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CASE REPORT

Atypical eclampsia: A case report

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Abstract

Background*: Most women with eclampsia have a previous history of preeclampsia (proteinuria and hypertension). However, there has been a paradigm shift in this philosophy. There is evidence that atypical eclampsia can occur even in the absence of proteinuria and hypertension which presents a diagnostic dilemma and challenge for obstetricians and gynecologists. The purpose of this case report is to raise awareness about nonclassical and atypical signs of eclampsia so as to avoid complications* ***Case:*** *A multigravida woman with 39-40 weeks gestational age came to the hospital with signs of labor. On examination, blood pressure was 120/70 mmHg without a history of hypertension during antenatal examination, fetal heart rate was 146-156 beats/minute, cervical dilatation was 7-8 cm, with routine blood results within normal limits and planned for vaginal delivery . When monitoring the patient suddenly had a tonic clonic seizure, a complete blood count was performed, liver, kidney, electrolyte function was within normal limits, and proteinuria was found to be +2. The patient was treated with magnesium sulfate and the baby was delivered by vacuum extraction. This patient was managed appropriately with good outcome* ***Discussion:*** *Atypical eclampsia accounts for about 8% of all cases of eclampsia. Atypical forms of eclampsia have an erratic onset. This experience highlights some of the difficulties in managing atypical cases of eclampsia, namely the erratic onset and unpredictable course of the disease that can interfere with timely diagnosis and treatment and contribute to maternal and fetal morbidity and mortality. While controlling seizures by initiating magnesium sulfate therapy,*

Keywords: *eclampsia; atypical eclampsia; non-classical eclampsia*

BACKGROUND

Hypertensive disorders in pregnancy contribute significantly to maternal and perinatal mortality in both industrialized and developing countries.[1] Eclampsia is defined as the occurrence of 1 or more generalized tonic-clonic seizures in a woman associated with a hypertensive disorder in pregnancy. Approximately 10% of pregnancies are affected by hypertensive disorders, and eclampsia occurs in 0.8% of women with hypertensive disorders.[2] The incidence of seizures in eclampsia is decreasing in countries where adequate health care is available. In industrialized countries the average incidence is 1 case from 2000 to 3000 deliveries each year. At Parkland Hospital, the incidence has decreased substantially over the past decade and is approaching 1 case in 2000 births. This frequency may be related to increased access to prenatal care and an active management approach.[3] In 2015, approximately 42,000 women died from hypertensive disorders of pregnancy worldwide; more than 99% of these deaths occur in low- and middle-income countries. [4] From WHO data in Indonesia, it was recorded that maternal deaths in 2019 were 177/100,000 live births with the cause of

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hypertension in pregnancy being the second highest position after bleeding. [5] Other sources state that the reported incidence of eclampsia is 1.6 to 10 per 10,000 deliveries in developed countries, while in developing countries it is 50 to 151 per 10,000 deliveries. In addition, low-resource countries have significantly higher rates of maternal and perinatal mortality and morbidity.[2]

However, very little is known about the atypical preeclampsia-eclampsia syndrome. This disorder is used to describe a nonclassical form of a hypertensive disorder that develops during pregnancy. Atypical eclampsia occurs in the absence of hypertension and/or proteinuria, before 20 weeks of gestation and 48 hours postpartum, or despite receiving magnesium sulfate.[6] There are very few data on this atypical eclampsia and it is associated with a very rare incidence of 8% of cases of eclampsia. [7]

CASE DESCRIPTION

A 31-year-old woman came to the emergency room at Sungai Dareh Dharmasraya Hospital on May 23, 2022 with signs of labor since 6 hours before entering the hospital. The discharge from the genitals is clear when he arrives at the ER and there is no profuse bleeding from the genitals. Active fetal movements are felt by the mother. The patient is 9 months pregnant with the first day of the last menstruation on August 21, 2021 and the estimated date of delivery is May 28, 2022. The patient is pregnant for the 3rd time with a history of normal delivery in the first pregnancy and miscarriage in the second pregnancy. A history of regular antenatal care at the midwife every month and never had high blood pressure during pregnancy and there was no history of previous seizures. He denied a family history of hypertension. He denied history of using hormonal contraception.

