Posterior reversible encephalopathy syndrome induced by food poisoning in a pediatric patient: a case report

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Abstract
Posterior reversible encephalopathy syndrome (PRES) can develop in patients following exposure to multiple triggers, including blood pressure fluctuations, kidney diseases, immunosuppressive agents, chemotherapy, or autoimmune disorders. However, to the best of our knowledge, the development of PRES secondary to food poisoning has not been previously reported, especially in a pediatric patient. Here, we report a 13-year-old boy who presented with PRES following the consumption of palmatum (a chicken feet dish). The patient presented with headache, vomiting, and altered consciousness. Neuroimaging findings revealed white matter hyperintensities in a bilateral, symmetrical, and parieto-occipital pattern. The patient was diagnosed with PRES and was managed with fluid expansion and a short-term mannitol regimen (1 g/kg every 12 hours for 3 days). Neuroimaging findings returned to normal at 8 days after admission. Food poisoning may therefore be a new possible trigger for PRES. A timely PRES diagnosis is recommended to prevent possible central nervous system complications.

Keywords
Posterior reversible encephalopathy syndrome, food poisoning, pediatric, white matter hyperintensities, headache, vomiting, altered consciousness

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**Introduction**

Posterior reversible encephalopathy syndrome (PRES) is a clinicoradiological syndrome that was first described in 1996 by Hinchey et al.¹ PRES is a reversible, subcortical, vascular brain edema disease, and is accompanied by a variety of acute nervous system symptoms. The clinical presentation of PRES often encompasses multiple symptoms, including headache, altered consciousness, seizures, and varying degrees of visual impairment.² The typical imaging features include subcortical white matter edema. The bilateral parietal and occipital lobes are most frequently involved. Other affected regions include the frontal lobe, temporal lobe, cerebellum, basal ganglia, brainstem, and thalamus. Most lesions are symmetrical, with mild space-occupying effects.³ To date, a number of triggers, including blood pressure fluctuations, eclampsia, renal diseases, and cytotoxic drugs, have been associated with the development of PRES.⁴–⁷ This study presents a case of PRES in a 13-year-old boy with a history of food poisoning caused by the consumption of palmatum (chicken feet braised with soy sauce). To the best of our knowledge, this is the first report of PRES in a patient with food poisoning.

**Case report**

A 13-year-old boy was admitted to the First Hospital of Jilin University for evaluation following headache, blurred vision, and trembling for 1 day. The patient ate palmatum 1 day before symptom onset. In addition, the patient’s pets (a cat and a dog) died 1 day after ingesting the palmatum. The patient had an otherwise unremarkable history. His blood pressure was 130/90 mmHg, and other physical examinations were normal. No abnormalities were detected in laboratory tests, a poison screening, ultrasound examination of the heart, electrocardiogram, ambulatory electroencephalogram, or chest radiography. However, magnetic resonance imaging (MRI) revealed high signal intensities in the corpus callosum and posterior areas of the brain (Figure 1). The patient was treated with fluid expansion and a short-term mannitol regimen (1 g/kg every 12 hours for 3 days). After 8 days, the brain MRI abnormalities were mostly resolved. These observations are indicative of PRES. After 1 week, the patient’s clinical symptoms (headache, blurred vision, and trembling) had also disappeared, and the patient was discharged from the hospital with an overall positive prognosis.

**Discussion**

The patient presented with headache, blurred vision, and trembling, which can be caused by elevated blood pressure. The patient’s symptoms resembled the most common clinical features of PRES, as have been previously reported.¹² Symptoms with acute or subacute onset usually resolve within hours or days, while other symptoms, such as encephalopathy (50%–80%), epilepsy (60%–75%), headache (50%), visual impairment (33%), focal neurological deficit (10%–15%), and status epilepticus (5%–15%), can last for several weeks.⁸⁹ Some studies have suggested that an overreaction of cerebrovascular self-regulation mechanisms results in
temporary vasospasm and reversible cerebral ischemia.\textsuperscript{8,9}

Reported cases of PRES have mostly been associated with certain conditions, including hypertension (53%), kidney diseases (45%), malignancy (32%), dialysis dependency (21%), and organ transplantation (24%).\textsuperscript{8,9} In addition to the “vasogenic” theory (described in the following paragraph), the pathogenesis of PRES can be closely associated with pathological activation of the immune system, suggesting that a rapid onset of hypertension leads to blood–brain barrier damage, causing vasogenic edema.\textsuperscript{2,10} Although PRES triggers have been identified, an increasing number of atypical PRES cases indicate the possible presence of novel, as-yet-unknown triggers.\textsuperscript{11–13} In the current report, the patient presented with clinicoradiological findings of PRES following the accidental ingestion of contaminated palmatum. This finding may indicate a novel foodborne trigger for PRES.

The failure of cerebral autoregulation following brain edema is a key factor in the pathophysiology of PRES. Four theories have been postulated to explain the cerebral dysregulation that occurs in PRES.\textsuperscript{14} The “vasogenic” theory indicates that an increase in blood pressure might lead to hyperperfusion and vasogenic edema, which in turn can lead to reversible cerebral edema. The “cytotoxic” theory suggests that toxins or chemokines in the blood lead to endothelial dysfunction, which can cause cerebral edema. In the “immunogenic” theory, T-cell activation and cytokines may increase endothelial permeability and vasogenic edema, thus

\textbf{Figure 1.} Neuroradiological findings from a 13-year-old boy who presented with headache, blurred vision, and trembling. \(a\): Diffusion-weighted imaging (DWI) showing white matter hyperintensities in the callosum and posterior areas of the brain, correlating with the vasogenic edemas, upon admission. \(b\): DWI demonstrating the disappearance of white matter hyperintensities at 8 days after admission.
causing cerebral edema. Finally, the “neuropeptide” theory suggests that the release of potent vasoconstrictors might lead to vasospasm and ischemia, and subsequent cerebral edema. In the present report, the pathophysiology of PRES was likely associated with the “cytotoxic” theory because the patient’s pets died after eating the palmatum, which might indicate the presence of certain toxins in the dish.

PRES is characterized by headache, disorders of consciousness, visual disturbances, epileptic seizures, and various focal neurological symptoms, all of which resolve within a few hours or days—usually within 3 to 8 days. Distinctive radiological findings of PRES include bilateral subcortical vasogenic edemas, which are reversible in days or weeks, and are located in the occipital and parietal lobes. In the present report, the headache, blurred vision, and trembling symptoms may have been associated with vasogenic edemas; these symptoms had diminished by 3 days after admission. In addition, the patient’s MRI findings were resolved by 8 days following admission. Clinical findings of PRES can vary among patients, and a radiographic examination is therefore essential to confirm the diagnosis.

In conclusion, this case report highlights that foodborne factors can possibly trigger PRES. A timely PRES diagnosis, along with prompt and proper treatment, can often lead to a favorable prognosis.

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Data availability
The datasets generated and analyzed during the present study are available from the corresponding author on reasonable request.

Declaration of conflicting interest
The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethics statement
This study was approved by the ethics committee of the First Hospital of Jilin University (approval no. 2020-587). All procedures involving human participants were in accordance with the ethical standards of the institutional and/or national research committee, and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Written informed consent was obtained from the patient’s parents.

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