

Difficult postpartum management of a patient complicated by severe PIH and prolonged PRES

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To the Editor:

Pregnancy-induced hypertension (PIH) and eclampsia can develop into a severe complication during maternal period. Posterior reversible encephalopathy syndrome (PRES) appears in a patient with eclampsia, hypertensive encephalopathy, renal failure, and immuno-suppression [1, 2]. It is a neuroradiological diagnosis established by magnetic resonance imaging findings in which regional brain edema is revealed—most likely in the occipital lobe [1–3]. Patients commonly present with headache, consciousness disturbances, visual disturbances, or seizure [1, 2]. Several differences between pregnant and non-pregnant patients with PRES have been reported, with the former characterized by a younger mean age (22 vs. 49 years, respectively), less premedical history, and a higher occurrence of headache [4]. The progression rate of eclampsia or pre-eclampsia to PRES is unknown at the present time. One hypothesis is that this regional brain edema is related to the vulnerability of auto-regulation due to fewer sympathetic innervations of the vertebrobasilar vasculature [1]. Here, we present a patient with severe PIH complicated by prolonged PRES that appeared in the postpartum period.

A 40-year-old female with PIH was urgently admitted to our hospital due to abruptio placenta at 35 weeks and

5 days of gestation. Urgent caesarean section was performed under general anesthesia, and the patient's condition subsequently became complicated by hypo-volemic shock due to excessive postpartum bleeding. The patient was extubated on post-operative day (POD) 1, but blood transfusion, including irradiated red cells concentrates leukocyte-reduced, fresh frozen plasma, and platelet concentrate, were required to treat the hypo-volemia, acute renal failure, and disseminated intravascular coagulation (DIC) in which the maximum D-dimer was 63 $\mu\text{g/mL}$ and the lowest platelet count was 44,000/ μL . Recombinant AT-III and continuous nafamostat-mesilate were also administered to treat the DIC. During this acute period, fluids, including blood products, were overloaded to maintain arterial blood pressure and urine volume (+18,000 mL post-surgical cumulative volume balance on POD 5) (Fig. 1). On POD 5, the patient suddenly developed general seizure, and a flair MRI demonstrated a high-intensity region located on the right posterior occipital lobe (Fig. 2). Postpartum eclampsia was suspected clinically, and neuroradiological imaging revealed PRES. Phenytoin 500 mg/day was administered in addition to diazepam, and continuous propofol infusion and mechanical ventilation were resumed after re-intubation. Central venous pressure and continuous cardiac output through the arterial catheter (APCO) were started to be monitored. Blood pressure was maintained 160–140/100–90 mmHg. After this period, the water balance was adjusted each day to maintain the threshold below zero until POD 21 because the APCO (APCI) was 6.0–9.8 L/min (4.0–6.5 L/min/ m^2). The C-reactive protein level ranged from 10.0 to 27.0 mg/dL during the period between POD 0 and POD 21. However, renal function subsequently worsened, and the patient had a recurrence of general seizure on POD 22, 3 days following the discontinuation of phenytoin. Although fluid

