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Loss of Consciousness Due to Eclampsia

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Abstract

This case report aimed to describe the clinical manifestations of decreased consciousness in patients with antepartum eclampsia, evaluate the medical and obstetric management provided, and review the clinical implications of eclampsia on maternal and perinatal outcomes. A 28-year-old woman, G1P0A0, at 33 weeks of gestation presented to the emergency department with two episodes of generalized tonic-clonic seizures occurring within 3 hours before admission. The seizures were preceded by severe headache, epigastric pain, and visual disturbances, followed by decreased consciousness with a Glasgow Coma Scale (GCS) score of 12. The patient had no previous history of hypertension, epilepsy, or chronic disease, and blood pressure measurements during previous antenatal visits were reported to be normal. On examination, her blood pressure was 151/103 mmHg, with +3 proteinuria. The patient was diagnosed with decreased consciousness et causa antepartum eclampsia. She received magnesium sulfate therapy, antihypertensive treatment, and supportive care. Due to the unstable maternal condition, preterm gestational age, and fetal status, pregnancy termination was performed through cesarean section. The newborn had a very low birth weight and was admitted to the neonatal intensive care unit (NICU). Seizures in eclampsia are associated with cerebral vasospasm, cerebral edema, and impaired autoregulation of cerebral blood flow, which may lead to decreased consciousness. The primary management approach focuses on maternal stabilization, with magnesium sulfate as the anticonvulsant of choice and blood pressure control as essential components of therapy. Delayed treatment may increase the risk of multisystem complications and maternal and perinatal mortality. Eclampsia is an obstetric emergency that requires prompt diagnosis and immediate management.

INTRODUCTION

The term eclampsia originates from the Greek word meaning "like a flash of lightning," describing the sudden onset of this condition without obvious warning signs. In more than 80% of cases, eclampsia is preceded by symptoms of severe preeclampsia. Eclampsia is defined as the occurrence of generalized tonic-clonic seizures and/or coma in women with preeclampsia that cannot be attributed to other neurological disorders (Aya et al., 2016; Edlow et al., 2016; Okikiade et al., 2025). This condition may occur in patients with preeclampsia alone or in those with preeclampsia superimposed on chronic hypertension or chronic kidney disease (Dutta, 2018).

Eclampsia is a pregnancy complication that remains a serious health problem, particularly in developing countries. The incidence of eclampsia has been reported at 1.6–10 cases per 10,000 births in developed countries, whereas in developing countries, the incidence

is substantially higher, ranging from approximately 50 to 151 cases per 10,000 births. The higher incidence in developing countries is associated with limited access to antenatal care, delayed detection of preeclampsia, delays in pregnancy termination when indicated, and limited healthcare resources. This condition contributes to increased maternal and perinatal morbidity and mortality rates (Fishel Bartal et al., 2022).

Eclamptic seizures occur in approximately 2% of women with severe preeclampsia who do not receive magnesium sulfate prophylaxis, whereas in patients receiving magnesium sulfate, the incidence decreases to less than 0.6%. Therefore, magnesium sulfate remains the primary therapy for the prevention and management of eclampsia. The pathogenesis of eclamptic seizures is not yet fully understood; however, the most widely accepted theory involves disruption of the blood–brain barrier, resulting in the movement of fluid, ions, and plasma proteins into the brain parenchyma, leading to cerebral edema, seizures, and decreased consciousness (Amaral et al., 2017; Bergman et al., 2019; Gasparini et al., 2019; Mahendra et al., 2021; Vera-González, 2022). Increased blood–brain barrier permeability is thought to be influenced by circulating factors associated with preeclampsia, including vascular endothelial growth factor and placental growth factor (Fishel Bartal et al., 2022).

Decreased consciousness in eclampsia is primarily associated with central nervous system involvement due to impaired cerebral autoregulation and dysfunction of the blood–brain barrier. In this condition, elevated blood pressure and circulating factors associated with preeclampsia impair the protective mechanisms of cerebral blood vessels, resulting in hyperperfusion and increased vascular permeability (Cipolla et al., 2018; Escudero et al., 2023; Ives et al., 2020; Mahendra et al., 2021; Miller, 2019). Consequently, plasma fluid, ions, and proteins enter the brain parenchyma, causing cerebral edema, which contributes to seizure development and increased intracranial pressure. Cerebral edema and subsequent neuronal injury disrupt cortical and subcortical brain functions, resulting in seizures accompanied by decreased consciousness. This condition is frequently associated with posterior reversible encephalopathy syndrome (PRES) and is generally reversible with appropriate treatment (Dutta, 2018).

Given the significant morbidity and mortality associated with eclampsia, early diagnosis and prompt management are essential to improve maternal and perinatal outcomes. Case reports play an important role in medical education by providing detailed clinical descriptions that enhance understanding of disease presentation, management, and outcomes. This case report aimed to describe the clinical manifestations of decreased consciousness in a patient with antepartum eclampsia, evaluate the appropriateness of the medical and obstetric management provided, and review the clinical implications of eclampsia on maternal and perinatal outcomes. The findings from this case are expected to contribute to the existing knowledge regarding eclampsia management and support clinical practice, particularly in settings where eclampsia remains a significant cause of maternal morbidity and mortality.

METHOD

This study was a descriptive case report presenting a detailed clinical case with educational and scientific relevance. A descriptive qualitative approach with a retrospective case study design was used to trace the patient's clinical course, including the initial presentation, diagnosis, management, and maternal and perinatal outcomes. This approach was

selected because the study aimed to describe the clinical manifestations of decreased consciousness in a patient with antepartum eclampsia and evaluate the appropriateness of the management provided based on current literature.

The data sources consisted of primary and secondary data. Primary data were obtained through direct anamnesis with the patient and alloanamnesis with the patient's husband, along with physical and obstetric examinations. Secondary data were obtained from medical records, including laboratory results, liver function tests, urinalysis, ultrasound findings, emergency department records, cesarean section reports, postoperative monitoring records, and neonatal outcomes. Relevant obstetric textbooks, clinical practice guidelines, and national and international scientific articles were also reviewed as supporting references. Data collection was conducted through clinical observation, interviews, medical record review, and literature review.

