

Late Postpartum Eclampsia 16 Days after Delivery: Case Report With Clinical, Radiologic, and Pathophysiologic Correlations

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Background: Postpartum eclampsia is a rare, frightening, and potentially tragic complication of hypertensive pregnancies, usually developing within 48 hours of delivery. Seizures occurring days to weeks after parturition are exceedingly uncommon and require rapid, precise clinical evaluation by multiple specialists.

Methods: A case presentation of delayed postpartum eclampsia illustrates unique features of the syndrome. Extensive review of the literature highlights pathogenesis, controversies, and dilemmas surrounding this enigmatic hypertensive disorder.

Results and Conclusions: A 39-year-old hypertensive patient had an uneventful full-term delivery by her family physician only to develop headache, double vision, and recurrent tonic-clonic seizures 16 days later. Initial evaluation showed severe hypertension, diplopia, hyperreflexia, proteinuria, and hyperuricemia. She was given a magnesium sulfate infusion. Magnetic resonance imaging (MRI) documented asymmetric ischemic foci within gray matter in the distribution of the posterior cerebral arteries. All symptoms, signs, and abnormal laboratory values resolved within 4 days. A follow-up MRI showed complete resolution of all cytotoxic cortical lesions. Based on human autopsy data, radiologic investigations, and animal studies, eclampsia is believed to result from explosive vasospasm, endothelial dysfunction, and cytotoxic edema of cerebral cortex. This central nervous system vasculopathy is most prominent in the posterior cerebral vasculature and is often rapidly reversible. Difficulties in differential diagnosis, typical findings on neuroimaging, and urgent management strategies are discussed. The time limit for postpartum eclampsia probably should be lengthened to 4 weeks, as indicated by our case and other clinical series. (J Am Board Fam Pract 2000;13:39–46.)

Eclampsia, a dramatic and often unpredictable complication of pregnancy-induced hypertensive disorders, is characterized by sudden hypertension, proteinuria, edema, and seizures.^{1–3} A relatively rare syndrome, eclampsia complicates approximately 3 in 1000 pregnancies, with higher incidence rates in preeclamptic or twin pregnancies, women of low socioeconomic status or in developing countries, and nulliparous patients younger than 20 years or multiparous patients older than 35 years of age.^{4,5} Whether preceded during prenatal visits by prodromal evidence of preeclampsia, or occurring without antecedent warning symptoms, the great majority of eclamptic seizures occur in the antepartum setting between 20 and 40 weeks of gestation or within a few hours to 2 days postpartum.

Most authorities report that 50%, 25%, and 25% of seizures occur in the antepartum, intrapartum, and postpartum periods, respectively.⁵ Controversy surrounds the occurrence of eclampsia developing longer than 48 hours after delivery. Some authors are skeptical that a relation exists between pregnancy and any seizure occurring more than 2 days postpartum.^{1,6} Others, however, acknowledge the development of postpartum eclampsia as late as 3 to 4 weeks after delivery.^{5,7–16} We describe a case of eclampsia occurring 16 days after parturition and compare the clinical, radiologic, and electroencephalographic findings with other published reports. Theoretical and proved pathophysiologic derangements of this serious pregnancy-related disorder are also reviewed.

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Methods

We describe a case of delayed postpartum eclampsia and illustrate the unique features of the syndrome. An extensive literature review highlights

the pathogenesis, controversies, and dilemmas surrounding this enigmatic hypertensive disorder.

Case Report

A 39-year-old African-American woman, gravida 5, para 3, abortus 2, weighing 256 pounds (116 kg), had a healthy 7-pound 7-ounce (3.5-kg) female infant delivered vaginally by her primary care physician at 40 weeks' gestation as determined by clinical dating and serial sonograms. Her blood pressure, which averaged 130/80 mm Hg during gestation, did not require antihypertensive medication and was not elevated during labor, delivery, or the 3-day postpartum hospital stay. Edema and proteinuria were not detected at any of the 12 prenatal visits. For 8 years before this pregnancy, the patient had mild hypertension, controlled at 120 to 130 mm Hg systolic and 80 to 86 mm Hg diastolic with the angiotensin-converting enzyme inhibitor enalapril 2.5 mg/d. This medication was discontinued immediately when pregnancy was diagnosed at 4 weeks of gestation because of proved risks for fetal renal failure and growth retardation. Prenatal visit findings were normal, as were all obstetric screening tests and periodic assessments of renal function. Her uric acid level at 38 weeks was 5.2 mg/dL. The patient had two full-term pregnancies that were uneventful and without hypertensive complications 12 and 16 years earlier. She had two spontaneous first-trimester miscarriages preceding this pregnancy. She reported no history of drug abuse, seizure disorder, or other neurologic disease.

