Eclampsia is defined as seizures that cannot be attributed to other causes in a woman with preeclampsia [1]. Postpartum eclampsia is defined as preeclampsia plus seizures after delivery and refers to the sudden onset of seizures shortly after giving birth. This condition has the potential to cause serious damage to the central nervous system, heart, and other major organs in the mother’s body if it is not recognized promptly [1,2]. Symptoms tend to arise within 48 hours after delivery, but it is possible to experience postpartum eclampsia several days or even weeks later.

If the condition worsens and becomes severe preeclampsia, the mother will have a higher rate of serious complications, including eclampsia or HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome, which may induce significant morbidity and mortality [1].

The patient is susceptible to postpartum eclampsia if the patient’s mother has preeclampsia in the antepartum period. Moreover, if the mother has postpartum preeclampsia, she will tend to have a greater chance of becoming eclamptic compared to those having antepartum preeclampsia [2–4].

A 29-year-old, para 0, woman with 37 + 4 gestational weeks was admitted to our hospital because of preterm rupture of the membrane without labor sign. The patient had neither hypertension nor diabetes mellitus prior to pregnancy, nor had pregnancy-induced hypertension. She became pregnant naturally. She received a routine prenatal visit at our hospital. The results of her blood examination were uneventful. Amniocentesis was not carried out because she was in the low risk group. Prenatal sonography revealed no abnormality. She declared no specific medications, herbs, or alternative medications during prenatal surveillance.

She was admitted to our hospital because of preterm rupture of the membrane without labor signs. We administered oxytocin for induction. She then received a cesarean section (CS) because of an arrest of cervical dilatation at 5 cm. A female baby weighing 3250 g was born. The Apgar score was 5 at 1 minute and 7 at 5 minutes.

The postoperative status was smooth. The mother was then transferred to the regular ward for further care. However, the patient suffered from vomiting several times after continuous epidural anesthesia. Eight hours after CS, a sudden onset of general convulsion was noted. She became unconscious and was unresponsive for a few minutes. The convulsion eventually stopped, and she regained consciousness after 5 minutes. We administered midazolam (5 mg, IVA) at that time. Her blood pressure was 137/81 mmHg when the convulsion stopped. The patient then behaved as usual the next morning. She had low-grade fever (37.8 °C) in the afternoon, but no sign of infection was noticed. The fever subsided after the patient had taken acetaminophen. A second general convulsion, however, developed 9 hours after the first attack. This lasted 5 minutes. The vital signs were as follows: body temperature = 37.4 °C, heart rate = 75/min, respiratory rate = 19/min. Lorazepam (2 mg) was administered. We consulted a neurologist, and a brain computed tomography scan was performed. We administered continuous MgSO4 dripping. The blood examination showed leukocytosis (white blood cell count = 16,900/cumm with left shift; segment = 87.9%) and thrombocytopenia (platelet = 14,9000/cumm). Elevation of C-reactive protein (5.53 mg/dL), lactic dehydrogenase (415 U/L), liver aminotransferase (alanine aminotransferase = 44 U/L; aspartate aminotransferase = 52 U/L), and acute renal function impairment (creatinine = 1.26 mg/dL) were noted. The serum calcium was within normal range (Ca = 8.2 mg/dL). We also administered calcium gluconate 20 mg. The other laboratory values, including electrolytes and blood sugar, were within normal range (Na = 141 mmol/L; K = 3.6 mmol/L; Cl = 107 mmol/L;
IP = 3.5 mg/dL; glucose = 106 mg/dL). Nevertheless, hypoalbuminemia (albumin = 2.6 g/dL) was identified.

The brain computed tomography scan showed the presence of air in the right side internal carotid artery, anterior communicating artery, and pericallosal artery. A suspicious enlargement of the pituitary gland with high density was also found (Fig. 1). The brain magnetic resonance imaging also revealed multiple small patches of high signal change on T2WI in the bilateral temporo-parieto-occipital and bilateral high frontal cortical regions with mild facilitated diffusion (Fig. 2). Moreover, swelling of bilateral basal ganglia with high heterogenous faint high signal intensity was noted on T2-weighted images, accompanied by heterogeneous facilitated diffusion and foci of minimal restricted diffusion on the diffusion-weighted images (Fig. 3). The above imaging findings are suggestive of vasogenic edema. The diagnosis was thought most likely to be PRES (posterior reversed encephalopathy syndrome) due to eclampsia.

