



Research Article

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Post-partum status epilepticus in a patient with no prodromal sign of preeclampsia undergoing cesarean delivery

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ABSTRACT

Preeclampsia and eclampsia are hypertensive disorders that can complicate pregnancies. In this manuscript we present a rare presentation of preeclampsia in a 36-year-old patient with no prodromal hypertension or proteinuria which turned into eclampsia with initial presentation of post-partum status epilepticus. The patient was treated with Dilantin, diazepam and magnesium sulfate. Further neurological imagings including magnetic resonance imaging, magnetic resonance venogram, CT scan of the brain and electro encephalography were all normal. Although these events are difficult to anticipate, as anesthesiologists, we have to be prepared to manage them.

Keywords: Eclampsia, Convulsion, Status epilepticus, Post-partum, Avocado, Osmotic, Chemical features, Ultrasound

INTRODUCTION

Expect everything so that nothing comes unexpected! As anesthesiologists, we encounter unusual situations on a daily basis. However, some operations are so commonly performed; it is hard to believe any unanticipated event would complicate them.

Preeclampsia is clinically defined by hypertension and proteinuria, with or without pathologic edema. However, patients can lack any of these signs and symptoms and still have preeclampsia (1).

Eclampsia is considered a complication of severe preeclampsia and is defined as new onset of grand mal seizure activity and/or unexplained coma during pregnancy (2). Unless proved otherwise, seizures happening during the peripartum period are attributed to eclampsia. This disease usually develops in the third trimester and less commonly in postpartum period, and has an incidence of 0.1 to 0.4% in the United States (3).

In this paper we present a case of emergency repeat cesarean section in a patient with polyhydramnios complicated with status epilepticus after delivery in the operating room.

Case Summary

A 36-year-old lady (gravida 6, abortion 2, dead 2, and live1) with 155 cm height and 80 Kg weight attended a university affiliated hospital for an emergency repeat cesarean section at 27 weeks of gestation due to polyhydramnios and premature contractions. The anesthesiology resident performed the pre-operation assessment before the surgery. She had developed gestational diabetes in her recent pregnancy since 20 days prior to admission which was controlled with dietary alterations. Her current medication included the occasional use of acetaminophen

codeine for headaches and prenatal multivitamins. Her previous surgical history included previous cesarean section under general anesthesia due to polyhydramnios (hydrops fetalis) without any complications two years ago. No definite cause was found for her previous abortions and the rheumatology test results including the antiphospholipid antibody, antinuclear antibody and anti-double strand DNA titers were within normal limits.

The patient had received prenatal care all through her pregnancy. Her pre-operation hemoglobin and hematocrit were 10.9 g/dL and 32.2% respectively. White blood cell count was 9700/mL, and platelet count was 234000/mL. Pre-operation physical exam was in normal range with a cuff blood pressure of 115/75 mmHg, heart rate of 100/min, respiratory rate of 18/min, and O₂ saturation of 99%. She had no history of proteinuria or hypertension. Airway assessment revealed a Mallampati class 3.

The patient consented to spinal anesthesia. Pre-anesthetic medications included gentamicin (100 mg IV), clindamycin (600 mg IV), and metoclopramide (10 mg IV). The patient was hydrated with 500 ml of ringer lactate solution before initiating the anesthesia. Spinal anesthesia was performed with the patient in sitting position. Anesthetic injection site was prepared with betadine and sterile draping. After injection of 1% lidocaine into the skin and subcutaneous tissue, a 25-gauge Whitacre spinal needle was inserted into the L4-5 intervertebral space. Eight mg bupivacaine in dextrose and 5 mg pethidine were injected into the subarachnoid space. The patient was then positioned supine with uterine displaced left sided. Sensory level of T4 was confirmed before making the incision. The patient was anxious throughout the procedure.

After rupture of membrane, 2 liters of amniotic fluid were suctioned and a male hydrops infant was delivered with 1 minute Apgar score of 8. During the delivery, the patient complained of feeling discomfort and pressure in her abdomen; therefore, 10mg ketamin was given to her intravenously. Twenty units of oxytocin were added to ringer lactate solution and infused.

Four to five minutes after delivery, the patient had an onset of grand mal seizure. Administration of 100% oxygen via face mask was initiated and midazolam (1.5 mg IV) was given. With application of sellick's maneuver, the patient promptly received thiopental (350 mg IV) and succinylcholine (140 mg IV), and trachea was intubated with a 7-mm endotracheal tube on the first attempt. Breathing sounds were clear and the patient's blood glucose level was 148 mg/dL. The patient's heart rate subsequently increased to approximately 140/min, and her blood pressure and O₂ saturation dropped to 65/47 mm Hg and 75% respectively. End-tidal CO₂ reached 15 mmHg and electrocardiographic changes (ST segment depression) appeared.

At this point, ephedrine was administered and the patient was ventilated manually. Gradually the blood pressure, O₂ saturation, and end-tidal CO₂ increased to 110/85 mmHg, 95%, and 32 mmHg respectively. Isoflurane (0.6%) and oxygen (100%) were administered to maintain general anesthesia.