The study was conducted in February 2026 at the Obstetrics and Gynecology Department/Medical Staff Unit (SMF) of the Faculty of Medicine, Syiah Kuala University, dr. Zainoel Abidin Hospital, Banda Aceh, Aceh, Indonesia. Case observation began when the patient was admitted to the Emergency Department on February 13, 2026, and continued through the postoperative treatment period.

Data were analyzed descriptively by arranging the patient's clinical information chronologically and systematically. The findings from the anamnesis, physical examination, supporting examinations, and management were compared with relevant theories and previous research findings. The analysis was used to explain the pathophysiological mechanisms of decreased consciousness in eclampsia, evaluate clinical decision-making, and formulate conclusions and recommendations applicable to similar cases. Patient confidentiality was maintained, and the preparation of this case report was conducted with approval from the patient and clinical supervisor.

RESULTS AND DISCUSSION

Case

A 28-year-old female patient, G1P0A0, pregnant 33 weeks 0 days based on 27/06/2025 with TTP 03/04/2026, came to the emergency room with complaints of sudden full-body bulge seizures since 3 hours of SMRS, seizures were felt at home with a frequency of 2 seizures with a 60-minute pause period between seizures. The first seizure occurs at 14.00 for 10 minutes, then around 15.00 the second seizure occurs for 5-10 minutes. The patient is conscious when the seizure occurs. Before the seizure, the patient admitted that he had experienced severe headaches throughout the head, which he felt was gone, with a duration of 30 minutes, and a moderate intensity of NRS 8. Then approximately 30 minutes after that the patient had a seizure. Complaints of heartburn accompanied by nausea are also found before the seizure. Nausea and vomiting are experienced 1x in the form of food that has just been eaten. After the seizure, the patient felt drowsy, and when examined in the hospital's emergency room, the consciousness of the somnolen patient with GCS 12. Complaints of bleeding from the birth canal and discharge of the water are denied. Fetal movements are felt by active patients. The patient admitted that she was pregnant with her first child, the history of miscarriage was denied. The patient admitted that so far she had only checked her pregnancy with Sp. OG 2 times and with a midwife once a month. During the patient's pregnancy control

never high blood pressure, every pregnancy control when checking the patient's blood pressure, according to the patient and her husband the patient's blood pressure range has been normal, the highest blood pressure of the patient during pregnancy is 111/80 mmHg before pregnancy also the patient has never had high blood pressure. The last 1 month of SMRS control and ultrasound stated that the fetus was in good condition, water was sufficient. The previous history of seizures was denied, the seizures were only experienced today. Patients also complained of blurred vision after the last seizure occurred. The history of vaginal discharge is denied. BAK and BOWEL within normal limits.

The patient had no history of hypertension, diabetes mellitus, heart disease, asthma, or allergies. There is no family history of similar diseases. Menarche menstrual history at the age of 12 years, regular cycle, menstrual length 5–6 days, changing pads 2–3 times per day, and not accompanied by dysmenorrhea. The patient was married once at the age of 25 and this was the first pregnancy. The patient works as a housewife with her last education being high school, and her husband works as a self-employed person.

On physical examination, the patient appears to be in a generally good state, with somnolent consciousness. Vital signs showed blood pressure of 151/103 mmHg, pulse of 89 times per minute, breathing rate of 20 times per minute, body temperature of 36.7°C, and oxygen saturation of 99% RA. The patient's weight before pregnancy was 58 kg with a height of 158 cm, resulting in a body mass index of 23.9 kg/m² which is included in the normal category. The patient's weight is currently 65 kg, resulting in a body mass index of 26.1 kg/m² which belongs to the overweight category. On examination of generalist status, the conjunctiva was not anemic and the sclera was not icteric. Regular heart sounds I and II without murmurs or gallops. The sound of vesicular breathing is heard the same on both the right and the left, without wheezing or ronkhi. Limbs feel warm, no edema, or cyanosis, Other physical examinations within normal limits.

On the obstetric status check, the inspection shows the vulva and urethra are calm. On examination of the inspector, it was found that portio livid, OUE was closed, flux (-), fluorine (-) and active bleeding were not found. VT is obtained with livid, posterior, soft and non-perceptible portio. Leopold I, found 33 cm TFU. In Leopold II, a flat and wide part of the fetus is found on the right side of the mother. With DJJ: 148 x/min, His (-). In Leopold III, the fetal part is obtained, hard, round, with the impression of a head. In Leopold IV, it was found that the fetus had not entered the upper door of the pelvis, converging 5/5. The impression of her obstetric status is that a single fetus lives intra uterine with a buttock presentation.

Ultrasound Examination :