Examination of the general status of the general condition appeared moderately ill with compos mentis consciousness, blood pressure 120/70 mmHg, pulse rate 92 times per minute, respiratory rate 22 times per minute, and temperature 36.8 degrees Celsius. Obese nutritional status with BMI 30.4kg/m2. On examination his obtained 3-4 times every 10 minutes with a duration of 45 seconds each his. Fetal heart rate is 146-156 beats per minute. Genital examination revealed cervical dilatation 7-8cm, membranes ruptured with clear residue, fetal head in Hogde I-II. The results of routine blood labor and ultrasound were within normal limits. The patient was diagnosed with active phase I stage I parturient G2P1A0H1 and planned for vaginal delivery.

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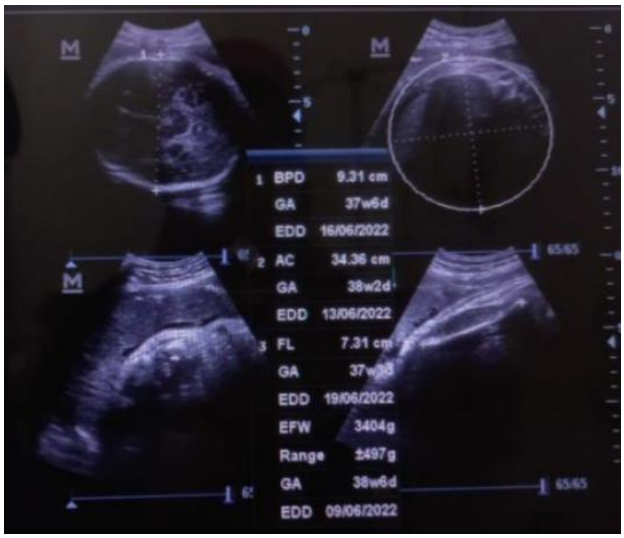

Figure 1. Normal Ultrasound Findings in Patients

When transferred to the delivery room the patient suddenly complained of sudden blurred vision and had a one-time tonic-clonic seizure for 3 minutes with somnolence after the seizure. Blood pressure was checked at 120/80 mmHg, pulse rate was 112 times per minute, respiratory rate was 18 times per minute, temperature was 36.8 degrees Celsius. Airway clearance was performed, oxygen was given 4 liters/minute with a nasal cannula, two-way infusion, urinary catheter, and a complete blood count cito was performed. The results were within normal limits with urine protein examination at that time the result was +2. Subsequently, an ECG was performed and the results were within normal limits. The patient was immediately given a regimen of magnesium sulfate with an initial dose of 4 grams and continued with a maintenance dose of 1 gram per hour. Obstetrical examination found his adequate, fetal heart rate within normal limits, and cervical dilatation is complete, the head is in Hodge II-III with the occiput anterior. The patient was then decided to accelerate the second stage with vacuum extraction. The baby was born with a male gender, weight 3200 grams, body length 50 cm, and Apgar score 7/9, the placenta was born complete and continued with monitoring the IV stage.

The patient underwent post partum treatment with magnesium sulfate regimen installed for 24 hours postpartum, oxytocin drip installed, and was consulted to a neurologist and no neurological abnormalities were found. As long as the patient is stable, he has never had high blood pressure. Oral therapy was given cefixime 2x200mg, paracetamol 3x500mg, vitamin C 1x500mg, and SF 1x180mg. The patient was discharged on the 3rd post partum day with the mother and baby in good condition.