The operation was completed 20 minutes after making incision. At the end of the surgery, the patient had spontaneous breathing with response to painful stimulation. However, she was spastic and after 30 minutes she had another episode of convulsion. Emergency neurologist consult was done and the attending physician ordered the infusion of 1200mg of Dilantin in 500 ml normal saline during 30 minutes, but status was continued and diazepam was administered in three doses of 10 mg.

The intubated patient was then transferred to intensive care unit (ICU) after cessation of seizure; while she did not regain her consciousness in the operating room. Magnesium sulfate was administered to her according to neurologist's order in ICU (4g IV stat).

During admission in the ICU, the patient had another episode of convulsion for which infusion of midazolam and propofol was administered to her for 3 days with gradual tapering.

Further workups in the ICU including, magnetic resonance imaging, magnetic resonance venogram, CT scan of the brain and electroencephalography were all normal. At the end of third day of admission, the patient was extubated and discharged with no sequels.

Results and Discussion

Seizures during pregnancy complicate less than 1% of all gestations; however, they are associated with increased adverse maternal and perinatal outcomes (4). Most seizures during pregnancy occur in women with previous history of this disease. Status epilepticus accounts for only 1-2% of all these seizures and if treated aggressively carries a low risk for morbidity and mortality (5).

Unless proved otherwise, seizures occurring during pregnancy are considered to be caused by eclampsia. Eclampsia is considered a complication of severe preeclampsia. Preeclampsia is a disorder of widespread vascular endothelial malfunction and vasospasm that occurs after 20 weeks' gestation and can present as late as 4-6 weeks postpartum. It is clinically defined by hypertension and proteinuria, with or without pathologic edema. The incidence of preeclampsia in the United States is estimated to range from 2% to 6% in healthy, nulliparous women (6, 7). Eclampsia is commonly defined as new onset of grand mal seizure activity and/or unexplained coma during pregnancy or postpartum in a woman with signs or symptoms of preeclampsia (8). It typically occurs during or after the 20th week of gestation or in the postpartum period. Nonetheless, eclampsia in the absence of hypertension with proteinuria has been demonstrated to occur in 38% of cases reported in the United Kingdom (9). Similarly, hypertension was absent in 16% of cases reviewed in the United States (5).

Most cases of eclampsia present in the third trimester of pregnancy, with about 80% of eclamptic seizures occurring intrapartum or within the first 48 hours following delivery. Rare cases have been reported before 20 weeks' gestation or as late as 23 days' postpartum. Other than early detection of preeclampsia, no reliable test or symptom complex predicts the development of eclampsia. The patient we introduced lacked both proteinuria and hypertension in her perinatal health records, which is a rare silent presentation of preeclampsia.

Seizures rarely occur in patients undergoing anesthesia and there have been few case reports of seizures occurring during cesarean delivery and fewer cases with presentation of status epilepticus (10, 11). The presentation of eclampsia was so atypical in our case that the diagnosis was made only after ruling out all the other causes by extensive neurological imaging and tests. The patient lacked any proteinuria, hypertension, or history of previous preeclampsia or seizures, so there were no way that these events could have been anticipated. Treatment of convulsions was based on the emergency neurologist consultation. Although benzodiazepine could have been used to suppress the patient's anxiety, it was avoided in the first place due to its amnestic effects on the mother for recalling the birth of her child.

Conclusion

Rare atypical presentations of diseases are hard to be anticipated and prevented; however, as physicians we have to be prepared to manage these manifestations. Seizures in their atypical form can occur in patients with no prodromal sign or symptom of preeclampsia and turn a pleasant event in a mother's life into a nightmare.

References

1. Gabbe SG, Niebyl JR, Simpson JL, eds. *Obstetrics: Normal and Problem Pregnancies*. 5th ed. New York, NY: Churchill Livingstone; 2007.
2. Tranquilli AL, Brown MA, Zeeman GG, Dekker G, Sibai BM. The definition of severe and early-onset preeclampsia. Statements from the International Society for the Study of Hypertension in Pregnancy (ISSHP). *Pregnancy Hypertens*. 2013; 3(1):44-7.
3. Turner JA. Diagnosis and management of pre-eclampsia: an update. *Int J Womens Health*. 2010; 2: 327–337.

4. Beach RL, Kaplan PW. Seizures in pregnancy: diagnosis and management. *Int Rev Neurobiol.* 2008; 83:259-71.
5. Hart LA, Sibai BM. Seizures in pregnancy: epilepsy, eclampsia, and stroke. *Semin Perinatol.* 2013; 37(4):207-24.
6. Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. *Obstet Gynecol.* 2003. 102(1):181-92.
7. Vatten LJ, Skjaerven R. Is pre-eclampsia more than one disease?. *BJOG.* 2004; 111(4):298-302.
8. Mattar, F, Sibai BM. Eclampsia. VIII. Risk Factors for maternal morbidity. *Am J Obstet Gynecol.* 1990; 163:1049-55.
9. Douglas KA, Redman CW. Eclampsia in the United Kingdom. *BMJ.* 1994; 309(6966):1395-400.
10. Niroomanesh S, Mirzaie F. Atypical postpartum eclampsia: status epilepticus without preeclamptic prodromi. *Women Birth.* 2008; 21(4):171-3.
11. Dag ZO, Isik Y, Turkel Y, Alpua M, Simsek Y. Atypical eclampsia and postpartum status epilepticus. *The Pan African Medical Journal.* 2015; 20:17.