

Successful rescue of antepartum eclampsia in a Chinese patient

Case report

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Abstract

Rationale: Eclampsia is a life-threatening complication of pregnancy. Eclampsia is a leading cause of maternal and neonatal morbidity and mortality with most injury being associated with the seizures that mark the onset of the disease. It is vital that medical worker managing eclampsia have an understanding of the disease process.

Patient concerns: A 28-year-old female, G4P1, with history of caesarean section was admitted at GA34+6, in addition to headache and severe hypertension (180/120 mm Hg) and proteinuria (+++). The evaluation of coagulation parameters showed positive D-dimer and increased fibrinogen and fibrinogen degradation product (FDP) and PT percent activity. Her biochemical analysis showed a decrease in total protein and an increase in alanine transaminase (ALT) and lactate dehydrogenase (LDH) and high serum uric acid and hyperlipidemia.

Diagnoses: She was diagnosed with severe preeclampsia (PE).

Interventions: First, the patient received magnesium sulfate therapy for convulsions control. Next, antihypertensive management of labetalol orally at a dose 100 mg and nifedipine orally at a dose 10 mg and glycerin trinitrate 10 mg iv were used to maintain blood pressure in a safe range. Then, corticosteroid was given for enhancing fetal lung maturation. During preparation for cesarean section, the patient experienced suddenly seizures that lasted approximately 2 to 8 minutes. The immediate therapy is to stop the convulsions and reduce blood pressure.

Outcomes: The patient and her baby were discharged from the hospital on the 7th day after the operation with normal blood pressure and being in a satisfactory condition.

Lessons: Eclampsia is defined as the occurrence of convulsions superimposed on the preeclampsia. The awareness of eclampsia enhances early diagnosis and timely administration of magnesium sulfate and calmativ drug which are critical to avoid fetomaternal complications.

Abbreviations: ALT = alanine transaminase, FDP = fibrinogen degradation product, IV = intravenous, IVP = intravenous push, LDH = lactate dehydrogenase, LDL = low density lipoprotein, PE = preeclampsia, PT = prothrombin time, TC = cholesterol total, TG = triglyceride.

Keywords: antepartum eclampsia, headache, preeclampsia, seizure, successful rescue

1. Introduction

Eclampsia is not only very serious, but can be life-threatening for pregnant women. It was found that 79% of cases of preeclampsia developed in eclampsia.^[1] Preeclampsia is a complex multisystem disorder of unknown etiology, characterized by the

combined development of high blood pressure and proteinuria (>300mg/24h) after the first 20 weeks of pregnancy.^[2] Eclampsia was defined as presence of generalized convulsions in the presence of pre-eclampsia and described as the grand mal type of seizure first appearing before, during labor or within 48 hours from delivery, and/or coma unrelated to other cerebral

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Table 1**Laboratory parameters for complete blood count.**

Main parameters	Admission	1 d preop	1 d postop	3 d postop	6 d postop	Normal range
Hemoglobin, g/L	122	118	99	95	98	112–172
Platelet count ($\times 10^9/L$)	198	137	157	187	207	85–320
Erythrocyte count ($\times 10^{12}/L$)	3.98	3.73	3.20	3.04	3.03	3.50–5.50
White-cell count ($\times 10^9/L$)	8.44	6.81	19.16	14.05	7.85	3.50–10.0

The normal ranges of the variables are based on the Chinese population.

conditions in women with preeclampsia.^[3] Eclampsia is one of the leading causes of maternal and perinatal morbidity and mortality worldwide. The prevalence of eclampsia is reported to be 0.2% in China.^[4] Recent studies found that optimizing health care to prevent and treat women with preeclampsia/eclampsia is a necessary step toward reducing maternal and infant mortality and morbidity.^[5] The present article describes a successful rescue case of eclampsia before labor in severe preeclampsia patients.