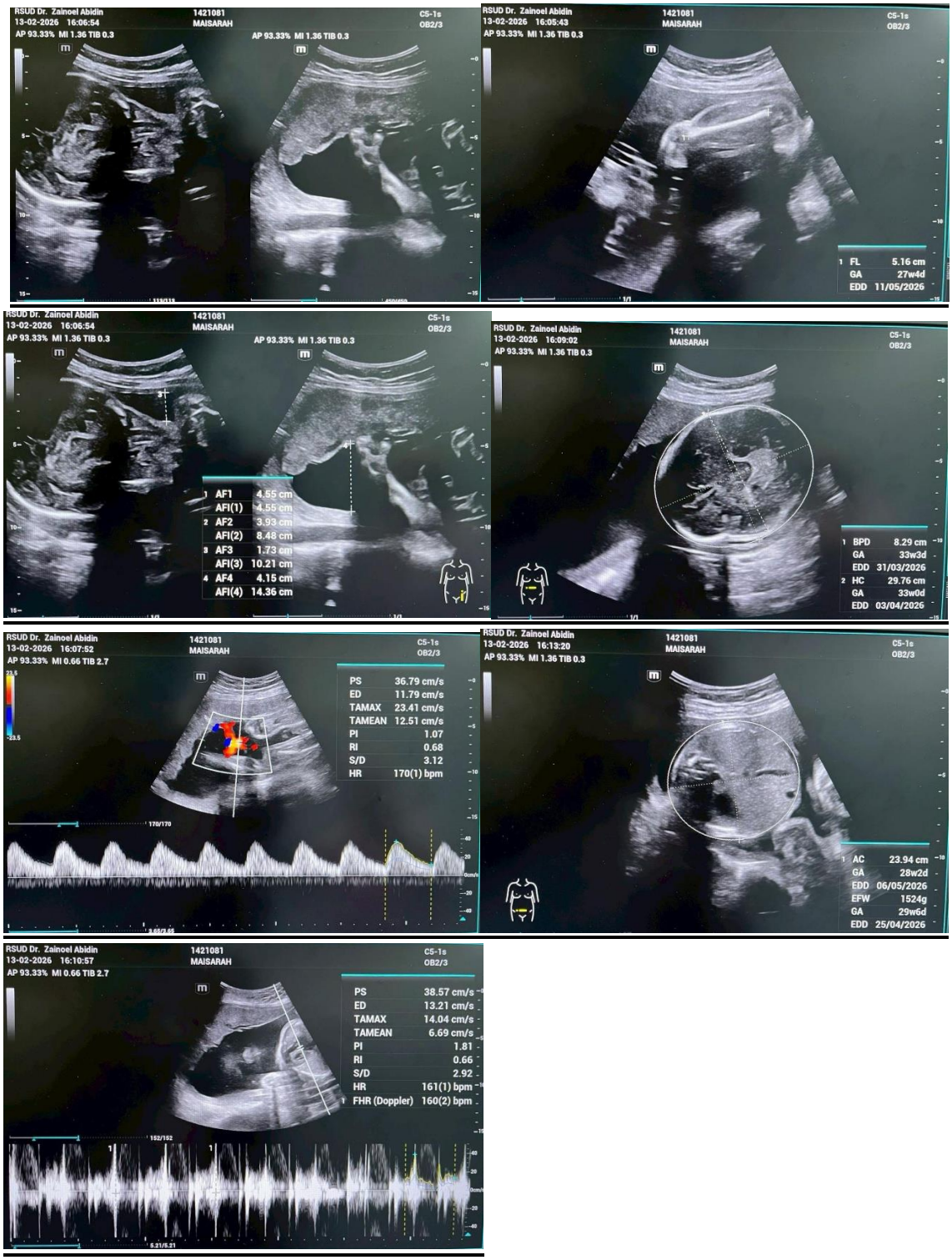


Figure 1. The fetus presents a single buttocks alive, according to the gestation

Ultrasound conclusion: The fetus presents a single buttocks alive, according to the gestation period of 33-34 weeks.

- BPD = 8.29 cm
- HC = 29.76 cm
- AC = 23.94 cm
- FL = 4.16 cm

- EFW = 1524 grams
- AFI = 14.36 cm
- Placenta = anterior corpus

Laboratory tests on February 13, 2026 showed hemoglobin of 12.1 g/dL and hematocrit of 36%, with the number of erythrocytes of 4.2 million/µL, leukocytes of 17,470/µL, and platelets of 159,000/µL. The erythrocyte index was obtained MCV 84 fL, MCH 29 pg, and MCHC 34 g/dL. Count the type of leukocytes shows eosinophils 0%, basophils 0%, rod neutrophils 0%, segment neutrophils 83%, lymphocytes 12%, and monocytes 5%. Liver function checks, SGOT 75 U/L and SGPT 50 U/L. Non-reactive HBsAg and blood sugar at 98 mg/dL. Urinalysis was also examined in this patient was tame, the color of the urine was brownish-yellow, cloudy, with a specific gravity of 1.030, pH 5.0, proteinuria (+3).

Table 1. Laboratory examination

Lab	13/2/26
Hb/ Ht/ E/ L/ T	12,1/36/4,2/17.470/159.000
MCV/MCH/MCHC	84/29/34
E/B/NB/NS/Lim/Mono	0/0/0/83/12/5
SGOT/SGPT	75/50
HBsAg	Non-Reactive
Blood sugar during	98
Urine Color	Brownish yellow
Clarity	Cloudy
BJ/pH/Proteinuria	1,030/5,0/(+3)

The patient was diagnosed with decreased consciousness ec eclampsia. The patient was initially administered in the emergency room where the patient was given RL 500 cc liquid for 6 hours, MgSO4 40% 4 gr extra bolus, and Drip MgSO4 40% 1 gr/hour, IV dexamethasone 12 mg, extra, and Nifedipine 10 mg. Taking into account the unstable maternal condition, the gestational age of 33 weeks, and the presentation of the buttocks, vaginal delivery was not the optimal choice, so it was decided to perform a cesarean section. The patient was then subjected to a C-Section, and was consulted to the Anesthesia department, from anesthesia suggesting that the patient be treated at HCU-S (13/2/2026). The baby was born on February 13, 2026 at 17.50 WIB, with a birth weight of 1,200 grams. The baby cried loudly, the baby was then referred to the Mother and Child Hospital and treated in the NICU, because the baby was diagnosed with Respiratory Distress Syndrome and Very Low Birth Weight (BBLSR). After surgery, patients are planned to monitor vital signs and bleeding every 30 minutes for 2 hours, if stable followed by monitoring every 4 hours. Then, urine monitoring is at least 0.5cc/KgBB/hour, and Hb 6 hours postoperatively, the patient is also given IVFD 2 RL and 1 Dex 10% as much as 100cc/hour, IV oxytocin 40 IU in RL, finished within 4 hours after surgery. After 4 hours of comfortable Oxiytocin, continue with IV MgSO4 1g/hour, maintenance up to 24 hours postpartum, for symptomatic therapy the patient is given IV Ketorolac/8 hours, PO Paracetamol 1000mg/8 hours, IV Omeprazole/12 hours.

The patient was a third trimester pregnant woman with a gestational age of 33 weeks who was diagnosed with decreased ec eclampsia awareness. The patient experienced generalized tonic-clonic seizures twice, each with a duration of about 10 minutes, with an

interval between seizures of ±60 minutes. Before the seizure, patients complain of severe headaches, heartburn, and blurred vision.

