CASE REPORT

A case of very early onset eclampsia, placental abruption and intrauterine fetal death

Kazutoshi Nakano1,2, Taihei Tsunemi1, Juria Akasaka1, Aiko Shigemitsu1, Katsuhiko Naruse1,3, Hiroshi Kobayashi1

1Department of Obstetrics and Gynecology, Nara Medical University, Nara, Japan, 2Yao Municipal Hospital, Osaka, Japan, 3St. Barnabas’ Hospital, Osaka, Japan

We observed a case of eclampsia suddenly occurring at 22 weeks’ gestation, which progressed to placental abruption and intrauterine fetal death. At 22 weeks’ gestation, the patient’s blood pressure increased, and she had convulsions and lost consciousness. The patient was transferred to the tertiary center and a course of observation was chosen due to the prematurity of the fetus. However, 2 days after onset of eclampsia, the patient experienced acute abdominal pain, and retro-placental hematoma and fetal death were confirmed on ultrasonography. In addition, findings typical of posterior reversible encephalopathy syndrome were demonstrated on MRI. Although labor was induced, the patient’s condition worsened and emergency cesarean section was performed. After the operation, the patient’s clinical course was good and she was discharged without any complications. Eclampsia onset at such an early gestational age is quite rare. We must be cognizant that the outcome of the disease may be poor.

Introduction

Eclampsia is one of the most severe obstetric diseases and commonly occurs in the third trimester of pregnancy.1) Recently, eclampsia was recognized as one type of hypertensive disorder in pregnancy.2) It is a life-threatening obstetric emergency and may lead to maternal or perinatal death.

Major features of this disease include acute seizures and an altered mental state.1,2) We encountered a case of primigravida presenting with eclamptic seizures at 22 weeks’ gestation, without focal neurological deficits. The patient was kept under observation because of the prematurity of the fetus, but the case progressed to intrauterine fetal death (IUF), multiple organ failure, and terminated with an emergency caesarean section within 2 days from the onset of convulsions.

Case presentation

A 37-year-old female presented with a first-time pregnancy without any reproductive assistance. She had no prior medical history, including epilepsy, allergies, history of oral medication, or any condition that represented a risk of placental abruption.3) Her routine prenatal medical checkup findings were normal. However, her blood pressure readings were 123/85 mmHg at 10 weeks 5 days, 141/76 mmHg at 14 weeks 4 days, and 128/79 mmHg at 20 weeks 0 days. The patient demonstrated pitting edema at 21 weeks. At 22 weeks 2 days, she visited a local obstetrics clinic, complaining of a headache and vomiting. During the clinical examination, the patient had a sudden tetanic seizure that lasted for 30 seconds. After intramuscular administration of diazepam (2.5 mg), the attack ended. Suspecting eclampsia, the patient was transferred by ambulance to our tertiary hospital for assessment and clinical management of the convulsions. Upon arrival, her seizures stopped and the patient appeared lucid. On admission, her vital signs were stable, with a blood pressure of 152/84 mmHg, heart rate of 66 bpm, and 98% SpO2. Transabdominal ultrasonography (TAUS) revealed that the fetus was in a breech presentation. In addition, the fetus presented with 329 g of estimated fetal weight.
Early eclampsia and IUFD on 22w GA

(−2.1 SD), an amniotic fluid index of 11.2 cm, and an umbilical artery flow resistance index of 0.75. No post placental hematoma or placental thickening was found. Cardiotocogram (CTG) indicated low variability and showed variable decelerations once. Her laboratory data showed a white blood cell count of 7,200/μl, hemoglobin level of 12.2 g/dl, hematocrit of 38.3%, platelet count of 189,000/μl, prothrombin time international normalized ratio of 0.91, activated partial thromboplastin time of 23.4 sec, fibrinogen degradation products of 10.0 μg/ml, fibrinogen level of 294 mg/dl, antithrombin activity of 76%, aspartate aminotransferase level of 20 U/l, alanine transaminase level of 13 U/l, blood urea nitrogen level of 13 mg/dl and creatinine of 0.67 mg/dl. Urine protein level was more than 300 mg/dl.

Owing to the prematurity of the fetus and stable condition of the patient, we chose a course of observation after informing the patient and receiving her consent. We used a continuous intravenous magnesium sulfate drip to prevent recurrent eclampsia and betamethasone for fetus maturation, per the guidelines for such cases.2) The next day, MRI showed a high-density area in the occipital lobe that indicated posterior reversible encephalopathy syndrome (PRES), a finding typical of eclampsia (Figure 1).3) Two days after administration, the patient complained of acute lower abdominal pain, and fetal bradycardia was found on CTG. TAUS revealed the absence of umbilical arterial Doppler flow. After 2 h, the fetal heartbeat disappeared and placental thickening occurred. With a diagnosis of IUFD and placental abruption, we attempted to induce vaginal delivery using oxytocin. However, blood pressure and serum creatinine levels gradually increased (Figure 2), suggesting multiple organ failure. To prevent the mother’s condition from worsening, emergency cesarean section was performed. The infant (female) was delivered weighing 298 g, and

Figure 1. MRI FLAIR imaging performed the day after the convulsive attack.
High-density areas in the bilateral occipital and parietal regions are evident, suggesting PRES after eclampsia.