On the 16th postpartum day while at home breast-feeding her infant, the patient suddenly developed a severe occipital headache, two episodes of emesis, and double vision. Within 5 minutes she experienced abrupt rhythmic abdominal muscle jerking, which progressed to a 2-minute generalized tonic-clonic seizure and loss of consciousness, as witnessed by her 2 teenaged children. When evaluated by emergency medical personnel 10 minutes later, she was drowsy, confused, and amnesic. Her blood pressure was 190/110 mm Hg, and she was incontinent of urine. She was transported by ambulance to the Medical College of Georgia Hospital, where she regained full consciousness and complained of persistent headache and diplopia (Figure 1). Her blood pressure on arrival was 195/105 mm Hg.



Figure 1. A 39-year-old hypertensive, hyperreflexic woman with headache and diplopia after an eclamptic seizure.

Initially her neurologic examination showed only hyperreflexia. There was no cognitive dysfunction, papilledema, hemiparesis, clonus, or nuchal rigidity. During an examination by 2 physicians in the emergency department, the patient had a second episode of abrupt abdominal jerking, followed immediately by a 2-minute tonic-clonic seizure involving all extremities, associated with interruption of consciousness, blood pressure of 180/120 mm Hg, and urinary incontinence. The seizure, which stopped after an administration of diazepam 5 mg intravenously, was followed by antegrade amnesia and fatigue. Laboratory data documented proteinuria (3+ [300 mg/dL]) and an elevated uric acid level of 9 mg/dL. Peripheral blood smear, hemoglobin level, platelet count, fibrinogen, clotting studies, chemistry panels, arterial blood gas measurement, and tests of hepatic and renal function were normal. An obstetrician and a neurologist who were promptly consulted documented hyperreflexia and diplopia. After careful review of all data, both consultants considered eclampsia unlikely because 16 days had elapsed since delivery. They recommended that epilepsy, intracranial hemorrhage or infarction, and brain tumor or abscess be considered as more likely

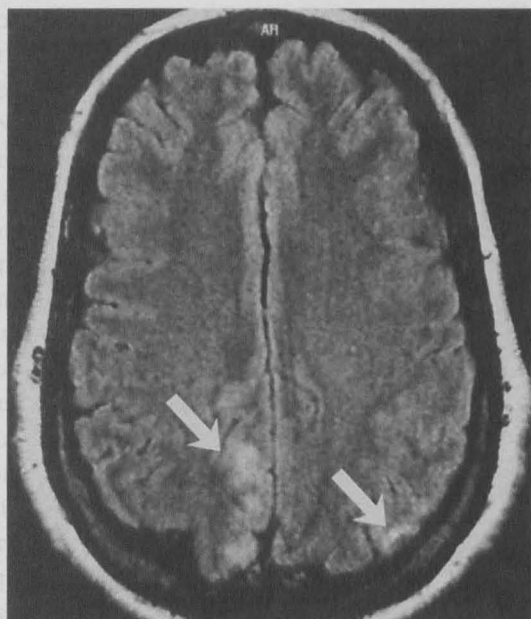


Figure 2. Magnetic resonance image 24 hours after eclamptic seizures. Hyperintense T2-weighted gray-matter lesions in right and left parietal lobes (arrows).



Figure 3. Magnetic resonance image 24 hours after eclamptic seizures. Hyperintense T2-weighted gray-matter lesion in left parieto-occipital lateral cortex (arrow).

causes for abrupt seizure activity in a patient 16 days postpartum.