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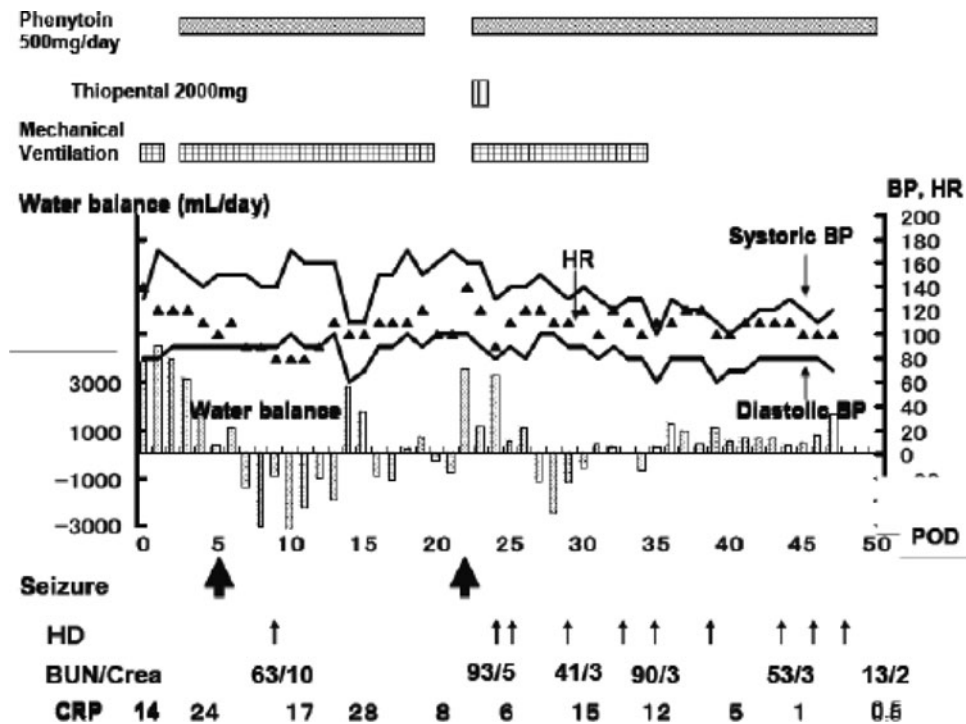


Fig. 1 For 5 days following the emergency caesarian section, fluids and blood products were infused to maintain circulating blood volume against increased vascular permeability. Seizure occurred suddenly on post-operative day (POD) 5 (arrow), at which time limitation of fluid administration was started. Blood pressure (BP) was maintained around 150/90 mmHg until POD 22, when a second seizure occurred

(arrow). Thereafter, the blood pressure was decreased to 120–140/80–100 mmHg using a nicardipine infusion. Although urine volume was 2,000–3,000 mL/day, hemodialysis (HD) was performed several times to improve the blood urea nitrogen (BUN):creatinine ratio. After 50 days in the Intensive Care Unit, the patient was discharged without any neurological deficit. CRP C-reactive protein, HR heart rate

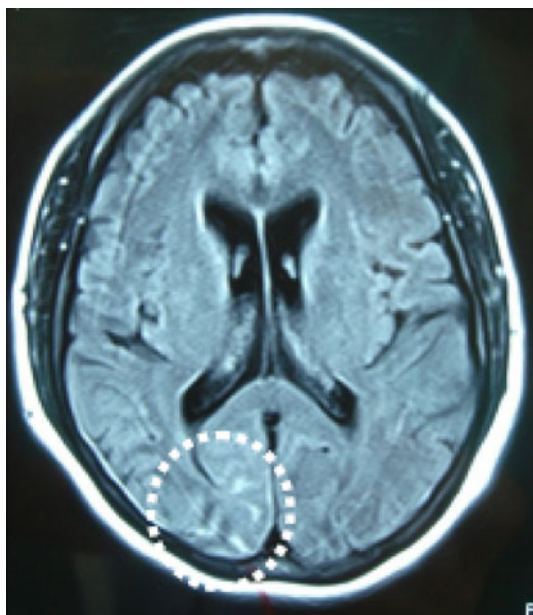


Fig. 2 On POD 5, flair magnetic resonance imaging (MRI) demonstrated a high-intensity region (encircled) in the right posterior occipital lobe