Figure 2. Course of the case.
Urine, urinary amount per day; Cre, serum creatinine; BP, blood pressure; FD, fetal death; C/S, cesarean section; POD, post-operative day.
the placenta showed evidence of abruption (about 50%). The disseminated intravascular coagulation (DIC) score was 10 points at that time (eclampsia, IUFD, oxygen therapy).

After the operation, the clinical course was good and no blood transfusion was required. The mother’s laboratory data were in the normal ranges on post-operative day 3, her blood pressure dropped back to normal on post-operative day 7, and proteinuria disappeared by day 3. Blood test revealed that her anti-beta-2 glycoprotein 1, anti-nuclear, lupus anticoagulant and anti-double stranded DNA IgG antibodies were all negative on post-operative day 5. On the follow-up MRI (post-operative day 9), the high-density area had disappeared, which matched the clinical course of PRES. The patient was discharged on day 9 and received follow-up treatment through our outpatient department, without complications.

Discussion

We observed a case of very early onset eclampsia at 22 weeks’ gestation, which rapidly progressed to IUFD due to placental abruption. In Japan, of the 330,399 deliveries registered in the perinatal database assembled by the Perinatal Committee of Japan, Society of Obstetrics and Gynecology between 2005 and 2009, only 246 cases of eclampsia (0.07%) were available for analysis, and included 4 cases of maternal mortality (data taken from the committee report, written in Japanese). However, the gestational ages at eclampsia onset were not shown in these data. In the United States, Schenone et al. reported 87 eclampsia cases of 59,388 deliveries from August 1998 to April 2011, 41 cases of which were diagnosed before 32 weeks of gestation. Even in this report, very early onset of eclampsia, such as that at 22 weeks’ gestation, appears to be quite rare. Only Sibai has reported some cases in which eclampsia occurred at 20 weeks’ gestation. The severity of the disease and potential clinical outcomes after conservative management specific to cases of very early onset eclampsia remain unknown. Therefore, our case may be an important example of a poor outcome.

Placental abruption has been reported to occur often with eclampsia. Previous reports suggest that the severity of the eclamptic symptoms influenced the degree of placental separation; in the present case, this was 50%. A course of observation alone for the patient after the onset of eclampsia might not be commonly chosen due to the possibility of recurrent eclampsia, brain stroke, multiple organ failure, or placental abruption, even after the convulsions appear to have ended. Due to the prematurity of the fetus, we opted for a conservative course of treatment, but this resulted in fetal death and emergency cesarean section. A detailed explanation regarding possible complications must be presented to the patient and family when considering the option not to terminate the pregnancy after eclampsia.

In the present case, the patient’s blood pressure increased once at 14 weeks’ gestation. In retrospect, the possibility of pre-existing hypertension should have been considered, as this can quickly develop into superimposed preeclampsia or eclampsia. According to the Japanese Guidelines, anti-hypertensive drug treatment is recommended (Grade C) in the presence of organ injury, even in cases with mild hypertension, and should aim for a blood pressure of less than 140/90 mmHg. In this case, the patient developed pitting edema at 21 weeks’ gestation, and headache, vomiting, and a convulsive attack at 22 weeks’ gestation. If she had begun home blood pressure monitoring beginning in the first trimester of pregnancy, acute increases in blood pressure could have been detected earlier and the patient could have received anti-hypertensive drugs or been admitted to the hospital earlier. It is necessary to consider the possibility of hypertensive disorders that can lead to severe complications in pregnancy, even if the patient is in the early stages of pregnancy.

Conflict of interest

None.

References

1. Sibai BM. Diagnosis, prevention, and management of eclampsia. Obstet Gynecol. 2005; 105: 402–410.
2. Takagi K, Yamasaki M, Nakamoto O, et al. A Review of Best Practice Guide 2015 for Care and Treatment of Hypertension in Pregnancy. Hypertens Res Pregnancy. 2015; 3: 65–103.
3. Ananth CV, Smulian JC, Demissie K, Vintzileos AM, Knuppel RA. Placental abruption among singleton and twin births in the United States: risk factor profiles. Am J Epidemiol. 2001; 153: 771–778.
4. Schenone MH, Miller D, Samson JE, Mari G. Eclampsia characteristics and outcomes: a comparison of two eras. J Pregnancy. 2013; 2013: 826045.
5. Usta IM, Sibai BM. Emergent management of puerperal eclampsia. Obstet Gynecol Clin North Am. 1995; 22: 315–335.
6. López-Llera M, de la Luz Espinosa M, Arratia C. Eclampsia and placental abruption: basic patterns, management and morbidity. Int J Gynaecol Obstet. 1988; 27: 335–342.