2. Case report

A 28-year-old female, G4P1, with history of caesarean section was admitted to our hospital at GA34⁺6. She complained of headache. A physical examination at admission revealed blood pressure 180/120 mmHg and proteinuria (+++) and quantitative proteinuria 3.23g/24h. Maternal heart rate was 88 beats per minute, respiratory rate was 18 breaths per minute. Ophthalmic

examination showed vasospasm of fundus oculi. Initial laboratory tests showed normal hemoglobin (120g/L) and platelet count ($198 \times 10^9/L$) (Table 1). The evaluation of coagulation parameters showed positive D-dimer (892 ng/mL) and decreased prothrombin time (PT, 9.00/s) and PTR ratio 0.79 and PT-INR 0.80 and increased fibrinogen (5.13 g/L) and fibrinogen degradation product (FDP, 7.9 ug/mL) and PT percent activity140 (Table 2). Her biochemical analysis for renal and hepatic function showed a decrease in total protein (55.50g/L) and albumin (27.0g/L) and albumin/globulin ratio (1.15) and elevation in alanine transaminase (ALT 40.2IU/L) and lactate dehydrogenase (LDH 248IU/L) and uric acid (636 $\mu\text{mol/L}$) (Table 3). Biochemical for lipid of cholesterol total (TC, 8.49 mmol/L) and triglyceride (TG, 7.12 mmol/L) and low density lipoprotein (LDL, 5.26 mmol/L) are shown in Table 4.

Obstetric ultrasound revealed fetus with no obvious malformations; blood flow velocity waveform indices of the fetal and fetoplacental circulation were within normal range; fetal heart

Table 2**Laboratory tests for coagulation parameters.**

Main parameters	Admission	1 d preop	1 d postop	3 d postop	6 d postop	Normal range
Prothrombin time, s	9.00	9.50	9.50	9.50	9.40	9.90–12.90
Fibrinogen, g/L	5.13	6.81	10.64	7.63	10.28	2.38–4.98
FDP, $\mu\text{g/mL}$	7.9	26.0	52.7	7.1	4.4	0.0–5.0
PTR ratio	0.79	0.83	0.83	0.83	0.82	0.82–2.00
PT-INR	0.80	0.84	0.84	0.84	0.83	0.82–2.00
PT percent activity	140	126	128	126	126	80–120
Thrombin time, s	20.5	18.6	16.6	17.1	15.9	15.8–24.9
D-dimer	892	6561	1317	927	669	0–255

The normal ranges of the variables are based on the Chinese population.

Table 3**Laboratory parameters for liver and renal function.**

Main parameters	Admission	1 d preop	1 d postop	3 d postop	Normal range
Total protein, g/L	55.50	42.10	40.70	45.30	60.00–83.00
Albumin, g/L	27.0	23.30	22.90	27.80	35.00–55.00
Albumin/globulin ratio	1.15	1.24	1.29	1.59	1.26–2.40
Total bilirubin, $\mu\text{mol/L}$	3.3	3.8	3.0	4.2	3.4–20.5
Bilirubin direct, $\mu\text{mol/L}$	2.1	2.8	1.3	2.1	0.0–6.8
Indirect bilirubin, $\mu\text{mol/L}$	1.2	1.0	1.7	2.1	0.0–15.0
Direct bilirubin/total bilirubin	0.64	0.73	0.43	0.50	0.00–0.60
Retinol binding protein, mg/L	81.20	65.40	41.20	33.60	25.00–70.00
Alanine aminotransferase, IU/L	40.20	31.10	40.30	23.80	4.0–40.0
Aspartate aminotransferase, IU/L	11.30	11.70	19.0	10.40	4.0–44.0
Lactate dehydrogenase, IU/L	248	294	287	198	109–245
Urea nitrogen, mmol/L	6.55	4.95	6.53	3.27	1.43–7.14
Creatinine, $\mu\text{mol/L}$	77.6	68.5	83.1	54.2	45.0–84.0
Uric acid, $\mu\text{mol/L}$	636	505	651	448	155–357

The normal ranges of the variables are based on the Chinese population.

Table 4**Laboratory parameters for maternal lipids.**

Main parameters, mmol/L	Admission	1 d preop	3 d postop	6 d postop	Normal range
Cholesterol total	8.49	8.28	6.26	5.39	3.10–5.20
Triglyceride	7.12	7.56	5.65	3.23	0.40–1.70
Low-density lipoprotein	5.26	5.22	3.95	3.25	0.00–3.37
High density lipoprotein	1.92	1.76	1.32	1.33	1.15–2.00

The normal ranges of the variables are based on the Chinese population.

rate 140 beats per minute; the biparietal diameter 8.5 cm; femoral diameter 6.3 cm; placental subtraction II (fetal bias small); depth of amniotic fluid 5.0 cm; singleton pregnancy; normal position of fetus and an estimated fetal weight of 2200 g.