The patient complained of blurred vision after the convulsions, but the ophthalmological examinations showed no significant finding. The blurred vision spontaneously showed gradual improvement. No more blurred vision was noted in the next day. The following vital signs and blood examinations showed improving results. No fever or hypertension was noted. Postoperative 24-hour urine protein was 0.59 g. We maintained MgSO4 infusion until the 4th day of hospitalization. She was discharged under a relatively stable condition without any sequels.

In the presented case, the patient had no history of antecedent hypertensive disease or proteinuria. She was admitted for induction because of a premature rupture of the membrane. Regarding the seizure, we arranged a series of examinations to rule out other etiologies, such as organic brain, or metabolic cause, such as infection. Eclamptic seizure was thought likely according to the obstetric history, high systolic blood pressure, results of laboratory values (proteinuria), and the imaging study.

The patient used pain control analgesia after the surgery. She then experienced nausea and vomiting. An opioid agent also induced similar symptoms. It would mimic the presenting symptoms and signs prior to eclamptic seizure developing.

![Fig. 1](image1.png)

**Fig. 1.** Air in the right side internal carotid artery and anterior communicating artery (arrow and open arrow). Air in the pericallosal artery (thin arrow). The borderline enlargement of the pituitary gland (curved arrow) with high density.

![Fig. 2](image2.png)

**Fig. 2.** (A) The axial T2-weighted MR image showed high signal intensity on T2-weighted images in the posterior cortex (white arrows). (B) The axial diffusion-weighted DWI MR image did not show restricted diffusion. Therefore, vasogenic edema instead of cytotoxic edema is confirmed by the image. DW = diffusion-weighted imaging; MR = magnetic resonance.
The patient fell abdominal pain prior to the first seizure. Nevertheless, wound pain and contraction pain are inevitable after a cesarean section. Therefore, abdominal pain may be more helpful in patients who do not receive surgery or several days after receiving surgery. It also indicated that we should not preclude the possibility of developing eclampsia when a patient has abdominal pain after surgery [2–4].

The patient’s vision recovered spontaneously. This was similar to the report by Saifudeen et al [3], who presented a case of postpartum eclampsia without a history of pre-eclampsia or eclampsia, accompanied by PRES. Another neurologic disorder, called “postpartum cerebral angiopathy,” also displays associated headache, seizure, and focal neurologic deficit. It is, however, caused by intracerebral hemorrhage with local mass effect, and the blood pressure is normotensive, unlike that in our patient [5].

Pituitary apoplexy is defined as acute hemorrhagic infarction in an existing pituitary adenoma or otherwise physiologically enlarging pituitary gland. The patient may experience severe headache, vomiting, and visual disturbances, including visual field defects and restricted eye movement. In our patient, pituitary apoplexy also might have contributed to the symptoms. The pituitary gland showed borderline enlargement, at a size of 7.3 mm.

The exact mechanism of eclampsia is still unknown. Nevertheless, it is likely to involve cytotoxic effects on the vascular endothelium leading to increased permeability and vasogenic edema [4]. Therefore, it may result in posterior reversible encephalopathy syndrome, which can be associated with several medical conditions, including hypertensive encephalopathy and uremia. Our patient’s magnetic resonance imaging scan presented bilateral temporo-parieto-occipital region and bilateral frontal cortical region compatible with PRES.

Treatment in pituitary apoplexy and eclampsia are supportive [2–4]. We still need to follow up on the endocrine condition of the patient to determine whether pituitary apoplexy caused hormone deficiency.

In Taiwan, a person has medical insurance “Jian bao” and a puerperium woman can be hospitalized for 3 days in NSD and 5 days in CS. This enables the physician to take preventive measures for peripartum and postpartum eclampsia in patients who already present preeclampsia. Nevertheless, we still cannot predict who will develop eclampsia if the patient has no history of hypertensive disease, except at a younger age [2]. We have to be concerned about the development of postpartum eclampsia when the patient experiences headache, nausea, vomiting, and epigastric pain. The blood pressure should be monitored and prophylactic MgSO4 can be given if hypertension arises [1–4].

In conclusion, postpartum eclampsia can occur, even with no antecedent or peripartum hypertensive disease, as in the case with our patient. The most common symptoms of postpartum eclampsia include headache, nausea, vomiting, epigastric pain, and blurred vision. The typical imaging finding is posterior reversible encephalopathy syndrome. Prevention by medical treatment is recommended. If it does occur, supportive care and prevention of recurrence are necessary. MgSO4 is the treatment of choice. Because of the associated morbidity and mortality, we should educate the patients, their families, and the clinical physicians to avoid delayed diagnosis and take proper management.

References


