Management and Consideration of Anesthesia Procedures in Eclampsia: A Case Report

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1. Introduction

Eclampsia, like a powerful storm that hits pregnancy, covers the mother and fetus with the shadow of serious complications, even death. Behind its seemingly simple symptoms - high blood pressure, proteinuria, and edema - there is a complexity of pathophysiology that has not yet been fully unraveled. Even though research continues, eclampsia is still a frightening threat with maternal mortality rates reaching 1-2% in developing countries and 0.05-0.1% in developed countries. This figure becomes even more frightening when accompanied by the risk of fetal death which reaches 20-25%. More than just gestational hypertension, eclampsia is an extreme manifestation of preeclampsia, characterized by tonic-clonic seizures and/or coma that occur in pregnant women after 20 weeks of gestation. This storm not only the cardiovascular system but also attacks the central nervous system, kidneys, liver, and hematology. The pathophysiology of eclampsia is still shrouded in mystery, but several theories have been proposed, such as endothelial dysfunction, vasospasm, brain ischemia, and excessive inflammatory activation. This complexity makes prediction and prevention difficult, making eclampsia one of the most challenging complications of pregnancy.1-3

Management of eclamptic patients often requires immediate delivery, either through spontaneous delivery or caesarean section. The choice of anesthetic technique in eclamptic patients is a crucial dilemma, where the balance between maternal and fetal safety must be the main priority. Regional anesthesia, such
as epidural and spinal, offers several advantages in the delivery of eclamptic patients. This technique provides effective analgesia, allows spontaneous labor, and minimizes the use of systemic anesthetic drugs, thereby reducing the risk of respiratory depression and drug transfer to the fetus. However, regional anesthesia is not without risks. Arterial hypotension, one of the main complications, can occur due to nerve block-induced vasodilation. This can harm the mother and fetus, especially if accompanied by hypovolemia or coagulopathy. Coagulopathy, a common complication of eclampsia, may exacerbate the risk of epidural or spinal hematoma. Unwanted extension of the nerve block, although rare, can delay labor and increase the risk of maternal and fetal complications. Nerve block failure may also occur, forcing a switch to general anesthesia. General anesthesia, on the other hand, offers effective seizure control and prevents aspiration in eclamptic patients. Rapid and controlled induction of general anesthesia can minimize the risk of recurrent seizures and aspiration of vomit. However, general anesthesia also has risks that need to be considered. Arterial hypotension, similar to regional anesthesia, may occur due to anesthetic drug-induced vasodilation. Respiratory depression, another serious complication, can result from inhalation and intravenous anesthetics. Transfer of anesthetic drugs to the fetus is also a major concern, especially in the third trimester of pregnancy. Anesthetic drugs can affect fetal physiology and potentially cause neonatal respiratory depression. Anesthesia plays a crucial role in ensuring the safety and smoothness of the birth process, minimizing the risk of complications, and delivering the mother and fetus to safety.\textsuperscript{4-6}

### 2. Case Presentation

A 21-year-old primigravida (G1P000 39 weeks with estimated fetal weight 2848 grams) was brought by an ambulance with her family with the chief complaint being a seizure. The seizure occurred two times for around 5 minutes and after the seizure, the patient wasn’t fully alert. During transport by ambulance, the seizure occurred again around 2 – 3 minutes, and after the seizure patient wasn’t fully alert. The patient was diagnosed with hypertension during pregnancy in the 24\textsuperscript{th} week and didn’t take any medication. The patient did antenatal care at midwife (3 times) and obstetric gynecologic specialist (1 times). After arriving at the emergency ward, the vital sign was checked with the results: Blood pressure 170/110 (MAP 130 mmHg), heart rate 98 times/minute, respiratory rate 26 times/minute, temperature 36.8°C, and oxygen saturation 98 \% using a non-rebreathing mask with flow 15 L/minute. Other physical and obstetric exams are within normal limits. Due to decreased consciousness, the patient was consulted by an Anesthesiologist for intubation and caesarian section. At the emergency ward the patient was given MgSO\textsubscript{4} 40\% 4mg IM with maintenance 6 mg MgSO\textsubscript{4} 40 \% in ringer lactate solution with 28 drops/minute, Nifedipine 10 mg every 8 hours, and Ceftriaxone 2 gr IV. Complete blood count, urinalysis, blood chemistry test, and LDH test was done. Chest X-ray within and complete blood count within normal limit.

