

POST-DELIVERY COMPLEX PARTIAL SEIZURE MIMICKING ECLAMPSIA

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Partial seizures are divided into simple, complex, and those that evolve into secondary generalized seizures. The difference between simple and complex seizures is that patients retain awareness during simple partial seizures, but lose awareness during complex partial seizures. Eclampsia is the most severe form of pregnancy-induced hypertension, and can lead to seizures. Eclampsia occurs in about one in 1,600 pregnancies and generally develops towards the end of pregnancy.

A 39-year-old woman, gravida 2, para 1, came to our delivery room with labor pains at 37 weeks of gestation. Fetal monitoring showed a non-reactive fetus with a variable and poor fetal heart beat. Ultrasound showed severe intrauterine fetal growth retardation with oligohydramnios. The patient had suffered from high blood pressure and the fetus had shown intrauterine fetal growth retardation since 32 weeks of gestation. The biophysical profile score of the fetus was 4 and the umbilical cord artery systolic/diastolic ratio was 3.3. The patient was given two doses of 15 mg dexamethasone (Decadron) by intramuscular injection every 24 hours to enhance fetal lung maturation at 33 weeks of gestation. Her blood pressure was 182/105 mmHg, urinary protein was 3+, and she suffered from generalized edema with swelling of the arms and face, accompanied by headache, changes in vision (blurred vision, double vision), dizziness, nausea, and vomiting for 1–2 days before admission. A cesarean delivery was performed immediately under the impression of severe preeclampsia with fetal distress, and the infant was severely meconium-stained. The baby weighed 1,650 g, with Apgar scores of 5–7, and was sent to the neonatal intensive care unit for further care.

The mother experienced a repeat episode of side-to-side head swinging with drowsy consciousness, but was still able to respond to commands; the episode occurred 2 hours after delivery and lasted for less than 1 minute.

Magnesium sulfate (MgSO_4 , 4 g loading dose intravenously and 2 g/hr intravenous infusion for 24 hours), diazepam and an antiepileptic agent (valproic acid) were given. The patient underwent electroencephalography (EEG) after the seizure stopped, and the results were normal, with no evidence of cortical dysfunction. The repeated partial seizures (> 10) subsided after 24 hours of treatment. Oral carbamazepine was prescribed for maintenance treatment. Her general condition improved and she was discharged on the sixth day after delivery. She was diagnosed with complex partial seizures with a psychotic component, which is a rare condition.

Eclampsia is defined as the development of convulsions or unexplained coma during pregnancy or postpartum in patients with signs and symptoms of preeclampsia. The reported incidence of eclampsia in Western countries ranges from 1/2,000 to 1/3,448 pregnancies [1–4]. The reported incidence is usually higher in tertiary referral centers, multifetal gestations, and populations with no prenatal care [5]. Eclampsia is the final and most severe phase of preeclampsia, and occurs when preeclampsia remains untreated. In addition to the previously mentioned signs of preeclampsia, women with eclampsia also often experience seizures. Eclampsia can cause coma and even maternal and fetal death, and can occur before, during, or after childbirth. Other possible causes of convulsions or seizures during labor, such as epilepsy, encephalitis, meningitis, cerebral tumor, cysticercosis, or rupture of a cerebral aneurysm, should be identified before a final diagnosis of eclampsia is made.

The pathogenesis of convulsions caused by eclampsia continues to be the subject of extensive investigation and speculation. Several theories and pathologic mechanisms have been implicated as possible etiologic factors, but none has yet been conclusively proven. It is not clear if the pathologic features of eclampsia are a cause or an effect of the convulsions [6].

Eclamptic convulsions are a life-threatening emergency and require proper care in order to minimize morbidity and mortality. An eclamptic convulsion is frightening to observe; the patient's face initially becomes distorted and the eyes protrude, followed by a congested facial expression. Foam often exudes from



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the mouth. The woman usually bites her tongue unless it is protected. Respirations are absent throughout the seizure. The convulsion, which can be divided into two phases, typically continues for 60–75 seconds. The first phase lasts for 15–20 seconds and begins with facial twitching, proceeding to the body becoming rigid with generalized muscular contractions. The second phase lasts for approximately 60 seconds and consists of the muscles of the body alternately contracting and relaxing in rapid succession. This phase begins with the muscles of the jaw, and rapidly involves the eyelids, other facial muscles, and then all the muscles of the body. Coma follows the convulsion, and the woman usually remembers nothing of the recent events. If she has repeated convulsions, some degree of consciousness returns after each convulsion. Rapid and deep respirations usually begin as soon as the convulsions end. Maintenance of oxygenation is usually not a problem after a single convulsion, and the risk of aspiration is low in the well-managed patient.

Complex partial seizures, by definition, include impaired awareness. Patients appear to be “out of touch”, “out of it”, or “staring into space” during these seizures. Complex partial seizures cause impaired consciousness and arise from a single brain region. Impaired consciousness implies decreased responsiveness and awareness of self and surroundings. During a complex partial seizure, the patient may not communicate, respond to commands, or remember events that have occurred. Consciousness might not be completely impaired. “Complex” symptoms called automatisms also occur, which consist of involuntary but coordinated movements that tend to be purposeless and repetitive. Common automatisms include lip smacking, chewing, fidgeting, walking, repeated scratching of the head, or searching for an object; some people may even undress.