DISCUSSION

Pre-eclampsia is a multisystem disorder that usually occurs before eclampsia. However, hypertension is only one of the signs and is not always present in patients with preeclampsia who progress to eclampsia. It is difficult to predict which organ systems will be involved.[8] The incidence of preeclampsia is also influenced by race, ethnicity, and genetic predisposition. Some of the clinical factors concluded by Bartsch et al (2016) with data involving more than

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25 million pregnancies are older age, nulliparity, obesity, diabetes, chronic hypertension, HELLP syndrome in previous pregnancies and underlying metabolic diseases, hyperhomocysteinemia, or chronic kidney disease.3 In the case we report, the risk factor associated with eclampsia was obesity with a BMI of 30.4kg/m2.

The pathogenesis/mechanism that explains the cause of preeclampsia is as follows:3

- Placental implantation with abnormal trophoblast invasion of uterine vasculature
- Dysfunctional immunological tolerance between mother, father (placenta), and fetal tissues
- Maternal maladaptation to cardiovascular and inflammatory changes in normal pregnancy
- Genetic factors

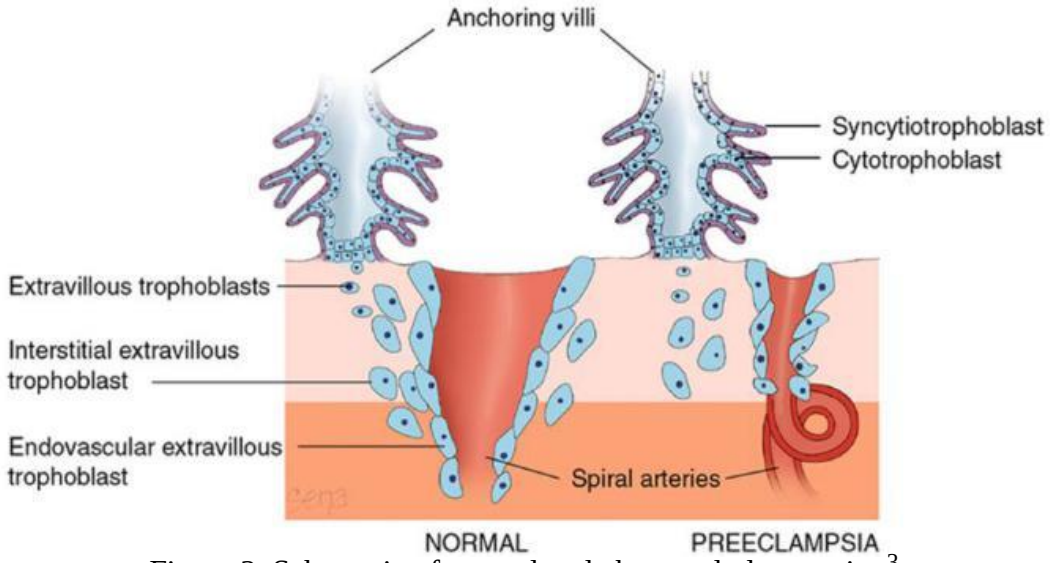

Figure 2. Schematic of normal and abnormal placentation[3]

Schematic representation of normal placental implantation showing proliferation of extravillous trophoblasts from anchoring villi. These trophoblasts invade the decidua and extend into the walls of the spiral arterioles to displace the endothelium and muscle walls to create low resistance that dilates the blood vessels. In the condition of preeclampsia, there is an implantation defect characterized by incomplete trophoblast invasion of the spiral arteriolar wall which causes failure to dilate blood vessels with high flow resistance.[3]

Eclampsia is a rare but serious complication of preeclampsia. This becomes more problematic in the absence of previous preeclampsia.[7] In the case we present the patient had no previous signs of preeclampsia and had preeclampsia with preeclampsia that was preceded by sudden blurring of vision. Seizures in eclampsia are caused by excessive release of excitatory neurotransmitters, especially glutamate, massive depolarization of tissue neurons, and bursts of action potentials. Clinical and experimental evidence suggests that prolonged seizures can lead to significant brain injury and brain dysfunction later in life. Scotomata is a term for blurred vision, or diplopia, which is common in severe preeclampsia and eclampsia. This usually improves with magnesium sulfate therapy and/or by lowering blood pressure. Permanent blindness is rare, usually reversible, and may arise from three potential areas: the