The patient was intubated at the emergency ward using Propofol 150 mg and Midazolam 1 mg as induction with a 7,5 number cuffed endotracheal tube and connected to the ventilator with the setting. General anesthesia was chosen and the patient was given ondansetron 8 mg as premedication and Atracurium 10 mg and Fentanyl 100 mcg during operation. Anesthesia was maintained with O\textsubscript{2} + N\textsubscript{2}O (2 : 2) and Sevoflurane 1\%. After delivery of the fetus, oxytocin infusion was started. The patient was given Fentanyl 300 mcg of 50 cc Nacl with an infusion drip of 2,1 cc/hour using a syringe pump and Paracetamol 3 x 1 gr IV for analgesia. The patient received 200 mL of Ringer’s lactate intraoperatively. At the end of the surgery, the patient was shifted to the ICU with condition unreversed and on a ventilator with mode PCBipap, PC 18, PS 16, RR=14, PEEP 5, Fio\textsubscript{2} = 40\%, and TV = 450-500mL. Due to sinus tachycardia (144 beats/minute), a consultation with a cardiologist was done and the patient was prescribed bisoprolol 1 x 1, 25mg. Over the course of one day, patients’ vitals stabilized and extubated on the second day postoperative day and transferred to the general ward.
3. Discussion

Eclampsia, a life-threatening complication of pregnancy, is characterized by severe arterial hypertension, proteinuria, and edema. Behind these striking clinical manifestations lies a complex physiological storm, with arterial hypotension being one of the main complications. Hypotension in eclampsia is caused by massive systemic vasodilation, triggered by the release of various inflammatory and vasoactive mediators. The biological mechanisms underlying hypotension are complex and multifactorial, involving multiple organ systems and physiological processes. Inflammatory and vasoactive mediators play a key role in vasodilation in eclampsia. Nitric oxide (NO), prostacyclin (PGI₂), and adrenaline are some of the main mediators involved. Nitric oxide (NO), produced by endothelial cells, is a powerful vasodilator that causes the relaxation of vascular smooth muscle. Elevated NO levels in eclampsia are associated with systemic vasodilation and hypotension. Prostacyclin (PGI₂) is also produced by endothelial cells and is a vasodilator and inhibitor of platelet aggregation. Elevated PGI₂ levels in eclampsia contribute to vasodilation and hypovolemia. Adrenaline, a stress hormone released by the adrenal glands, can cause vasodilation through stimulation of beta-adrenergic receptors. In eclampsia, increased adrenaline levels can worsen vasodilation and hypotension. In addition to inflammatory and vasoactive mediators, several other factors may contribute to hypotension in eclampsia. Fluid leak into the interstitial space due to vasodilation and increased capillary permeability causes hypovolemia, which worsens hypotension. Endothelial dysfunction, characterized by damage to endothelial cells, can impair vasoconstrictor production and exacerbate vasodilation. A decrease in circulating blood volume due to hypovolemia and hemodilution (decreased hemoglobin concentration) can cause hypotension. Both anesthetic techniques, regional and general, can contribute to hypotension in eclamptic patients. Regional anesthesia can cause systemic vasodilation through sympathetic nerve block, which reduces vascular resistance and increases peripheral blood flow. Regional anesthesia can cause hypovolemia through diuretic effects and redistribution of fluid from the intravascular space to the interstitial space. General anesthetics such as propofol and sevoflurane can cause systemic vasodilation through direct vasodilator effects on vascular smooth muscle. General anesthetics can depress the sympathetic nervous system, which reduces vascular resistance and exacerbates vasodilation. Scientific research has supported the role of inflammatory and vasoactive mediators, hypovolemia, endothelial dysfunction, and reduced circulating blood volume in eclamptic hypotension. An observational study of 50 eclamptic patients found that NO and PGI₂ levels were significantly higher in patients with hypotension compared with patients without hypotension. An experimental study in a rat model of eclampsia demonstrated that hypovolemia and endothelial dysfunction contribute to hypotension. A meta-analysis of 10 clinical studies found that regional anesthesia increased the risk of hypotension in eclamptic patients compared with general anesthesia.⁸⁻¹²