An unenhanced computerized tomographic (CT) scan performed 2 hours after the second seizure was without evidence of intracranial lesions. The patient was monitored in the intensive care unit and treated presumptively for eclampsia with intravenous magnesium sulfate in a 4-g bolus, then by 2 to 3 g/hr, to maintain serum magnesium concentrations of 4 to 6 mg/dL. Magnetic resonance imaging (MRI) obtained 24 hours after the second seizure showed six abnormal foci of increased signal in axial fluid-attenuated T2 images (FLAIR technique). Lesions were asymmetric, bilateral, and in a predominantly gray-matter distribution in posterior parietal (Figure 2) and occipital (Figure 3) lobes, with a single focus in the left medial temporal lobe (not shown). All lesions were located in the vascular distribution of the posterior cerebral arteries. Neuroradiologic interpretation was consistent with a pattern of cytotoxic edema as the basis for the observed lesions. Magnetic resonance venography (MRV) showed no evidence of central vein or dorsal sinus thrombosis (Figure 4).

During the subsequent 4-day hospitalization, there was no seizure recurrence. Her diplopia and proteinuria resolved within 24 hours, and her head-

ache within 48 hours. Her blood pressure remained 120 to 140 mm Hg systolic and 80 to 100 mm Hg diastolic without antihypertensive medication. A pelvic sonogram indicated no retained products of conception. An electroencephalogram (EEG), obtained 48 hours after the last seizure, was normal. Given these composite data, the consulting obstetrician and neurologist were now impressed that



Figure 4. Normal findings on a magnetic resonance venogram.

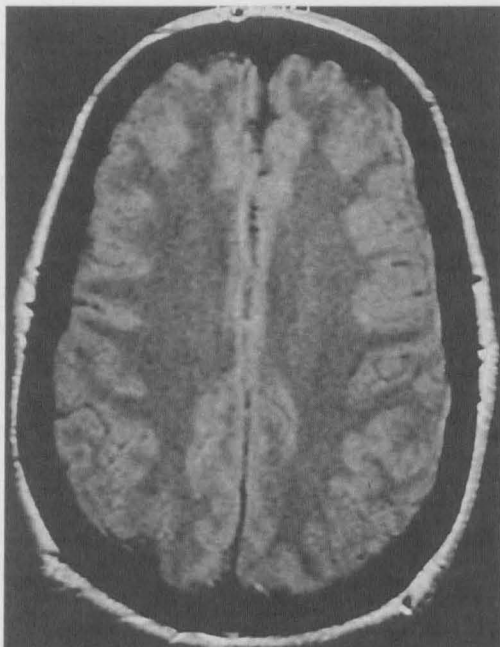


Figure 5. Magnetic resonance image of the parietal lobes 9 days after eclamptic seizures. All lesions have resolved.

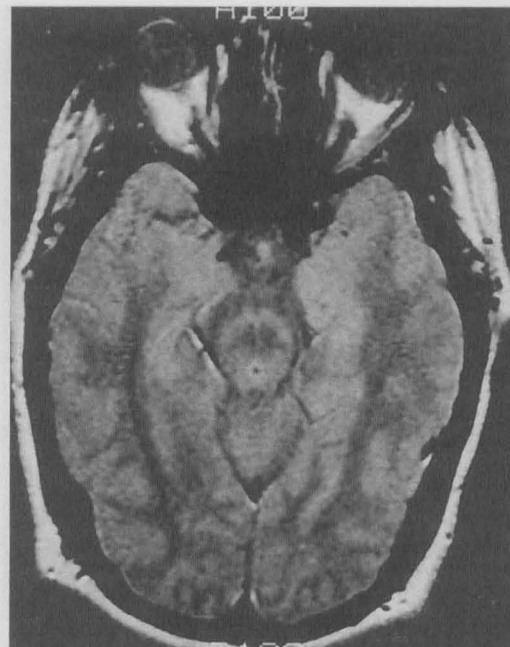


Figure 6. Magnetic resonance image of the left parieto-occipital lateral cortex 9 days after eclamptic seizures. The lesion has resolved.

the clinical, CT, MRI, and EEG findings were highly suggestive of eclampsia despite the delayed onset. Magnesium sulfate administration was stopped after 48 hours, and no further anticonvulsant therapy was prescribed. The patient was discharged on the 4th hospital day, asymptomatic; her blood pressure was 140/80 mm Hg, and she had normal findings on a neurologic examination. She was instructed to resume taking enalapril 2.5 mg/d to prevent blood pressure elevations at home.

An MRI 9 days after the repetitive seizure activity showed resolution of all previously identified cortical cytotoxic lesions (Figures 5 and 6), with no visible evidence for residual vascular or parenchymal CNS sequelae. Uric acid levels decreased to 6.5 mg/dL.