administration was limited during this period, flair MRI again demonstrated a high-intensity region located on the bilateral posterior occipital lobe, the intensity of which appeared to have worsened compared to earlier findings (Fig. 3). The blood pressure at this time was 170–150/100–80 mmHg. After intravenous diazepam had been administered several times, thiopental (3–5 mg/kg/h) was infused continuously for 1 day, with the burst and suppression confirmed on the electrocardiogram monitor, and then phenytoin was restarted. Thereafter, we changed the therapeutic goal to a brain-targeted therapy that focused on decreasing the systolic blood pressure to <140 mmHg with intravenous nicardipine (1–10 mg/h) infusion while maintaining a hypo-volemic condition. Since the blood urea nitrogen (BUN) and creatinine levels had increased despite a urine output of 2000–3000 mL per day, the patient was put on hemodialysis (HD) several times after the second seizure attack. The consciousness level improved gradually and reached 15 points on the Glasgow Coma Scale on POD 41. The patient was discharged from the Intensive Care Unit on POD 50 without any neurological deficits, and flair MRI did not detect any sign of brain edema at that time.

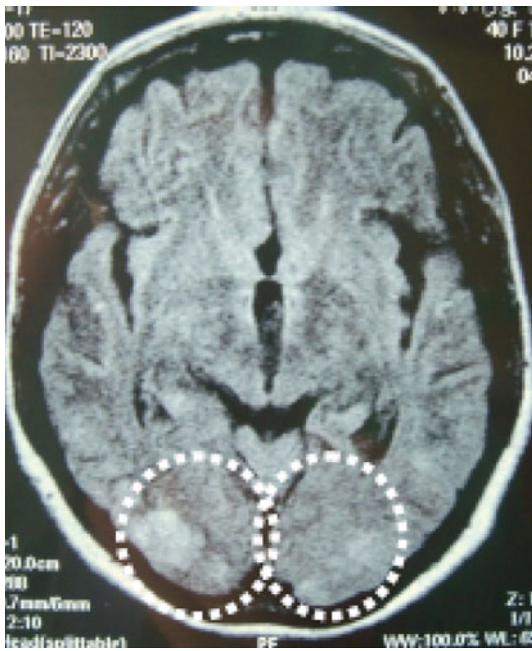


Fig. 3 On POD 22, flair MRI demonstrated high-intensity regions (*encircled*) in the bilateral posterior occipital lobe

Two recommended treatments for improving PRES are to decrease the blood pressure and to decrease fluid administration [1, 2]. Under the condition of severe systemic inflammation caused by PIH and infection, these treatments may be difficult choices for the physician to make because other organs may fail as a consequence of a decreased blood flow. However, it has also been reported that PRES may not be reversible if not treated promptly

[1, 2]. Therefore, we consider that priority should be given to improving the neurological abnormality despite the risk of worsening failure of other organs. In our case, vasodilator should have been administered more promptly to prevent PRES even though the blood pressure was not extremely high. The goal of blood pressure control to avoid postpartum eclampsia or PRES may vary among each patient. We also put the patient on HD several times to support the impaired renal function. However, the HD may have worsened the brain edema due to dialysis disequilibrium syndrome (DDS). A slower hemodialysis process, such as continuous hemodiafiltration (CHDF) may be more beneficial in providing support for the impaired renal function and fluid management without worsening the brain edema [5].

References

1. Thackeray E, Tielborg M. Posterior encephalopathy syndrome in a patient with severe preeclampsia. *Anesth Analg*. 2007;105:184–6.
2. Paresaei M, Derwig I, Yoon J, Erskine KJ, Jarman PR. Posterior reversible leukoencephalopathy in a case of post partum eclampsia. *Am J Obstet Gynecol*. 2005;193:885–6.
3. Tsuzuki N, Katoh H, Toyooka T. Magnetic resonance imaging in patients with eclampsia and preeclampsia. *Neurosurg Emerg*. 2007;12:78–86.
4. Ross C, Ferbert A. Posterior reversible encephalopathy syndrome: is there a difference between pregnant and non-pregnant patients? *Eur Neurol*. 2009;62:142–8.
5. Kennedy AC, Linton AL, Eaton JC. Urea levels in cerebrospinal fluid after hemodialysis. *Lancet* 1962;410–11.