Eclampsia, a serious complication of pregnancy, is characterized not only by hypertension, proteinuria, and edema but also by complex dysfunction of the coagulation system. Coagulopathy in eclampsia, often called consumptive coagulopathy, is a complex syndrome involving various factors and biological mechanisms. Platelets, a key element in blood clotting, undergo excessive activation in eclamptic patients. It is caused by a variety of factors, including vascular endothelial damage, release of platelet microparticles, and activation of the intrinsic coagulation pathway. This abnormal platelet activation leads to excessive platelet consumption and thrombotic microangiopathy, which contributes to coagulation factor deficiencies and fibrinolysis. Excessive platelet consumption and thrombotic microangiopathy in eclampsia lead to deficiencies of various coagulation factors, including factor V, factor VII, factor VIII, and fibrinogen. A deficiency of these factors disrupts the blood clotting process and increases the risk of bleeding. Fibrinolysis, the process of breaking down fibrin (a protein that forms blood clots), is also increased in eclampsia patients. This is caused by
excessive activation of plasminogen activator inhibitor-1 (PAI-1) and deficiency of antithrombin III. Uncontrolled fibrinolysis can dissolve newly formed blood clots and make bleeding worse. Coagulopathy in eclampsia increases the risk of massive bleeding during labor and cesarean section. Postpartum hemorrhage, a serious complication that can be life-threatening, is more common in eclampsia patients. It is caused by a combination of coagulopathy, drug-induced vasodilation, and birth trauma. Regional anesthesia, especially epidural and spinal, can worsen coagulopathy in eclamptic patients. The local anesthetic used in regional anesthesia has an anticoagulant effect which can interfere with the blood clotting process. Epidural or spinal hematoma, a rare complication of regional anesthesia, can cause bleeding in the epidural or subarachnoid space. This bleeding can worsen coagulopathy and be fatal. The choice of anesthetic technique in eclamptic patients must carefully consider the risks and benefits. Regional anesthesia offers several advantages, such as effective analgesia and minimizing the use of systemic anesthetic drugs. However, the risk of exacerbated coagulopathy should be carefully considered. General anesthesia, on the other hand, offers effective seizure control and prevents aspiration. However, general anesthesia also carries a risk of coagulopathy, although the risk is generally lower than regional anesthesia. The final decision regarding anesthetic technique in eclamptic patients must be made on an individual basis taking into account various factors, including maternal condition, coagulation status, and experience of the anesthesia team. Consultation with a hematologist may be necessary to assess the risk of coagulopathy and determine the safest anesthetic option.13-15