At 6 weeks postpartum, the patient felt well. Her blood pressure was 120/90 mm Hg, and exercise-induced weight loss was 4 kg. On subsequent follow-up examinations for a 24-month period, she has had adequate blood pressure control with enalapril 2.5 mg/d and no recurrence of neurologic symptoms or proteinuria.

Discussion

Eclampsia is an enigmatic syndrome, both in its pathogenesis and in its temporal relation to gesta-

tion. This pregnancy-related hypertensive encephalopathy and has been attributed to intense cerebral vasospasm with breakthrough of autoregulation of intracranial arterial vasculature. Subsequent cerebral edema results from microischemic damage to the blood-brain barrier. Proposed triggers include endothelial damage,^{17,18} imbalance between vasoconstrictive and vasodilatory prostaglandins, sympathetic overactivity,¹⁹ and abnormal placental function.²⁰

Theoretically two derangements have been proposed to underlie the development of focal cerebral edema: vasospasm and forced dilation.²¹ The vasoconstrictive theory suggests that cerebral overregulation leads to vasoconstrictive responses of arterial vasculature after explosive increases in blood pressure. Vasospasm, in turn, produces local ischemia, resulting in protein extravasation, arteriolar necrosis, disruption of the blood-brain barrier, and microischemic CNS lesions. In contrast, the forced dilation theory suggests that during sustained and extreme elevation of blood pressure, autoregulatory vasoconstrictive forces are overwhelmed, forced vasodilation ensues, tight junctions are opened, and the blood-brain barrier is disrupted through endothelial damage. Deregulated vascular injury to blood-brain barrier endothelium leads to edema.

protein extravasation, and fibrinoid necrosis.^{22,23} Either of these proposed vasogenic events could provide the anatomic substrate for widespread CNS insults with resultant premonitory symptoms and convulsions that characterize eclampsia.

Angiographic studies of eclamptic humans²⁴⁻²⁸ and direct arterial visualization in experimentally hypertensive rats,²⁹ cats,³⁰ and monkey³¹ have confirmed the vasospastic derangements underlying states of hypertensive encephalopathy and implicated such processes in the development of cerebral edema. Autopsy studies of fatal eclampsia and preeclampsia^{22,32-36} have documented pathologic lesions, such as fibrinoid necrosis, perivascular microinfarction or microhemorrhage, and focal cerebral edema, attributed to microischemic and hypoxic damage to the blood-brain barrier. In some cases, frank subarachnoid or intraparenchymal hemorrhage has been observed, often correlating with greater severity of neurologic morbidity before death. Such evidence suggests that the pathologic process in eclampsia is indeed a CNS vasculopathy related to acute hypertensive states leading to loss of vessel wall autoregulation and eventual endothelial dysfunction. Identical microvascular processes have been documented in conditions of hypertensive dysfunction involving other human organs, such as kidney, lung, liver, and placenta.^{21-22,32,33,37} Predominant involvement of posterior cortical regions of cerebral white matter in eclampsia has been documented by several authors,^{14,38-46} suggesting selective or intensified vasospasm of posterior cerebral arterial vasculature or regional variations in sympathetic innervation.

Although etiologic features of eclampsia might be poorly understood, recognition is clinically obvious and highly alarming once seizures occur. Most common in pregnancy near term, but with onset generally between 20 weeks of gestation and the first 48 hours postpartum, eclampsia is characterized by volatile hypertension, proteinuria, edema, and seizures. Convulsions commonly occur after the onset of one or more isolated prodromal symptoms (headache, visual change, epigastric pain, facial or hand edema, or sudden weight gain), or they can accompany the emergence of overt preeclampsia in women receiving regular obstetric care. Yet there can be absolutely no premonitory warning symptomatology or documented blood pressure increase until just before convulsions, as was the instance in our case. In fact, Sibai⁴⁷ ob-

served that 19% to 32% of eclamptic patients analyzed in a recent series were without antecedent indicators of hypertensive pregnancy complications.