She was diagnosed with severe preeclampsia. Corticosteroid was given for enhancing fetal lung maturation and magnesium sulfate regimen includes a loading dose of 6 g intravenous (iv) over 20 minutes followed by a continuous infusion of 2 g/h starting during the period of observation for prophylaxis convulsions and antihypertensive medications of labetalol oral doses 100 mg q8h and nifedipine oral doses 10 mg q6h and glycerin trinitrate 10 mg iv are used to maintain blood pressure in a safe range.

Two days after admission, the patient general condition deteriorated with fluctuation of blood pressure between 186–117/68–120 mm Hg after antihypertensive treatment. Urine tests showed a gradual increase in proteinuria: from 3.23 g/L to 4.57 g/L. The course of preeclampsia was accompanied by liver dysfunction which was shown by the elevated levels of liver enzymes in the peripheral blood: alanine transaminase (40.2–46.3 IU/L) and lactate dehydrogenase (248–287 IU/L), and decrease total protein (55.50–40.70 g/L) and albumin (27.0–22.90 g/L) and globulin (23.5–17.5 g/L) and D-dimer (892–6561). After expert evaluation, further continuing the conservative treatment was considered unreasonable and the patient underwent an emergency cesarean section diagnosed with severe preeclampsia. During prepare for cesarean section, the patient experienced suddenly seizures that lasted approximately 2 to 8 minutes. The patient's blood pressure was 218/127 mm Hg, heart rate 108 beat/min, respiratory rate of 20 breaths/min, reduced level of consciousness with the Glasgow Coma Score of 11, convulsion and neck stiffness—a diagnosis of eclampsia was made. The patient was given magnesium sulfate 10 g solution diluted in 0.9% sodium chloride, given intravenously continuous infusion; glycerin trinitrate 10 mg for high blood pressure control; received midazolam immediately in a loading dose of 0.05 mg/kg in 100 mL of isotonic saline for sedation; diazepam 10 mg intravenous push (IVP) was administered, which successful halted the seizures. Midazolam is a fast acting benzodiazepine and has been used for sedation and as an anticonvulsant including eclampsia in the case. Subsequent follow-up with emergency cesarean section was performed, delivering a live-born male infant 2200 g with Apgar scores 5 and 8 at 1 and 5 minutes, respectively. Neonate was admitted to the neonatal intensive care unit due to neonatal asphyxia.

Following delivery, the patient's clinical condition and laboratory values deteriorated, with progressive liver insufficiency (total protein 39.7 g/L, albumin 20.8 g/L, globulin 16.9 g/L) 2 days postpartum. Laboratory values showed a sharp increase in WBC level to 19,160/mm³, and a platelet count nadir of 95,000/mm³. Bedside evaluation of coagulation parameters showed positive D-dimer (927 ng/mL) and decreased PT 9.50/s and

increased fibrinogen 7.63 g/L and FDP 7.1 ug/mL and PT percent activity 126 (Table 2). The patient exhibited hypo-proteinemia and low hemoglobin and infection. The albumin (10 g per day) and blood component transfusion and the use of anti-infection drugs were administered. The patient and her baby were discharged from the hospital on the 7th day after with normal blood pressure and being in a satisfactory condition. The ethics committee of the Maternity and Child Health Hospital (Zhenjiang, China) approved the study protocol, and the patient provided written informed consent.

3. Discussion

Eclampsia itself is a rare condition, occurring 0.2% in our country population, carries a significant risk for poor maternal and neonatal outcomes. Physicians and nurse should have an elevated sense of caution with preeclampsia patients who have developed eclampsia and are aware of the complications that arise, and further are aware that eclampsia may be a sign that the high blood pressure disease process is worsening.^[6] The pathophysiology of preeclampsia is well studied yet poorly defined with multiple theories—many of which center on endothelial dysfunction and vasoconstriction.^[7] It is proposed that cerebral endothelial dysfunction and changes in vascular permeability result in cerebral edema at the time of severe PE that can result in the development of eclampsia involving seizures.^[8] For our patient, we assume that cerebral endothelial dysfunction and vasoconstriction in PE had a role in the development of eclampsia.