From the physical examination, it was found that there was a decrease in consciousness (Somnolen) which was characterized by GCS 13, the patient appeared moderately sick and drowsy, when the blood pressure was checked, the patient was found to have high blood pressure, which was 150/103 mmHg, Blood pressure was previously always within normal limits at routine antenatal visits, with the highest value of 111/80 mmHg. For generalized status, everything is within normal limits. Then, from the ultrasound results, it was obtained that a single fetus lived intrauterine with a presentation of the buttocks, according to the gestational period, and from the supporting examination, there was a slight decrease in hematocrit values of 36%, an increase in leukocytes to 17,470, an increase in liver function, namely AST 75 U/L and ALT 50 U/L, a decrease in albumin of 3.15 g/dL, a decrease in electrolytes, namely calcium and chloride, 7.9 mg/dL and 109 mmol/L, respectively, and increased kidney function, namely creatinine 1.36 mg/dL.

From the symptoms reported by the patient such as the presence of seizures, which were previously preceded by blurred vision, heartburn, and headache. Clinically, almost all previous eclampsia patients showed severe preeclampsia manifestations in the form of symptoms such as severe headaches, visual disturbances, or epigastric pain. Which, all of these symptoms are found in this patient. Eclampsia is defined as the occurrence of generalized tonic-clonic seizures with or without decreased consciousness in mothers with preeclampsia without other neurological causes. In these patients, the enforcement of the diagnosis was supported by recurrent seizures, new onset hypertension, and the absence of a previous history of epilepsy. In mothers with preeclampsia that is not caused by other neurological disorders, which is supported by a denial of a history of previous epilepsy or seizures in the patient. This condition can occur in patients with pure preeclampsia or preeclampsia due to chronic hypertension or chronic kidney disease. The absence of a history of chronic hypertension, kidney disease, or increased blood pressure at previous antenatal visits supports that eclampsia in these patients occurs as a complication of pure preeclampsia. Although seizures are recurrent, patients do not meet the criteria for epileptic status because there is a conscious interval between seizures and the duration of seizures does not persist without recovery (Dutta, 2018).

Eclampsia is still a significant global health challenge, contributing to maternal morbidity and mortality worldwide. Globally, the incidence of eclampsia varies, with developing countries often experiencing higher rates due to limited access to health services. For example, a study in 10 low- and middle-income countries reported the incidence of eclampsia ranged from 19.6 to 142 cases per 10,000 births, with the highest incidence in Sierra Leone. In contrast, countries with more advanced healthcare systems, such as the US and some parts of Europe, generally report lower numbers due to better prenatal care (Syairaji et al., 2024; Ginting et al., 2025; Vousden et al., 2019).

Hypertension is the most common medical complication found during pregnancy. As a significant contributor to maternal and perinatal morbidity and mortality, hypertension is estimated to have been a complication in about 7% to 10% of all pregnancies. Preeclampsia and eclampsia are the cause of 30-40% of perinatal deaths and are one of the obstetric complications that cause a lot of morbidity and mortality in addition to bleeding and infection (Triana & Sa, 2019).

In Indonesia, hypertensive disorders, including eclampsia, are one of the leading causes of maternal death, contributing to the majority of maternal mortality in the country. According to a systematic review, hypertension disorders cause 23% of maternal deaths in Indonesia (Syairaji et al., 2024). Despite efforts to reduce maternal mortality, Indonesia's maternal mortality rate (AKI) has stagnated in recent years, with areas such as Sulawesi reporting AKI as high as 1,811 per 100,000 live births in some studies. Eclampsia also occurs frequently in primigravida and most appear in the 3rd trimester, a theory taken from Dutta's book, according to the circumstances of the patient who has just conceived the first child. And currently the patient's gestational age is 33 weeks (3rd trimester) (Dutta, 2018).

The risk factors of preeclampsia are categorized into low, moderate, and high risk levels, by the American College of Obstetricians and Gynecologists (ACOG). Included in the low-risk category are childbirth with full-term babies. Moderate risk factors are nuliparity, BMI >30 kg/m2, family history with previous preeclampsia, age >35 years, previous pregnancy >10 years or low birth weight. High risk factors, such as multiple pregnancies, previous preeclampsia, long-term hypertension, kidney disease, type 1 or 2 DM, and autoimmune disorders. In this patient, moderate and high risk factors were found to be nulliparity and low birth weight with a BBL of 1,200 grams. Low birth weight babies are babies with a birth weight (BBLR) <2500 g. Very low birth weight babies (BBLSR) are babies with birth weight <1500 g and very low birth weight babies (BBLASR) are babies with birth weight <1000 g (Bakri et al., 2024; Ministry of Health of the Republic of Indonesia, 2018).

Some of the signs and symptoms that precede eclampsia can be a sudden increase in blood pressure, headaches, heartburn, visual and mental changes, fluid retention, and hyperreflexia, photophobia, irritability, nausea and vomiting. To determine for sure the neuropathological condition that triggers seizures, diagnostic examinations such as X-rays, CT scans or MRIs can be carried out (Syairaji et al., 2024). The presence of diffuse cerebral edema will give rise to a picture of seizures in eclampsia (Ginting et al., 2025). Reversible vasogenic edema is the most predominant so that eclampsia almost causes permanent neurology (Andriana, 2022).

Pathophysiology of Eclampsia

Pathophysiologically, eclampsia is closely related to the occurrence of systemic endothelial dysfunction. In women with eclampsia, an increase in various mediators that reflect endothelial activation and damage is found, such as von Willebrand factor, cellular fibronectin, proinflammatory cytokines (interleukin-6 and tumor necrosis factor-α), as well as cell adhesion molecules including ICAM-1, VCAM-1, and P-selectin. The increase in these factors causes impaired endothelial function which plays a role in the occurrence of vasoconstriction, increased capillary permeability, and coagulation activation (Fadhillah & Widyastuti, 2022).

