

# Ultra-Early Magnetic Resonance Imaging Findings of Eclampsia

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**Objective:** To report the very early magnetic resonance imaging features of eclampsia.

**Design:** Case report.

**Setting:** Inpatient neurology service.

**Patient:** A 35-year-old woman who developed late postpartum eclampsia with cerebral edema, caudate hemorrhage, and ischemic strokes.

**Main Outcome Measure:** Very early magnetic resonance imaging features.

**Results:** Magnetic resonance imaging with gadolinium showed posterior sulcal hyperintensity and leptomeningeal enhancement preceding the development of posterior reversible encephalopathy syndrome, hemorrhage, and ischemic strokes by days.

**Conclusion:** Magnetic resonance imaging with gadolinium shows early characteristic findings that precede the more classic clinical presentation of eclampsia.

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**E**CLAMPسيا IS A COMPLEX HYPERTENSIVE disorder of pregnancy, commonly affecting the central nervous system. Cerebrovascular involvement is the direct mechanism of death in 40% of patients.<sup>1</sup> Typical brain magnetic resonance imaging (MRI) findings in eclampsia, including vasogenic edema with T2 hyperintensity in the posterior subcortical and deep white matter, are also characteristic of posterior reversible encephalopathy syndrome (PRES).<sup>2-5</sup> Less commonly, postpartum angiopathy with segmental vasoconstriction can manifest as ischemia with cytotoxic edema or hemorrhage.<sup>6-8</sup> Approximately 44% of late postpartum eclampsia, defined as occurring more than 48 hours but less than 4 weeks postpartum, fails to manifest the hypertension, pedal edema, and proteinuria of preeclampsia prior to convulsion,<sup>9</sup> which makes early diagnosis difficult.

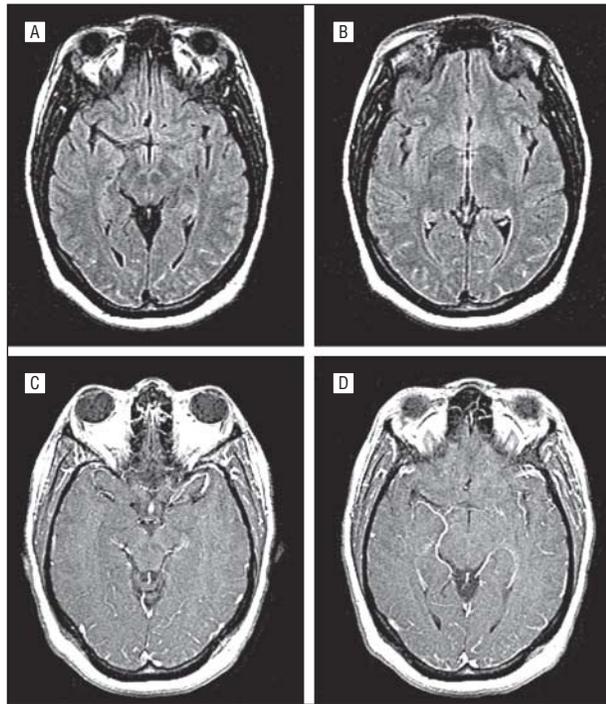
We describe the very early MRI changes of posterior sulcal hyperintensity and leptomeningeal enhancement as the first radiological sign, preceding the development of postpartum eclamptic PRES, hemorrhage, and ischemic strokes by days.

## REPORT OF A CASE

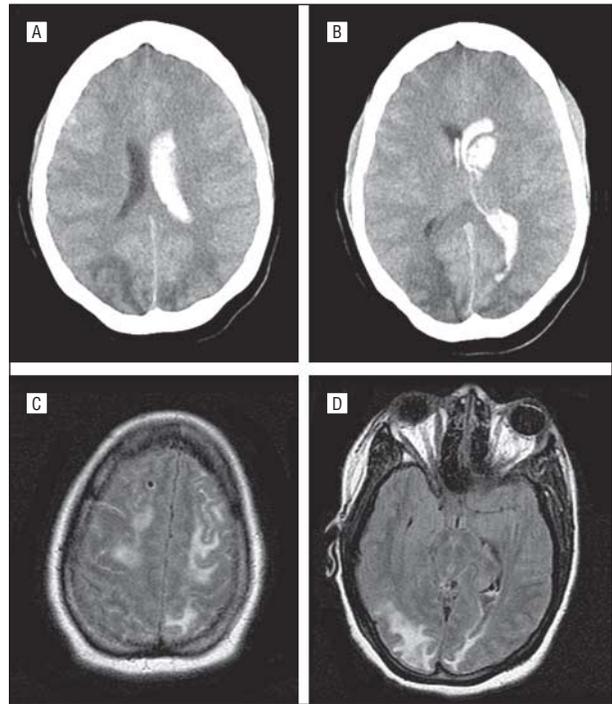
A 35-year-old woman (gravida 3, para 3) with previous migraines had an uncom-

