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LATE POSTPARTUM ECLAMPSIA WITH POSTPARTUM ANGIOPATHY: AN UNCOMMON DIAGNOSIS IN THE EMERGENCY DEPARTMENT

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☐ Abstract—Background: Late postpartum eclampsia is defined as occurrence of eclampsia >48 h after delivery and is a rare clinical entity. The delayed onset and nonspecific symptoms at presentation make this entity a challenge to diagnose in patients presenting to the emergency department (ED); however, early recognition and timely interventions are the keys to reducing morbidity and mortality in patients with late postpartum eclampsia. Case Report: A 28-year-old woman presented to our ED with a chief complaint of headache of 4 days duration, 8 days after an uncomplicated, normal vaginal delivery. Her past medical history was unremarkable and her entire pregnancy was without medical incident. The patient's examination was within normal limits other than a blood pressure of 152/ 111 mm Hg and pulse of 54 beats/min. Given her undifferentiated headache and the possibility of preeclampsia, the patient was treated with magnesium sulfate, which was subsequently stopped due to worsening bradycardia. Hydralazine was administered for blood pressure control. Three hours after the magnesium was stopped, the patient reported blurry vision, which was immediately followed by a generalized tonic-clonic seizure. After the seizure, lorazepam was given for control of seizures, and the patient was admitted to the medical intensive care unit. The patient was transferred to the postpartum floor 6 days later in stable condition and without any further seizure activity. Why Should an Emergency Physician Be Aware of This?: Patients with late postpartum eclampsia are infrequently encountered in the ED due to the rarity of this condition. Increased awareness of this entity among emergency physicians will

lead to early interventions, which are crucial in decreasing morbidity and mortality in these patients. $\,\,^{\odot}$ 2015 Elsevier Inc.

☐ Keywords—headache; late postpartum eclampsia; seizure

INTRODUCTION

Eclampsia is defined as a complication of pregnancy after 20 weeks gestation and involves seizure activity superimposed on preeclampsia, which is characterized by elevated blood pressure and one or more of a host of complications that include new-onset proteinuria, low platelets, abnormal liver function tests, renal impairment, pulmonary edema, and neurological or visual symptoms (1). It can present during the antepartum, intrapartum, or postpartum periods. Most patients with postpartum eclampsia present within 24 to 48 h after delivery, whereas late postpartum eclampsia is defined as the occurrence of eclampsia >2 days after delivery. The incidence of eclampsia in developed countries is 1.6-10 cases per 10,000 deliveries, and 11-44% of cases occur in the postpartum period (2,3). Early diagnosis and timely management are crucial steps in reducing the morbidity and mortality associated with this disorder; if left untreated, eclampsia can lead to maternal death due to cerebral edema and seizure activity.

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CASE PRESENTATION

A 28-year-old woman, gravid 1 para 1, presented to the ED on postpartum day 8 with complaints of headache for 4 days duration. Four days prior to arrival, she had a visit to the emergency department (ED) of another hospital after the onset of the same headache, at which time she had a normal neurological examination and computed tomography (CT) of the head. At that time, the patient was discharged home with analgesics. Eight days prior to arrival (and 4 days prior to the first ED visit), the patient had vaginally delivered a full-term baby with epidural analgesia after an uncomplicated pregnancy. The patient denied any past medical history or any obstetrical complications, including seizure disorder, migraines, hypertension, and chronic headaches. The headache was described as intermittent, sharp, and was localized to the bilateral frontal and temporal regions with a pain score of 10/10. The headache was exacerbated when supine, and improved in the upright position. She denied any fever, chills, or neck stiffness. The patient's blood pressure during the pregnancy and the delivery was normal, and at the time of discharge was 90–110/50–70.

On physical examination, the patient was in mild distress secondary to the headache. Her initial vital signs in the ED revealed a temperature of 36.6°C (97.8°F), blood pressure of 152/111 mm Hg, pulse of 54 beats/min, respiratory rate of 18 breaths/min, and oxygen saturation of 100% while breathing ambient air. There was no evidence of head trauma, and the neck was supple. Examination of the heart, lungs, and abdomen were unremarkable. The patient's neurological examination, including deep tendon reflexes, was normal, as was the fundoscopic examination.

Laboratory studies including complete blood count, basic metabolic panel (serum sodium, potassium, chloride, bicarbonate, blood urea nitrogen, creatinine, glucose, and calcium levels), and liver function tests were all within normal limits. There was no proteinuria. As the patient had an uncomplicated pregnancy with no history of gestational hypertension or preeclampsia in the antepartum, intrapartum, or immediate postpartum periods, and with a reported recent normal head CT scan, venous sinus thrombosis was a diagnostic consideration. Therefore, magnetic resonance venography of the brain was obtained, which showed evidence of intracranial hypotension but no venous sinus thrombosis. The patient was initially started on magnesium sulfate, but this medication was discontinued after 2 h due to bradycardia of 39 beats/min; however, the blood pressure remained at 140/100 mm Hg, for which hydralazine was given.

Consultation was obtained from both Obstetrics and Neurology. The patient was evaluated by the Obstetrics team, which recommended intensive care monitoring of the blood pressure due to possible preeclampsia. The neurologist recommended a magnetic resonance imaging (MRI) study of the brain, valproate for seizure prophylaxis, and intravenous lorazepam as needed for seizure activity, given the bradycardia during magnesium administration. After the consultations were obtained, the patient developed blurry vision followed by a generalized tonic-clonic seizure 3 h after stopping the magnesium. Lorazepam was administered with subsequent resolution of the seizure activity. The patient was admitted to the medical intensive care unit. A CT scan of the head was normal. MRI of the brain showed posterior reversible encephalopathy syndrome (PRES) (Figure 1). A magnetic resonance angiogram of the brain demonstrated evidence of postpartum angiopathy (Figure 2). The patient had no further complications during a 6-day course in the Intensive Care Unit and was transferred to the postpartum unit in stable condition.

DISCUSSION

Eclampsia occurs in 38–53% of patients during the antepartum, 18–36% of patients in intrapartum, and 11–44% of patients in the postpartum period (3). Postpartum eclamptic seizures usually occur within 48 h of delivery, although they have been reported up to 23 days postpartum (4). It is important to rapidly diagnose late postpartum eclampsia to initiate appropriate interventions and hopefully prevent further complications including coagulopathy, renal failure, pulmonary edema, hepatocellular damage, cerebral ischemia, intracerebral hemorrhage, and even death, in rare cases (3,5).

The revised definition of preeclampsia by the American College of Obstetricians and Gynecologists (ACOG) Task Force Report is described in Table 1 (1). Eclampsia is defined as seizure activity in conjunction with preeclampsia. Our patient initially did not meet the criteria of preeclampsia, as she had a normal platelet count, creatinine and transaminase levels, and an absence of proteinuria. As per the most recent ACOG guidelines for the diagnosis of preeclampsia, the presence of hypertension along with neurological symptoms such as headache in a pregnant or recently pregnant patient, even if the patient does not have proteinuria, should raise suspicion for preeclampsia. Other systemic findings have been included in the definition of preeclampsia, as maternal and fetal outcomes cannot be predicted by the degree of proteinuria. Awareness of postpartum preeclampsia has been emphasized, along with minimizing use of nonsteroidal antiinflammatory drugs in patients with elevated blood pressure in the postpartum period.

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