In addition, eclampsia is also associated with disorders of uterovascular development. In normal pregnancy, remodeling of the uteroplastic spiral artery occurs to meet the needs of fetal perfusion. However, in eclampsia this process is inhibited due to abnormal interaction between the mother and the placenta, thus causing uteropplacental perfusion disorders. This condition contributes to the release of circulatory factors that aggravate endothelial dysfunction systemically (Fadhillah & Widyastuti, 2022).

At the level of the central nervous system, eclampsia is characterized by a disruption of the regulation of cerebral blood flow. Severe hypertension that occurs suddenly causes a failure of cerebral self-regulation mechanisms, resulting in hyperperfusion. This condition triggers

dilatation of the cerebral blood vessels and increased permeability of the cerebral blood barrier, which leads to cerebral edema. Cerebral edema can subsequently cause encephalopathy, ischemia, as well as neurological manifestations in the form of seizures and decreased consciousness. Histopathological findings in some cases indicate the presence of fibrinoid necrosis and edema of the endothelium of blood vessels, which support the mechanism (Fadhillah & Widyastuti, 2022).

Endothelial dysfunction in eclampsia is also affected by an increase in antiangiogenic factors, such as vascular endothelial growth factor (VEGF) antagonists, soluble fms-like tyrosine kinase-1 (sFlt-1), and activin A. Increased levels of these proteins lead to decreased VEGF activity, which plays an important role in maintaining endothelial integrity. As a result, protein leakage occurs from blood vessels that triggers generalized edema and aggravates systemic vascular disorders in preeclampsia and eclampsia (Fadhillah & Widyastuti, 2022).

In addition, oxidative stress plays a role in the pathogenesis of eclampsia. Increased leptin levels in pregnant women with eclampsia are known to induce oxidative stress and increase platelet aggregation, thus affecting the coagulation system. Oxidative stress can also affect the production and secretion of antiangiogenic factors such as activin A from endothelial cells and the placenta, which further exacerbates endothelial dysfunction and clinical manifestations of eclampsia (Fadhillah & Widyastuti, 2022).

The main change that occurs in preeclampsia is the presence of blood vessel spasms accompanied by salt and water retention. On the kidney biopsy, a severe spasm of the glomerular arteriola was found. So if all arterioles in the body experience spasms, then blood pressure will rise, in an effort to overcome the increase in peripheral pressure so that tissue oxygen can be sufficient. While proteinuria is caused by arteriole spasm resulting in changes in the glomerulus (Andriana, 2022).

The pathophysiology of preeclampsia-eclampsia is more emphasized towards implantation disharmony and endothelial tissue dysfunction. The end result of the disharmony of blood implantation is the widening of the spiral arteries that were previously thick and muscular to form an elastic, low-resistance and fast-flowing sac, and free from normal neurovascular control, thus allowing adequate flow for the entry of oxygen and nutrients to the fetus. Meanwhile, the definition of endothelial dysfunction itself means a reduction to the loss of the ability of endothelial cells to regulate vasodilation (Andriana, 2022).

If preeclampsia is not treated properly, it can develop into eclampsia which not only harms the mother but also the fetus in the mother's womb. The most severe are maternal and fetal deaths, placental solution, hypofibrinogemia, hemolysis, brain hemorrhage, eye abnormalities, pulmonary edema, liver necrosis, Hemolysis syndrome, Elevated Liver enzyme, and Low Platelets (HELLP), and liver abnormalities (Ministry of Health of the Republic of Indonesia, 2018). Cerebral edema has also been found as a complication of eclampsia (Andriana, 2022). These changes cause maternal organ perfusion to decrease, including perfusion to the fetus-uteroplacental unit. Cyclic vasospasm further lowers the perfusion of the organ by destroying red blood cells, so that maternal oxygen capacity decreases. Genetic predisposition can be another immunological factor. It was found that there was a frequency of preeclampsia and eclampsia in children and grandchildren of women who had a history of eclampsia, indicating an autosomal recessive gene that regulates the maternal immune response (Andriana, 2022).

Stages of Seizures in Eclampsia

The seizures that occur in eclampsia are epileptiform (Dutta, 2018), and consist of four stages, as follows:

1. Premonitory phase, the patient suddenly loses consciousness, accompanied by twitching of the muscles of the face, tongue, and extremities, as well as rotating or fixed eyeballs that last 30 seconds (Dutta, 2018).
2. Tonic phase, stiff spasms of the whole body, flexion extremities, clenched hands, breathing stopped, tongue bites, and the onset of cyanosis lasting 30 seconds (Dutta, 2018).
3. Clonic phase, contraction and relaxation of muscles that take turns starting from the face and spreading throughout the body, accompanied by loud breathing and foamy secretions that can sometimes mix with blood due to tongue bites that last 1-4 minutes (Dutta, 2018).
4. Coma phase, which can last a short time or persist until subsequent seizures (Dutta, 2018).

Therapeutic Management in Eclampsia

Eclampsia management includes prevention, early management, general treatment, and specific therapies. Most cases of eclampsia are preceded by severe preeclampsia, so the prevention of eclampsia mainly depends on early detection and adequate management of preeclampsia in health facilities, including the administration of antihypertensives, anticonvulsant prophylactic therapy, and timely termination of pregnancy. However, eclampsia can occur without being preceded by preeclampsia so it is not always preventable and is often difficult to predict due to atypical clinical manifestations. Close monitoring during labor and up to 24 hours postpartum is essential. The Magpie Trial study (2002) showed that the prophylactic use of magnesium sulfate can reduce the risk of eclampsia, although about 30–85% of cases remain unpreventable (Dutta, 2018).

Initial management outside the hospital emphasizes the importance of immediate referral to a tertiary referral hospital as it is not recommended to continue treatment in peripheral facilities. During the referral process, blood pressure should be controlled and seizures stopped. Magnesium sulfate is given as an initial dose, accompanied by the administration of antihypertensives such as intravenous labetalol to control hypertension. Diuretics are given when pulmonary edema is present. When used, diazepam should be given slowly to prevent apnea or cardiac arrest. Patients must be accompanied by trained medical personnel during transport by ambulance equipped to prevent injuries, recurrent seizures, and airway disturbances (Dutta, 2018).