plicated term vaginal delivery with spinal epidural anesthesia. Seven days postpartum, she developed severe left-sided headache accompanied by photophobia, phonophobia, nausea, and a fever. At 10 days postpartum, she was seen in the emergency department with a fever of 102.3°F and blood pressure of 160/82 mm Hg. She had no proteinuria or peripheral edema. Cerebrospinal fluid values for white blood cells were 1/mm<sup>3</sup>; red blood cells, 73/mm<sup>3</sup>; and protein, 42 mg/dL. Magnetic resonance imaging showed mild sulcal hyperintensity on fluid-attenuated inversion recovery (FLAIR) sequences and leptomeningeal enhancement on postgadolinium T1 sequences in the posterior parietal, temporal, and occipital lobes (**Figure 1**). There was no venous occlusion on a magnetic resonance venogram. Fourteen days postpartum, she was found unresponsive, with blood pressure of 200/100 mm Hg. Brain computed tomography (CT) and MRI showed left caudate hemorrhage with intraventricular extension and diffuse vasogenic edema in the bilateral posterior parietal, occipital, and frontal subcortical white matters (**Figure 2**). Computed tomography angiogram showed no evidence of vasoconstriction or vascular malformation. She developed refractory hypertension requiring intravenous magnesium sulfate and multiple antihypertensive agents. An external ventricular drain was placed, and hy-

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**Figure 1.** Magnetic resonance imaging fluid-attenuated inversion recovery on postpartum day 10 showing sulcal T2 hyperintensity in the posterior parietal, temporal, and occipital lobes (A and B). Postgadolinium T1 image showing leptomeningeal enhancement in the corresponding area (C and D).

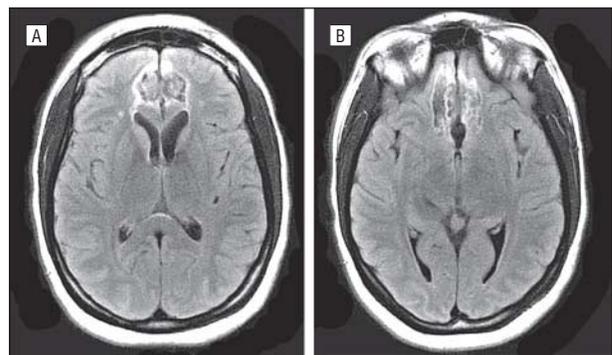


**Figure 2.** Noncontrast head computed tomography on postpartum day 14 showing left caudate hemorrhage with intraventricular extension and bilateral frontal, parietal, and occipital lobe vasogenic edema (A and B). Magnetic resonance imaging fluid-attenuated inversion recovery on postpartum day 18 showing vasogenic edema in the corresponding area (C and D).

perosmolar therapy was started for elevated intracranial pressure (peak intracranial pressure, 37 mm H<sub>2</sub>O). A brain CT 29 days postpartum showed hypodensities consistent with cerebral infarction in the bilateral proximal anterior cerebral artery territories that were absent 3 days prior. Repeated CT angiogram did not show any evidence of major arterial vasoconstriction or occlusion. She was disinhibited and severely inattentive and had both anterograde and retrograde amnesia. Five months later, the MRI showed evolution of the bilateral anterior cerebral artery territory infarctions and complete resolution of the subcortical vasogenic edema (**Figure 3**).

#### COMMENT

This patient had subtle MRI findings 4 days before the appearance of more typical eclamptic PRES and intracerebral hemorrhage. The early findings were sulcal hyperintensity on the FLAIR sequence and leptomeningeal enhancement by gadolinium. There was no cerebrospinal abnormality, such as subarachnoid hemorrhage or meningitis, to account for the increased T2 signal in the subarachnoid space. Although the first MRI was obtained 10 hours after the lumbar puncture, the absence of dural enhancement makes it unlikely that the findings were the result of low pressure.<sup>10</sup> Alteration of the regional hemodynamics from congestion, inflammation, or slow flow has been suggested as the cause of sulcal hyperintensity on FLAIR imaging.<sup>11</sup> Furthermore, endothelial damage associated with eclamptic PRES<sup>12</sup> may contribute to the increased



**Figure 3.** Magnetic resonance imaging fluid-attenuated inversion recovery after 5 months showing resolution of the vasogenic edema and evolution of the bilateral anterior cerebral artery territory infarctions.

blood-brain barrier permeability, thus causing leptomeningeal enhancement.

We conclude that MRI with contrast may show characteristic findings of sulcal hyperintensity and leptomeningeal enhancement in early preeclampsia that could precede the more classic clinical presentation, such as hypertension, proteinuria, brain edema, or seizure. These early imaging characteristics support the hypothesis that regional changes in cerebral hemodynamics and blood-brain barrier permeability, independent of blood pressure, may play a major role in the pathogenesis of cerebrovascular manifestation of eclampsia. Identifying these characteristic MRI findings in the appropriate clinical setting may lead to earlier recognition and prompt treatment of eclampsia.

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