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## Research Letter

### Management of seizure on Postpartum Day 8: A case report of late postpartum eclampsia



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#### ARTICLE INFO

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##### Dear Editor,

A healthy 25-year-old primigravida, at 38 weeks and 4 days of pregnancy, was admitted to our hospital in active labor. Epidural anesthesia was administered; the patient underwent a cesarean section due to cervical dystocia and a healthy boy was delivered. Orthostatic headache was noted 1 day after, and suspecting an accidental dural puncture, a therapy with paracetamol, hydration, and bed rest was administered. The headache was stable, and a brain magnetic resonance imaging (MRI) study was proposed, but not executed because of patient refusal. Lumbar spinal MRI, blood pressure recordings, and blood tests were all normal. On Postpartum Day 8, the patient had a generalized tonic–clonic seizure that was terminated with diazepam. A brain computed tomography (CT) scan was performed, which showed a small amount of subarachnoid hemorrhage. At the end of the scan, the patient had a second seizure that was treated with midazolam, sedation, and intubation. A CT angiogram showed no evidence of vascular malformations. A subsequent MRI scan showed MRI multiple bilateral T2-weighted hyperintensity in the cortical and subcortical white matter, subarachnoid hyperintensity on fluid attenuated inversion recovery sequences, and leptomeningeal enhancement. Vasogenic edema was suspected in diffusion weighted images (Figure 1).

The patient was admitted to the intensive care unit. After the interruption of sedation, she developed a third generalized seizure, which was treated by propofol. Systolic blood pressure was 220 mmHg. The patient was sedated and given phenytoin, hydralazine, and labetalol intravenously. Electroencephalography was

normal, and a CT scan showed no change. A magnesium sulfate drip was started (5 g loading dose followed by 1 g/hour). On Postpartum Day 15, an MRI scan was performed, showing complete resolution of the T2-weighted signal alterations; extubation was performed and the sedation was interrupted. On Postpartum Day 18, the patient was admitted to the gynecology department and was discharged home on Postpartum Day 24.

Late postpartum eclampsia is defined as eclamptic seizure developing later than 48 hours postpartum, and in 44% of the cases, it occurs without pre-eclamptic signs and symptoms, making an early diagnosis difficult [1]. Headache was the only symptom present at onset in our patient. Epidural analgesia contributed to the delay in diagnosis, because the headache could be a side effect of the procedure. Moreover, other causes of seizure could be excluded, but the patient refused MRI.

Owing to the CT finding of smooth subarachnoid hemorrhage, we decided to administer conventional anticonvulsants. Since subarachnoid hemorrhage most often originates from a ruptured aneurysm or from arteriovenous malformations, we performed a CT angiogram study that proved to be normal. Brain MRI evaluation showed findings suggestive of eclamptic posterior reversible encephalopathy syndrome (PRES), and we decided to treat her as an eclamptic patient.