Because delivery is the definitive treatment for severe preeclampsia and antepartum eclampsia, the diagnosis of postpartum eclampsia with seizure onset delayed for days to weeks after parturition is understandably regarded with skepticism. Clinicians must be aware that sudden blood pressure surges, headache, visual and mental status changes, fluid retention, and hyperreflexia are worrisome disturbances heralding late postpartum eclampsia and must be thoroughly evaluated in patients who have recently given birth.⁴⁸ Elevated serum uric acid values, clotting abnormalities, decreased creatinine clearance, and HELLP (hemolysis, elevated liver function tests, and low platelet count) syndrome can further support the diagnosis.⁴⁹ Finally, EEG findings after eclamptic seizures can show diffuse or focal slowing, or can, as in our patient, be entirely normal.⁵⁰

Timely radiologic investigation is essential in distinguishing postpartum eclampsia from other catastrophic causes of seizures, such as intracranial hemorrhage, cerebral infarction, brain tumor, or CNS abscess. CT, the most readily available modality, is the preferred technique for excluding acute hemorrhage or space-occupying lesions. In eclampsia, CT scans might show nonenhancing, low-attenuation foci, primarily in the occipital lobes (posterior cerebral artery circulation), or in terminal interface regions supplied by major arteries (watershed zones) of the brain. Such lesions on CT scans are radiologically suggestive of cerebral edema. Yet in many cases, CT imaging in eclampsia is completely normal.^{38,44,51-55} MRIs are more definitive than CT scans in showing microischemic injury patterns in the parieto-occipital lobes, occasionally with involvement of basal ganglia.^{56,57} Heightened signal intensity of T2-weighted images on an MRI represents increased water content in visualized radiologic lesions, an indication of focal brain edema or microischemia. All the above MRI findings can be undetectable by currently available CT methods, as occurred in our case. Furthermore, microischemic lesions observed by either CT or MRI in eclamptic patients typically resolve within days to weeks, as reflected in our case, again suggesting that the visualized lesions are due to revers-

ible vasogenic or cytotoxic edema, not parenchymal necrosis.

We believe that our patient was truly eclamptic despite her delayed symptoms at 16 days postpartum. The history of chronic hypertension, older age, and African-American race placed her at higher risk for preeclampsia or eclampsia.⁵⁸ Alternative causes of hypertensive encephalopathy (epilepsy, stroke, hemorrhage, abscess, tumor) were aggressively evaluated and excluded. MRI confirmation of reversible cortical lesions in the territory of the posterior cerebral artery is consistent with published series of eclampsia from other institutions, as previously cited. Clinical, laboratory, and radiologic abnormalities in our case were transient and fully reversible within hours to days, as would be expected in a disorder of CNS vascular autoregulation such as eclampsia. The lesions observed on the MRI in a parieto-occipital distribution would seem to account for the headache and diplopia we observed, whereas lesions in the temporal lobe involving basal ganglia and deeper white matter probably accounted for the mental status changes of postictal lethargy, confusion, and amnesia documented in our case.

The time limit of 48 hours after delivery no longer applies to postpartum eclampsia, because onset of convulsions might be delayed for 3 to 4 weeks, as reported by others.^{5,7-16} Women who are pregnant or have recently given birth and who are experiencing headache, visual blurring, hypertension, proteinuria, and edema require urgent evaluation by physicians qualified in obstetric care. Eclampsia can occur without warning, with trivial blood pressure elevations, and in seemingly uncomplicated antepartum, intrapartum, or postpartum patients. Once seizures occur, prompt obstetric and neurologic consultation is essential. CT scanning is a rapid and appropriate initial imaging tool to exclude disastrous but potentially treatable intracranial hemorrhage or space-occupying lesions. MRI is superior to CT in showing gray-white matter junction lesions that are considered diagnostic of eclampsia by some authors.^{14,59}

Until the differential diagnosis is clarified, primary care clinicians should presumptively manage postpartum eclampsia syndromes with intravenous benzodiazepines or phenytoin for immediate termination of seizure activity,^{60,61} correct acidosis or hypoxia, if present, treat with intravenous magnesium sulfate for 48 to 72 hours to prevent further

seizures,^{62,63} and optimize blood pressure control at 140/90 mm Hg or less with intravenous hydralazine or labetalol, if required.² Referral to centers with experience in hypertensive disorders of pregnancy might be judicious as well. Eclampsia (Greek for "lightning") can strike with sudden, unanticipated violence, as illustrated in our case. Because eclampsia can have a wide spectrum of clinical manifestations arising days to weeks after parturition, physicians in a broad variety of specialties must be vigilant for this explosive vasculopathy.⁴⁸

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