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visual cortex of the occipital lobe, the lateral geniculate nucleus, and the retina. In the retina, pathological lesions may result in ischemia, infarction, or detachment.[8]

Several other medical conditions can also cause seizures during pregnancy. The differential diagnosis of eclampsia is cerebrovascular trauma such as bleeding, aneurysm rupture or malformation, embolism or arterial thrombosis, cerebral venous thrombosis, hypoxic ischemic encephalopathy, hypertensive encephalopathy, seizure disorders, previously undiagnosed brain tumors, metastatic gestational trophoblastic disease, metabolic disorders such as hypoglycemia, hyponatremia, reversible posterior leukoencephalopathy syndrome, thrombophilia (catastrophic antiphospholipid syndrome), thrombotic thrombocytopenia purpura, postdural puncture syndrome, and cerebral vasculitis.[9] This differential diagnosis is important especially if there is a focal neurological deficit, prolonged coma, or atypical eclampsia, so it is necessary to evaluate the patient's cardiac and neurological condition.[6] In this patient's case, neurological, cardiac and metabolic conditions were investigated but no abnormalities were found.

The case of eclampsia in this patient occurred at term gestational age, which was 39-40 weeks of gestation with a proteinuria result of +2. Based on the literature, eclampsia is most common in the last trimester and is more common near term. In recent years, the incidence of postpartum eclampsia has decreased. This may be related to increased access to prenatal care, early detection of antepartum preeclampsia, and prophylactic use of magnesium sulfate. Importantly, other diagnoses should be considered in women with seizures more than 48 hours postpartum or in women with focal neurologic deficits, prolonged coma, or atypical eclampsia. At the time of seizure the patient must be protected, especially the airway. Strong muscle movements can make the patient fall out of bed, and if not protected, his tongue will be bitten by violent jaw movements. This phase occurs in which the muscles alternately contract and relax which can last about a minute. Gradually, muscle movements become smaller and less frequent until the seizures stop. 8 The severity of 24-hour proteinuria can predict adverse outcomes in women with preeclampsia, so proteinuria is still used as a monitoring index in patients with preeclampsia.[10]

After the diagnosis of eclampsia was established, this patient was immediately given an initial dose of MgSO4 regimen and continued maintenance until 24 hours after delivery. According to Cunningham, et al in 2018 that magnesium sulfate is very effective for preventing seizures in women with preeclampsia and stopping them in women with eclampsia and is an effective anticonvulsant that prevents central nervous system depression. The dose for severe preeclampsia is the same as for eclampsia. Because delivery is a more likely time for seizures to occur, women with preeclampsia-eclampsia are usually given magnesium sulfate during labor and for 24 hours postpartum.[8]

The philosophy that is still adhered to in America today regarding the management of eclampsia is: 1. Controlling seizures using an initial dose of magnesium sulfate given intravenously followed by a maintenance dose. 2. Administration of antihypertensive drugs intermittently to lower blood pressure. 3. Avoid diuretics unless pulmonary edema is evident, limit intravenous fluid administration unless fluid loss is excessive, and avoid hyperosmotic agents. 4. Termination of pregnancy.[8]

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Regarding maternal and perinatal outcomes Shin JE et al, in their retrospective study compared the outcomes between typical (classic) and atypical eclampsia, and concluded that perinatal outcomes were better in atypical cases, but maternal outcomes were similar.[11] Therefore, more attention needs to be focused on to atypical eclampsia

CONCLUSION

Hypertension in pregnancy is a serious threat to the life of the mother and fetus. Atypical eclampsia further complicates diagnosis, resulting in delays in diagnosis and management. In this report, we report a case of atypical eclampsia whose diagnosis was made based on clinical and supporting findings and ruled out other diagnoses that could cause seizures in pregnant women. The patient was managed appropriately with good results. This extremely rare case is a challenge and a diagnostic dilemma for clinicians and is ready to take immediate action. Awareness of the clinical entity, awareness of risk factors and prompt identification can certainly improve the fetomaternal prognosis.

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