Eclampsia is defined as seizure activity in conjunction with preeclampsia. In the case, patient initially should meet the criteria of preeclampsia, as she had elevated blood pressure and proteinuria (+++ and 3.23 g/24 h). 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 should raise suspicion for preeclampsia. Although PE and eclampsia do not always occur in succession, it was found that 79% cases of PE developed into eclampsia. Therefore, expectant management may be acceptable before delivery. This should include close maternal and fetal surveillance maternal vital signs and Doppler examination for fetal assessment as well as laboratory assessments. Magnesium sulfate is the drug of choice in prevention of convulsions in eclampsia, but conventional anticonvulsants could be administered when the diagnosis is certain.^[9] In addition, corticosteroid to accelerate fetal lung maturity and labetalol and nifedipine antihypertensive therapy. Treatment, however, must be individualized. In our patient who manifested severe hypertension, the treatment of choice was labetalol and nifedipine and glycerin trinitrate. As per the new ACOG guidelines, oral labetalol and nifedipine and IVP glycerin trinitrate have also been suggested as an effective initial antihypertensive treatment.

The patient in this case had experienced acute-onset headaches and abnormal fluctuations in blood pressure for 2 days before seizure activity beginning. During the acute episode of seizure, the immediate therapy is to stop the convulsions, reduce blood pressure, establish a clear airway, positive pressure ventilation, and prevent major complications. It has been postulated that hypertension in eclampsia leads to increased perfusion in the cerebral circulation and vasogenic edema, which can lead to seizure.^[10] The case suggests that mortality could be lowered with anticonvulsants and antihypertensive therapy in patients with seizure.^[11] The exact mechanism of seizure in eclampsia is not very clear, but is most likely secondary to a combination of cerebral edema, ischemia, and transitory vasospasm of the cerebral vasculature. The etiology, pathogenesis, and prognosis of eclampsia are associated with the primary disease. Thus, early diagnosis and prompt treatment of the primary disease should improve the prognosis.

After delivery, the patients exhibited hypo-proteinemia and low hemoglobin and infection that albumin (10g per day) and blood component transfusion and anti-infection drugs were administered. The patient general condition started to resolve. She was discharged from the hospital on the 11 day after admission. In the case presented, medical teams acted quickly and appropriately recognition and prompt interventions with a good outcome for both mother and fetus. In addition, a key finding of our case was that high serum uric acid levels are associated with patient. The etiology of high serum uric acid levels in PE is thought to be multifactorial, with contributions from decreased renal tubular secretion, increased oxidative stress as the result of placental ischemia and maternal reduction in glomerular filtration rate.^[12,13] Lipid profiling plays an important role in evaluating cardiovascular risk in PE patients. Biochemical identification of PE patients at increased risk of cardiovascular is guided by measurement of triglyceride and total cholesterol and low density lipoprotein which could reflect a compromised vascular function and endothelial dysfunction. Known warning signs of eclampsia include elevated blood pressure, headaches, vision changes, and peripheral edema outside dependent areas. Furthermore, majority of deaths due to eclampsia are avoidable through the provision of timely and effective care to the women presenting with these complications.

One limitation of this study is the missing data due to its follow-up design. The strengths are that it is important to rapidly diagnose antepartum eclampsia to initiate appropriate interventions and hopefully prevent further complications, and even death, in the rare cases. In addition, a key finding of our case is that high serum uric acid levels and high levels of triglyceride and total cholesterol and low density lipoprotein are associated with patient could reflect a compromised vascular function and endothelial and renal dysfunction. In addition, we carefully collected most of the data from individual medical records to ensure data accuracy.

Taken together, eclampsia itself is a rare and serious and life-threatening condition encountered by pregnant women. When severe preeclampsia is accompanied by seizures, it is called eclampsia. It is vital that medical worker managing preeclampsia have an understanding of the disease process. In our case, physicians and nurse can know warning signs of eclampsia and realize optimal therapy weighing the benefits and risks is based mainly on an overall understanding and comprehensive assessment of the eclampsia pathophysiological conditions. The case illustrates that early recognition and prompt interventions are the keys to reducing morbidity and mortality in patients with eclampsia.

Author contributions

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