General medical and nursing management focuses on supportive care, namely prevention of injury due to seizures, prevention of aspiration, maintenance of the airway, and ensuring adequate oxygenation. The patient is treated in a fenced bed, positioned laterally to prevent aspiration, with regular cleaning of oral secretions. Oxygen is administered through a mask with a flow rate of 8–10 L/min and oxygen saturation is monitored using a pulse oximeter. Arterial blood gas analysis is performed when oxygen saturation drops below 92%. The history of the disease was obtained from the family regarding the gestational age, the number of seizures, and the therapy that had been given previously. After the condition is stable, a complete physical examination and the installation of a urinary catheter are carried out for

diuresis monitoring and urine protein examination. Vital signs are monitored regularly, including blood pressure, pulse, breathing rate, and urine production. If the patient has not given birth, the progress of labor and the fetal heart rate must be closely monitored (Dutta, 2018).

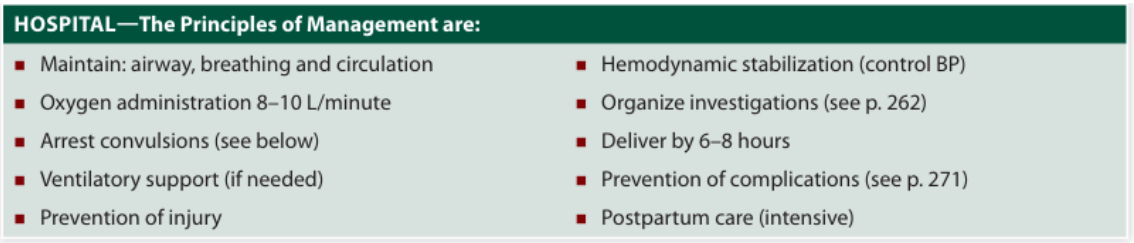


Figure 2. Key Management of Eclampsia while in the Hospital

Fluid management is done carefully to prevent excess fluids. Crystalloid liquids such as Ringer lactate are given as an initial option, with total fluid not exceeding the amount of urine 24 hours prior plus 1000 mL for insensible loss. Generally, the liquid does not exceed 2 liters in 24 hours. Despite the presence of intravascular hypovolemia, the body's tissues are overloaded with fluids so excessive fluid administration can worsen pulmonary edema and acute respiratory distress syndrome. Colloidal use is carried out selectively and invasive hemodynamic monitoring is rarely required. Prophylactic antibiotics such as intravenous ceftriaxone are given to prevent infection (Dutta, 2018).

Specific management of eclampsia is the administration of anticonvulsants with the aim of stopping seizures and preventing recurrence. Magnesium sulfate is the main drug of choice because it is effective and safe. Magnesium sulfate acts as a nerve membrane stabilizer and neuroprotector by decreasing neuromuscular sensitivity to acetylcholine, inhibiting the entry of calcium to neurons, as well as causing cerebral and uteroplacental vasodilation. The drug also increases the production of endothelial prostasicline and inhibits platelet activation. Magnesium sulfate has no adverse effects on the fetus in therapeutic levels and has been shown to significantly reduce maternal mortality rates. However, this drug does not work as an antihypertensive so blood pressure control still requires additional therapy (Dutta, 2018).

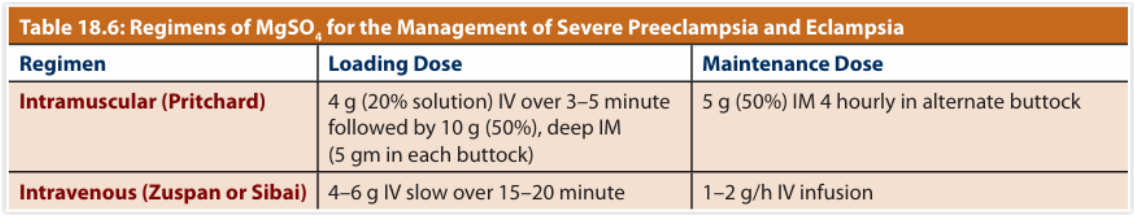


Table 18.6: Regimens of $MgSO_4$ for the Management of Severe Preeclampsia and Eclampsia

Regimen	Loading Dose	Maintenance Dose
Intramuscular (Pritchard)	4 g (20% solution) IV over 3–5 minute followed by 10 g (50%), deep IM (5 gm in each buttock)	5 g (50%) IM 4 hourly in alternate buttock
Intravenous (Zuspan or Sibai)	4–6 g IV slow over 15–20 minute	1–2 g/h IV infusion

Figure 3. MgSO4 Regimen for the Management of Eclampsia and Preeclampsia

Monitoring and therapy of magnesium sulfate should be strictly carried out to prevent toxicity. Redose of magnesium sulfate is only done when the patella reflex is still present, the respiratory rate is more than 12 times per minute, and urine production exceeds 30 mL per hour. The therapeutic level of serum magnesium that is effective for controlling seizures is 4–7 mEq/L (4.8–8.4 mg/dL). Serum magnesium levels are checked in certain cases such as renal

insufficiency or when the tendon reflex disappears. Signs of magnesium toxicity include loss of the deep tendon reflex at levels greater than 7 mEq/L, respiratory depression at levels above 10 mEq/L, and cardiac arrest at levels greater than 25 mEq/L. Magnesium sulfate is continued for 24 hours after the last seizure or after delivery, whichever is slower. In case of relapse, an additional bolus of magnesium sulfate 2 g intravenously is given for 5 minutes. If seizures persist despite adequate magnesium therapy, midazolam may be given 1–2 mg intravenously and repeated if necessary (Dutta, 2018).

Magnesium toxicity monitoring was carried out by assessing deep tendon reflexes, breathing frequency, urine volume, and the presence of complaints of chest pain or heart rhythm disorders. If magnesium toxicity occurs, magnesium therapy must be stopped immediately, serum magnesium and creatinine levels are checked, and antidotum in the form of calcium gluconate 10 mL 10% solution intravenously (Dutta, 2018).