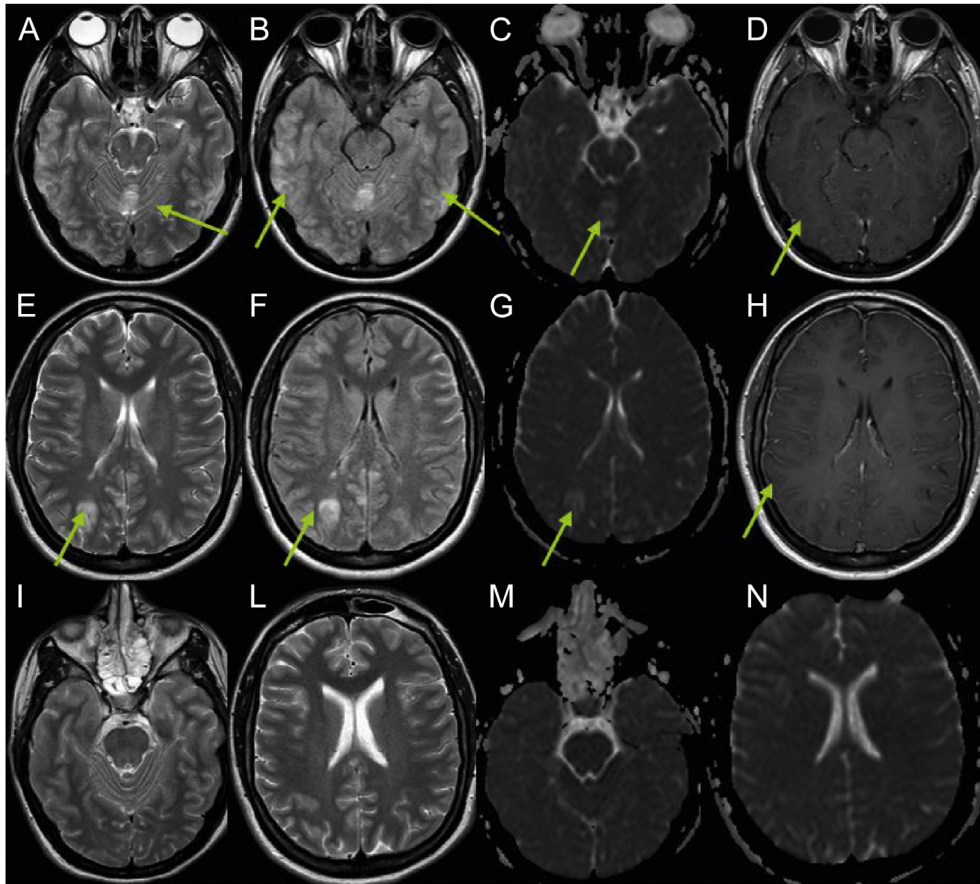
Magnesium sulfate is the drug of choice in prevention of recurrent convulsions in eclampsia [2], but conventional anticonvulsants could be administered when the diagnosis is uncertain.

Recently, it has been reported that all eclamptic patients who underwent MRI had findings of PRES [3].

PRES has in most cases a typical MRI appearance, characterized by white-matter vasogenic edema predominantly affecting the parieto-occipital regions of the cerebral hemispheres, with involvement of both cortical and subcortical tissue. It usually increases in the first days of onset and decreases when the systemic changes come under control. Some variants of presentation have been described showing a nonposterior distribution, mainly in watershed areas, and the presence of combined vasogenic and cytotoxic edema. Diffusion weighted imaging is usually normal because of the absence of a true acute infarct, and in some cases the apparent diffusion coefficient (ADC) signal increases in affected regions due to increased diffusion [4,5].

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**Figure 1.** (A) Axial T2, (B) axial FLAIR, (C) axial diffusion map, (D) axial T1 after gadolinium (gd) at the temporo-occipital level, and (E) axial T2, (F) axial FLAIR, (G) axial diffusion map, (H) axial T1 after gd at the frontotemporo-occipital level; note the multiple focal hyperintensities (arrows) and enhancement of the dura. (I) Axial T2 at the temporo-occipital level, (L) axial T2 at the frontotemporo-occipital level, (M) axial diffusion map at the temporo-occipital level, and (N) axial diffusion map at frontotemporo-occipital level; brain MRI is normal without signal abnormalities.

Our patient had MRI findings suggestive of eclamptic PRES; therefore, magnesium sulfate and antihypertensive therapy were administered.

The pathogenic mechanism of seizure could be the same in late postpartum eclampsia and PRES: a loss of autoregulation of cerebral blood flow resulting in cerebral hyperperfusion and an increase in permeability of the blood–brain barrier leading to vasogenic edema.

In addition, PRES has been described in a patient with pre-eclampsia [6], indicating PRES as an antecedent to an eclamptic seizure and not as the result. Early MRI changes such as posterior sulcal hyperintensity and leptomeningeal enhancement were found preceding PRES by several days [7].

Considering the significant morbidity of late postpartum eclampsia, we recommend training medical staff and patients on the prodromal symptoms and clinic–radiological presentation.

#### Conflicts of interest

The authors have no conflicts of interest relevant to this article.

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