Eclampsia, a serious complication of pregnancy, is characterized by hypertension, proteinuria, and edema. Seizures, one of the most serious complications of eclampsia, are a neurological manifestation of brain dysfunction. Seizures occur due to excessive abnormal electrical discharges in brain neurons. Factors contributing to seizures in eclampsia include hypertension, cerebral ischemia, and cerebral edema. Seizures in eclampsia are the result of a complex interaction between various biological factors. Severe arterial hypertension in eclampsia causes mechanical stress on the cerebral vessels, which can disrupt blood flow and cause cerebral ischemia. Brain ischemia triggers the release of excitatory neurotransmitters, such as glutamate, which increases seizure activity in the brain. Eclampsia is often associated with brain ischemia, reduced blood flow to the brain. Brain ischemia can occur due to vasospasm, hypovolemia, and cerebral edema. Lack of oxygen and nutrients in the brain triggers neuronal dysfunction and increases seizure activity. Cerebral edema, a buildup of water in the brain, is a common complication in eclampsia. Cerebral edema increases intracranial pressure and causes cerebral ischemia, which in turn triggers the release of excitatory neurotransmitters and seizure activity. Endothelial dysfunction, damage to the cells lining blood vessels, is an important factor in the pathophysiology of eclampsia. Endothelial dysfunction causes inflammation and platelet activation, which can worsen brain ischemia and trigger seizures. Eclampsia is associated with changes in levels of neurotransmitters in the brain, especially glutamate and GABA. Glutamate, an excitatory neurotransmitter, increases, while GABA, an inhibitory neurotransmitter, decreases. An imbalance of these neurotransmitters increases seizure activity. General anesthesia is the main choice for controlling seizures in eclampsia patients. General anesthetic drugs such as propofol and barbiturates work by suppressing nerve transmission in the brain, thereby reducing seizure activity. General anesthetic drugs work by modulating ion channels in nerve cell membranes. These ion channels are responsible for the flow of ions into and out of cells, which is important for nerve transmission. General anesthetic drugs can open potassium ion channels and close calcium ion channels, thereby reducing seizure activity. General anesthetic drugs also work by depressing the central nervous system (CNS). The CNS consists of the brain and spinal cord and is responsible for consciousness, sensation, and movement. General anesthetics can inhibit neuronal activity in the CNS, thereby reducing seizure activity.
and inducing sedation. General anesthetic drugs have anticonvulsant effects, which means they can prevent or control seizures. These anticonvulsant effects are due to a combination of ion channel modulation and CNS depression. Several studies have shown the effectiveness of general anesthesia in controlling seizures in eclamptic patients. One study found that propofol, a general anesthetic, was effective in controlling seizures in eclamptic patients with minimal side effects. Another study found that barbiturates, another type of general anesthetic drug, were also effective in controlling seizures in eclamptic patients.

Seizures in eclampsia are a serious complication that requires immediate medical treatment. General anesthesia is the main choice for controlling seizures in eclamptic patients. General anesthetic drugs such as propofol and barbiturates work by suppressing nerve transmission in the brain, thereby reducing seizure activity. Scientific research supports the use of general anesthesia to control seizures in eclamptic patients.15-17

Respiratory depression, a serious complication that can occur with both regional and general anesthesia techniques, is one of the main concerns in the management of eclampsia. In eclamptic patients, respiratory depression can worsen the condition and be fatal. In regional anesthesia, respiratory depression can occur due to intercostal nerve block which causes respiratory muscle paralysis. The intercostal nerves, which innervate the intercostal muscles, play an important role in respiratory mechanisms. This nerve block can interfere with the transmission of nerve impulses to the respiratory muscles, causing muscle weakness or paralysis. In general anesthesia, respiratory depression can occur due to the sedative effect of the anesthetic drug on the respiratory center in the brainstem. The respiratory center in the brainstem is an important area that controls the rhythm and depth of breathing. Anesthetic drugs, such as opioids and barbiturates, can suppress the activity of the respiratory center, thereby slowing or stopping breathing. Hypovolemia, which frequently occurs in eclamptic patients, can exacerbate the hypotensive effects of anesthetic agents and exacerbate respiratory depression. Obesity can increase the risk of respiratory depression by reducing lung compliance and disrupting respiratory mechanisms. Eclamptic patients with underlying lung disease, such as asthma or chronic bronchitis, have a higher risk of respiratory depression during anesthesia. Use of sedatives or opioids may increase the risk of additive respiratory depression with anesthetic agents. A meta-analysis study examining 28 studies found that respiratory depression occurred in 0.5% of eclamptic patients receiving regional anesthesia. Risk factors associated with respiratory depression include high nerve block, obesity, and lung disease. A cohort study examining 1,000 eclamptic patients receiving general anesthesia found that respiratory depression occurred in 2.5% of patients. Risk factors associated with respiratory depression include hypovolemia, obesity, and the use of sedatives. Close monitoring of respiratory function during anesthesia is essential to detect early respiratory depression. In cases of severe respiratory depression, mechanical ventilation may be necessary to assist the patient's breathing. Antagonist drugs, such as naloxone for opioids and flumazenil for benzodiazepines, can be used to reverse the sedative effects of anesthetic drugs and help restore breathing.

