

Short Communication

Alcohol withdrawal induced posterior reversible encephalopathy syndrome

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Posterior Reversible Encephalopathy Syndrome (PRES) is a neurological condition characterized by various clinical findings and imaging abnormalities. Patients can present with a myriad of symptoms ranging from headaches and confusion to seizures and focal neurologic deficits (hemiparesis, aphasia) [1-3]. We report a rare case of PRES induced by acute, malignant hypertension associated with alcohol withdrawal.

A 58-year-old female with a history of hypertension and alcohol abuse was admitted to our critical care unit after she was found unresponsive at home, requiring intubation and sedation by paramedics. Of note, she had been discharged two days earlier following treatment for alcohol intoxication and acute kidney injury. Following that discharge, ramipril was stopped and she abstained from further alcohol use. Her blood pressure was 195/108 mmHg, pulse 102 bpm. She was normothermic with otherwise unremarkable neurologic, cardiac and pulmonary examinations, except for obtundation. Urinalysis, TSH, Ammonia, CT Thorax, urine toxicology, blood ethanol levels, CSF studies, and VBG were unremarkable. CT and CTA head showed subacute infarct within the right parietal lobe near the vertex. MRI brain revealed several hyperintense lesions in the posterior region of bilateral occipital lobes and along the watershed areas between the ACA and MCA territories, involving the subcortical white matter without restricted diffusion and apparent diffusion coefficient (ADC) findings suggestive of vasogenic edema, consistent with PRES. EEG showed frontal intermittent rhythmic delta activity (FIRDA); neurology was consulted and she was started on levetiracetam. Her blood pressure and encephalopathy improved following treatment of withdrawal with intravenous benzodiazepine. She was extubated later that day and was discharged at her baseline two days later on levetiracetam and ramipril. She followed with neurology, underwent repeat MRI showing interval resolution of occipital vasogenic edema and was weaned off of levetiracetam.

There are two proposed mechanisms responsible for PRES that have been extensively discussed in literature: autoregulation/hyperperfusion and cerebral vasoconstriction/hypoperfusion theories.

The autoregulation/hyperperfusion theory describes a failure in the ability of cerebral vessels to respond to variations in blood pressure [4]. With acute and significant elevations in blood pressure, exceeding the upper limit of autoregulation, animal studies have shown cerebral hyperperfusion due to arteriolar dilation, vasogenic edema, and vessel injury [5].

The favoured cerebral vasoconstriction/hypoperfusion theory suggests that cerebral vasoconstriction in response to severe hypertension decreases blood flow to the brain leading to ischemia

and subsequent hypoxia. Release and activation of endothelial factors in response to hypoxia promotes angiogenesis, increased membrane permeability, and breakdown of the blood-brain barrier causing vasogenic edema [4,5]. Findings that support this theory include the tendency of watershed areas to be affected, as evident in our patient's MRI.

Radiologic evidence of bilateral and symmetric gray and white matter edema of parietal and occipital lobes is most common, although the frontal lobe and cerebellum may also be affected [1,2].

PRES can occur in numerous conditions, including sepsis, high-dose chemotherapy, immunosuppressant therapy, pre-eclampsia/eclampsia, and acute hypertension [1]. PRES has been attributed to uncontrolled hypertension in nearly 75% of patients [2].

Resolution of PRES usually occurs with treatment of the underlying cause. Complete recovery has been observed in 2-8 days. However, in severe cases, persistent neurological abnormalities and death can occur [4]. One study evaluated predictors of mortality in PRES and found that elevated CRP, coagulation abnormalities, subarachnoid hemorrhage, and altered mental state were associated with unfavourable outcomes [6].

Learning points for clinicians

It can be difficult to distinguish PRES from other neurologic conditions, particularly alcohol withdrawal and requires a high index of suspicion for timely diagnosis. It requires early imaging and immediate attention. With correction of underlying etiology, which can be quite varied, full neurologic recovery is possible.

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References

1. Hinduja A, Habetz K, Raina SK, Fitzgerald RT (2016) Predictors of intensive care unit utilization in patients with posterior reversible encephalopathy syndrome. *Acta Neurol Belg.* [[Crossref](#)]

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2. Zhang YX, Zheng Y, Zhang BJ, Zhang Y, Ding MP, et al. (2016) Variant Type of Reversible Encephalopathy Syndrome with Diffuse Cerebral White Matter and Brainstem Involvement Associated with Intracranial Hypertension. *Journal of Stroke and Cerebrovascular disease*.
3. Lee VH, Wijidicks EF, Manno EM, Rabinstein AA (2008) Clinical spectrum of reversible posterior leukoencephalopathy syndrome. *Arch Neurol* 65: 205-210. [[Crossref](#)]
4. Fugate JE, Rabinstein AA (2015) Posterior reversible encephalopathy syndrome: clinical and radiological manifestations, pathophysiology, and outstanding questions. *Lancet Neurol* 14: 914-925.
5. Bartynski WS (2008) Posterior Reversible Encephalopathy Syndrome, Part 2: Controversies Surrounding Pathophysiology of Vasogenic Edema. *AJNR Am J Neuroradiol* 29:1043-1049. [[Crossref](#)]
6. Siebert E, Bohner G, Liebig T, Endres M, Liman TG (2016) Factors associated with fatal outcome in posterior reversible encephalopathy syndrome: a retrospective analysis of the Berlin PRES study. *J Neurol* 264(2): 237-242. [[Crossref](#)]

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