Monitoring of Magnesium Toxicity	Management for Magnesium Toxicity
▪ Loss of deep tendon reflexes	♦ Stop magnesium therapy
▪ Decreased respiratory rate (<12 per minute)	♦ Estimation of serum magnesium and creatinine levels
▪ Urine output (< 30 mL/h)	♦ Injection calcium gluconate 10 mL (10% solution), IV
▪ Chest pain, heart block	

Figure 4. Magnesium Toxicity Monitoring and Management

Alternative anticonvulsant therapies that have been used include lytic cocktail, diazepam, and phenytoin, but compared to other regimens, magnesium sulfate has been shown to be superior because it is effective in controlling seizures without causing depression in mothers and neonates, lowering the risk of recurrent seizures, lowering maternal mortality, and lowering perinatal mortality (Dutta, 2018).

Hypertension control is necessary when blood pressure remains ≥160/110 mmHg even though anticonvulsant therapy has been given. First-line antihypertensives are labetalol and hydralazine, with systolic blood pressure targets of 140–160 mmHg and diastolic 90–100 mmHg. Labetalol is administered intravenously with an initial dose of 20 mg and can be repeated every 10 minutes when needed. An alternative is 5–10 mg intravenous hydralazine with repetition after 20 minutes if the response is inadequate. Diuretics, especially furosemides, are given when pulmonary edema is present (Dutta, 2018).

Management during seizures includes the installation of mouth gags in the premonitory phase to prevent tongue bites, clearing the airway from secretions, positioning the head to one side, and giving oxygen until the signs of cyanosis disappear. In the eclampticus status, thiopental sodium can be administered intravenously slowly under the supervision of an anesthesiologist. If that fails, general anesthesia with muscle relaxants and auxiliary ventilation may be performed. In unresponsive cases, cesarean section can be a life-saving measure (Dutta, 2018).

Indications of intubation include decreased postoperative consciousness, uncontrolled seizures, signs of aspiration, and persistent hypoxia. Complications such as pulmonary edema, heart failure, anuria, hyperthermia, and psychosis are treated according to their respective protocols. Patients with multiorgan dysfunction require intensive care in the ICU with a multidisciplinary approach (Dutta, 2018).

Obstetric management in eclampsia is carried out after the mother's condition is stable. In most cases of antepartum eclampsia, labor usually begins spontaneously after a seizure. If labor has not yet begun, the decision to terminate the pregnancy is determined by the success of the seizure control and the level of fetal maturity. When the seizures are controlled and the fetus is mature, labor must be carried out immediately. In favorable cervix and without vaginal labor contraindications, an amniotomy is performed with or without oxytocin augmentation. If the cervix is immature, cervical maturation is carried out using prostaglandins before amniotomy. Cesarean section is performed when the cervix is unfavorable or there are other obstetric indications. In premature fetuses, delivery is still recommended in a neonatal intensive care unit facility because the process of preeclampsia–eclampsia disease will only stop after the pregnancy ends, and there is a risk of recurrent seizures and intrauterine fetal death. Corticosteroids are recommended when the gestational age is less than 34 weeks. When the fetus has died, the disease process usually gradually improves, but induction of labor can be carried out if spontaneous labor does not occur (Dutta, 2018).

If the seizures cannot be controlled within 6–8 hours despite adequate anticonvulsant therapy, termination of pregnancy should be carried out immediately. During childbirth, if there are no contraindications, an early amniotomy is performed to speed up labor. Period II is shortened with operative measures when necessary. Giving uterotonics that can increase blood pressure, such as ergometrin, should be avoided; instead oxytocin is given to prevent postpartum bleeding. Cesarean section is indicated in uncontrolled seizures, the patient is unaware of the poor prospect of vaginal delivery, or the presence of obstetric indications such as malpresentation (Dutta, 2018).

Follow-up and prognosis include postnatal evaluation for up to 6 weeks. The persistence of hypertension, proteinuria, or biochemical abnormalities requires further examination and consultation with an internal medicine. The next pregnancy is recommended to be postponed until the maternal condition is under control. The risk of recurrence of eclampsia ranges from 2–25%. Atypical preeclampsia can occur without meeting the classic criteria of hypertension or proteinuria, such as in early onset before 20 weeks, advanced postpartum onset, or in patients with hypertension or gestational proteinuria with signs of severity. The case should be managed like severe preeclampsia, including parenteral magnesium sulfate (Dutta, 2018).