Complex partial seizures of the temporal lobe often begin with a motionless stare followed by simple oral or motor automatisms. In contrast, frontal-lobe seizures often begin with vigorous motor automatisms or stereotypic clonic or tonic activity. Extratemporal-lobe seizures may spread quickly to the frontal lobe and produce motor behaviors similar to those associated with complex partial seizures of the frontal lobe. Tonic and dystonic arm posturing can occur in the arm contralateral to the seizure focus. Sustained head or eye turning contralateral to the seizure focus can occur immediately before, or simultaneously with, clonic or tonic activity elsewhere. This activity often lasts from 30 seconds to 2 minutes. Longer seizures can occur, particularly when the seizures become generalized convulsions. Complex partial status epilepticus can also occur, with prolonged episodes of waxing and waning of consciousness.

The differential diagnosis of eclampsia and complex partial seizures, including the presenting symptoms, clinical findings, and many of the laboratory findings, overlap with a number of medical and surgical conditions [7,8]. Although eclampsia is the most common cause of convulsions developing in association with hypertension or proteinuria during pregnancy or immediately postpartum, other etiologies producing convulsions in pregnancy or postpartum can occasionally mimic eclampsia [6]. These diagnoses are particularly important in the presence of focal neurologic deficits, prolonged coma, or in the presence of atypical eclampsia. In addition, gestational hypertension or preeclampsia may develop in some patients in association with connective tissue disease, thrombophilia, seizure disorder, or hypertensive encephalopathy, further contributing to the diagnostic difficulty [7]. Efforts should therefore be made to make an accurate diagnosis, given that the management strategies for these conditions differ.

Several clinical symptoms are potentially helpful in establishing the diagnosis of eclampsia, including persistent occipital or frontal headaches, blurred vision, photophobia, epigastric and/or right upper quadrant pain, and altered mental status. Patients have at least one of these symptoms in 59–75% of cases [2,5,9]. Headaches are reported by 50–75% of patients and visual changes are reported in 19–32%. These symptoms can occur before or after the onset of a convulsion [6].

Several neurodiagnostic tests such as EEG, cerebral Doppler velocimetry, magnetic resonance imaging, and cerebral angiography (both traditional and magnetic resonance imaging angiography) have been used on women with eclampsia. EEG is acutely abnormal in the majority of eclamptic patients; however, these abnormalities are not pathognomonic for eclampsia. In addition, the abnormal EEG findings are not affected by the use of MgSO_4 [10].

The treatment of eclampsia or any other seizure before, during, or after delivery is similar. MgSO_4 and intravenous antiepileptic drug infusion are the first choices. Organic lesions should be ruled out by EEG and brain computed tomography if medication fails after 24 hours. MgSO_4 should be discontinued 24 hours after delivery to prevent overdose intoxication effects, such as respiratory deterioration. Because eclampsia is frightening, there is a tendency to try and stop the convulsion. However, drugs such as diazepam (valium) should not be given in an attempt to stop or shorten the convulsion, especially if the patient does not have an intravenous line in place and if nobody skilled in intubation is available. If diazepam is used, no more than 5 mg should be given over a 60-second period. Rapid administration of diazepam may lead to apnea and/or cardiac arrest.

In conclusion, the current patient was diagnosed with complex partial seizure, rather than eclampsia. As eclampsia always presenting as convulsions or unexplained coma during pregnancy, and in the postpartum period only develops in patients with signs and symptoms of preeclampsia, including persistent occipital or frontal headaches, blurred vision, photophobia, epigastric and/or right upper quadrant pain, and altered mental status. The EEG is acutely abnormal in the majority of eclamptic patients, while complex partial seizures may not completely impair consciousness, and some patients may be able to make simple verbal responses and follow simple commands.

References

1. Saftlas AF, Oldon DR, Franks AC, Atrash HK, Pokras R. Epidemiology of preeclampsia and eclampsia in the United States. 1979–1986. *Am J Obstet Gynecol* 1990;163:460–5.
2. Douglas KA, Redman CW. Eclampsia in the United Kingdom. *BMJ* 1994;309:1395.
3. Lee W, O'Connell CM, Baskett TF. Maternal and perinatal outcomes of eclampsia: Nova Scotia 1981–2000. *J Obstet Gynaecol Can* 2004;26:119–23.
4. Rugard O, Carling MS, Berg G. Eclampsia at a tertiary hospital 1973–99. *Acta Obstet Gynecol Scand* 2004;83:240–5.
5. Katz VL, Farmer R, Kuller J. Preeclampsia into eclampsia: toward a new paradigm. *Am J Obstet Gynecol* 2000;182:1389–96.
6. Sibai BM. Diagnosis, prevention, and management of eclampsia. *Obstet Gynecol* 2005;105:402–10.
7. Witlin AG, Friedman SA, Egerman RS, Frangieh AY, Sibai BM. Cerebrovascular disorders complicating pregnancy – beyond eclampsia. *Am J Obstet Gynecol* 1997;176:1139–45; discussion 1145–8.
8. Shearer VE, Harish SJ, Cunningham FG. Puerperal seizures after post-dural puncture headache. *Obstet Gynecol* 1995;85:255–60.
9. Chames MC, Livingston JC, Ivester TS, Barton JR, Sibai BM. Late postpartum eclampsia: A preventable disease? *Am J Obstet Gynecol* 2002;186:1174–7.
10. Barton JR, Sibai BM. Cerebral pathology in eclampsia. In: Sibai BM, ed. *Clinics Perinatology*, Vol. 18. Philadelphia: WB Saunders Company, 1991;891–910.