Dear Editor,

A 27-year-old female patient was admitted to the emergency department with symptoms of severe headache, blurred vision, and imbalance. From her past medical history, it was learned that she gave birth to her second baby by cesarean section one week before, her headache started on the second postnatal day along with nausea and vomiting, and that her systolic blood pressure was measured as 190 mmHg during this period. Her arterial blood pressure was 155/90 mmHg in the emergency department. Other systemic examination findings were normal. A neurologic examination revealed no pathologic findings except truncal ataxia, and the fundus examination was normal. Computed tomography of the patient revealed narrowed cerebral sulci and compressed ventricles. Diffuse cortical hyperintensity was observed in the cerebellar vermis, bilateral parietooccipital, frontal and temporal regions on FLAIR-weighted magnetic resonance imaging (MRI) (Figure 1). Cranial MR venography demonstrated patent venous structures. The patient was hospitalized with a diagnosis of posterior reversible encephalopathy syndrome (PRES) and amlodipine 5 mg/day was initiated for her high blood pressure. Proteinuria was detected (urine total protein 503.64 mg/day; normal <150 mg/day). In light of the radiologic and clinical findings, it was thought that the headache and ataxia had developed due to PRES on the basis of preeclampsia. The patient, whose hypertension was under control, was discharged after a follow-up period of five days, with significant improvement in ataxia and headache. Two days later (postpartum 12th day), the patient was re-admitted to the emergency department with a generalized tonic clonic seizure. A 24-hour magnesium infusion was initiated following a gynecology consultation with the diagnosis of eclampsia and no further seizures were observed during the follow-up. On the 15th day, her blood pressure values were within normal limits and it was observed that the hyperintensity in the posterior area on MRI decreased. MRI was completely normal in the 1st month (Figures 2, 3).

PRES is defined as an encephalopathic condition in which parietal and occipital regions are more affected in neuroimaging (1). The pathophysiology involves development of focal vasogenic edema in the brain with disruption of the blood brain barrier (BBB) (2). As in our case, high and uncontrolled blood pressure is thought to disrupt the BBB. However, the development of generalized convulsions five days after achieving normal blood pressure values in our case suggests that a process other than high blood pressure prolongs the pathophysiologic process in eclampsia. In fact, it has been suggested that factors such as factor 8 antigen and fibronectin occurring due to fetomaternal immunologic disorder in eclampsia spread to the whole system and cause endothelial damage (3).

PRES may also present with altered levels of consciousness, ranging from somnolence to lethargy, as well as with headache, nausea, vomiting, visual changes, weakness in the extremities, and convulsive seizures (4). There are very few PRES cases with predominant ataxia in the postpartum period, as in our case (5). In addition, PRES usually develops between the 20th week of gestation and the first 48 hours after delivery in patients with eclampsia, and rarely in the late postpartum period (postpartum 48th hour - 30 days), as in our case (6).

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Received/Geliş Tarihi: 13.05.2018 Accepted/Kabul Tarihi: 29.06.2018
Figure 1. FLAIR magnetic resonance images on postpartum 5th day. Hyperintensities in the cerebellar vermis (A), temporooccipital cortex (B) and frontoparietal cortex (C) are shown by arrows.

Figure 2. FLAIR magnetic resonance images on the cerebellar vermis (A), temporooccipital cortex (B) and frontoparietal cortex (C) postpartum 15th day.

Figure 3. FLAIR magnetic resonance images in the cerebellar vermis (A), temporooccipital cortex (B) and frontoparietal cortex (C) first month of the treatment.

Ethics

Informed Consent: Consent form was filled out by all participants.

Peer-review: Internally peer-reviewed.

Authorship Contributions


Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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