or treating the cause, aids in the early recovery of the patient with good clinical outcome [7].

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