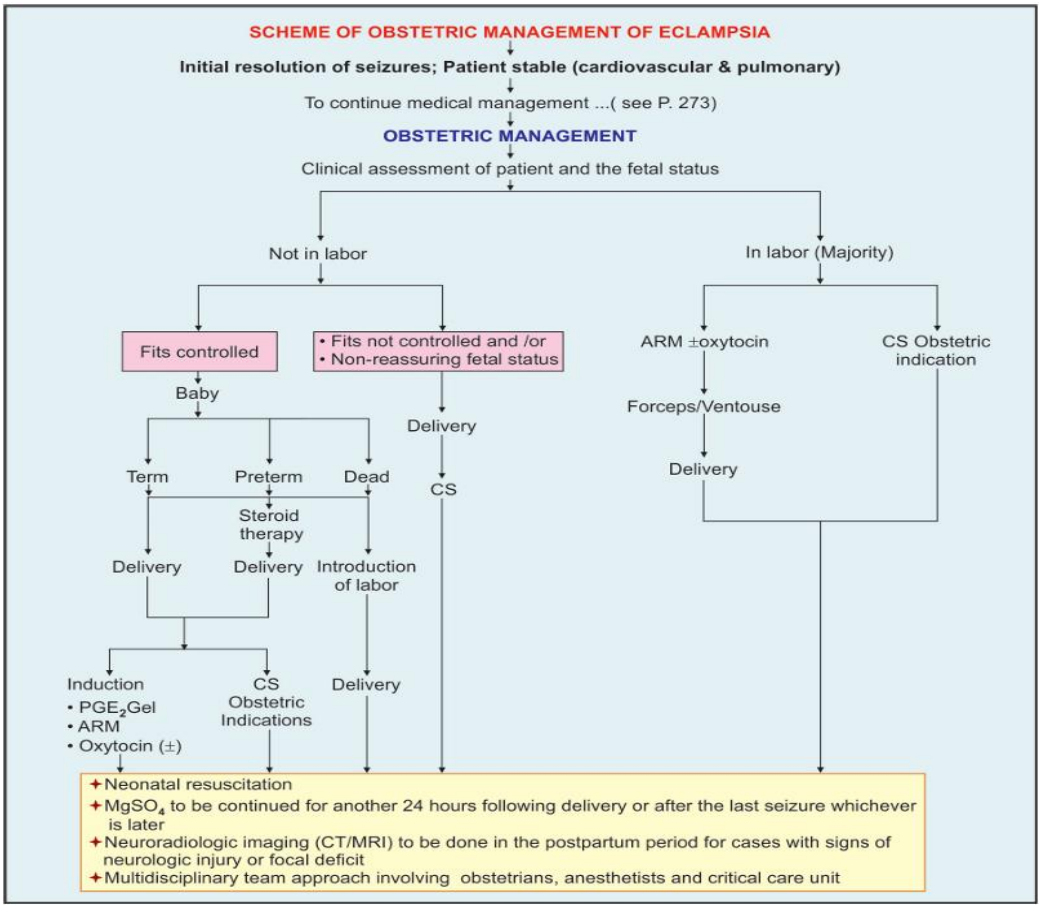


Figure 5. Eclampsia Obstetric Management Scheme

Prognosis and Complications of Eclampsia

The maternal prognosis will worsen if there is a long interval between the onset of the seizure and the start of therapy, antepartum eclampsia especially when accompanied by a long interval of labor, the number of seizures more than ten times, the presence of a coma between seizures, body temperature above 102°F with a pulse rate of more than 120 times per minute, systolic blood pressure exceeding 200 mmHg, oliguria less than 400 mL per 24 hours with proteinuria of more than 5 g per 24 hours, no response to treatment, and the presence of jaundice. The maternal mortality rate due to eclampsia in Indonesia is still high, ranging from 2–30%, with higher rates in rural hospitals than in urban areas. However, with prompt and adequate treatment, the mortality rate can be reduced to less than 2%. Causes of maternal mortality include heart failure, pulmonary edema, aspiration and/or sepsis pneumonia, cerebral hemorrhage, acute renal failure, cardiac and pulmonary arrest, acute respiratory distress syndrome (ARDS), pulmonary embolism, postpartum shock, and puerperalis sepsis (Dutta, 2018).

Maternal complications are more commonly found in antepartum eclampsia. After passing the acute phase, most patients experience an improvement in condition within 2-3 weeks. Recurrence of eclampsia in subsequent pregnancies is rare, but the risk of preeclampsia is about 30%. As for the fetal prognosis in eclampsia, it is poor with a high perinatal mortality rate, which is around 30–50%. The main causes of perinatal death are prematurity, both

spontaneous and iatrogenic, as well as intrauterine asphyxia due to placental insufficiency caused by placental infarction, retroplacental hemorrhage, and vascular spasms in the uteroplacental circulation (Dutta, 2018).

CONCLUSION

Based on the clinical synthesis of this case, a 28-year-old primigravida at 33 weeks of gestation experienced antepartum eclampsia characterized by recurrent generalized tonic-clonic seizures accompanied by decreased consciousness, new-onset hypertension, and severe proteinuria, without a history of neurological disease or chronic hypertension. Prodromal manifestations, including severe headache, epigastric pain, and visual disturbances preceding seizures, were consistent with severe preeclampsia progressing to eclampsia. Laboratory findings, including elevated liver enzymes and +3 proteinuria, supported the diagnosis of eclampsia as part of the spectrum of hypertensive disorders of pregnancy. Pathophysiologically, the condition was most plausibly explained by systemic endothelial dysfunction and impaired cerebral autoregulation, resulting in cerebral hyperperfusion, increased blood–brain barrier permeability, and cerebral edema. These mechanisms contributed directly to seizure occurrence and decreased consciousness, which can be reversible with prompt and appropriate treatment. The absence of major risk factors, such as chronic hypertension or previous kidney disease, suggests that eclampsia may occur acutely even in pregnancies with a previously normotensive antenatal history.

The decision to terminate the pregnancy through cesarean section was an appropriate and rational management step, considering the unstable maternal condition, preterm gestational age, and breech fetal presentation. Comprehensive management, including magnesium sulfate administration as the primary anticonvulsant therapy, blood pressure control, intensive postpartum care, and close maternal and neonatal monitoring, reflected the principles of eclampsia management according to obstetric practice standards. The neonatal outcome of a very low birth weight infant with respiratory distress demonstrated the impact of eclampsia on uteroplacental perfusion and the need for intensive neonatal care.

The important clinical implication of this case is the need for heightened vigilance toward the signs and symptoms of severe preeclampsia, even in patients with a history of normal blood pressure during pregnancy. Antenatal screening should focus not only on blood pressure measurements and proteinuria assessment but also on evaluating subjective symptoms, such as severe headache, visual disturbances, and epigastric pain, which may indicate early central nervous system involvement. Magnesium sulfate administration remains the gold standard for the prevention and management of eclampsia seizures and should be accompanied by careful monitoring for signs of toxicity. Management of eclampsia requires a multidisciplinary approach involving obstetricians, anesthesiologists, intensive care specialists, and neonatologists to optimize maternal and perinatal outcomes. In addition, decisions regarding the timing and mode of delivery should prioritize maternal stability because the progression of the preeclampsia–eclampsia disease process can only be definitively resolved after delivery.

Regarding long-term prevention, patients with a history of eclampsia require postpartum counseling on the risk of recurrence in subsequent pregnancies, the importance of pregnancy planning, and regular monitoring of blood pressure and organ function. Future

pregnancies should be planned after maternal conditions have stabilized, with close antenatal supervision initiated from early gestation. Early detection, timely referral, and appropriate referral-based management within healthcare systems are key strategies for reducing maternal and perinatal morbidity and mortality associated with eclampsia.

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