Administering anesthesia to pregnant patients is a complex clinical dilemma. On the one hand, anesthesia is necessary to control pain and anxiety during medical procedures, including childbirth and cesarean sections. On the other hand, the transfer of anesthetic drugs to the fetus through the placenta can pose significant risks to fetal health, including neonatal respiratory depression, bradycardia, and even death. The transfer of anesthetic drugs to the fetus occurs through the placenta, a vital organ that connects the mother and fetus. The placenta consists of chorionic villi, finger-like structures, which are surrounded by maternal sinuses containing maternal blood. Fat-soluble anesthetic drugs can diffuse across
the chorionic villus membrane from high concentrations in the maternal blood to low concentrations in the fetal blood. Certain anesthetic drugs can be actively transported across the placenta by transporter proteins. High uteroplacental blood flow can increase the transfer of anesthetic drugs to the fetus. Anesthetic drugs with low molecular weight, high-fat solubility, and low protein binding levels more easily cross the placenta. As pregnancy progresses, the permeability of the placental membrane increases, thereby increasing the transfer of anesthetic drugs. High uteroplacental blood flow, as in multiple pregnancies or preeclamptic gestation, may increase the transfer of anesthetic drugs. Neonatal respiratory depression is the most common complication associated with anesthetic drug transfer. This is due to the depressant effect of anesthetic drugs on the fetal respiratory center. Bradycardia, or slow fetal heart rate, can occur due to the cardio-depressant effects of anesthetic drugs. In rare cases, excessive transfer of anesthetic medication can cause fetal death. Scientific research has shown that the transfer of anesthetic drugs to the fetus can have significant effects on fetal physiology. Pharmacological studies have examined the transfer of individual anesthetic drugs and their effects on the fetus. Clinical studies have evaluated the impact of anesthetic drug transfer on labor outcomes and neonatal health. Giving anesthesia to pregnant patients must consider the balance between the benefits of anesthesia for the mother and the risk of transferring anesthetic drugs to the fetus. Selection of appropriate anesthetic drugs, careful maternal and fetal monitoring, and appropriate neonatal resuscitation interventions can help minimize risks and ensure maternal and fetal safety.17-19

Regional anesthesia, such as epidural and spinal, offers some interesting biological benefits in eclamptic patients. This technique allows spontaneous labor and minimizes the use of systemic anesthetic drugs, thereby reducing the risk of respiratory depression and drug transfer to the fetus. Regional anesthesia works by blocking sensory and motor nerves at the spinal level, producing effective analgesia and minimizing the need for systemic anesthetic drugs. This can help reduce the risk of respiratory depression, a serious complication that can occur in eclampsia patients, especially those who already have respiratory problems due to pulmonary edema or pulmonary hypertension. Studies have shown that regional anesthesia can reduce the need for inhaled anesthetic drugs by up to 50% compared with general anesthesia in eclamptic patients. This has a positive impact on the patient’s respiratory function, with studies showing that regional anesthesia is associated with better spontaneous ventilation and a lower risk of postoperative intubation. Transfer of anesthetic drugs to the fetus through the placenta is a major concern in anesthesia in pregnant patients. Anesthetic drugs can affect fetal physiology, including neonatal respiratory depression and bradycardia. Regional anesthesia, with its use of less local anesthetic drugs and minimization of systemic anesthetic drugs, offers advantages in terms of drug transfer to the fetus. Research has shown that levels of anesthetic drugs in fetal blood are lower in regional anesthesia compared to general anesthesia. This shows that regional anesthesia can help protect the fetus from the side effects of anesthetic drugs. Although regional anesthesia offers some biological benefits in eclamptic patients, it is important to consider the risks associated with this technique. One of the main risks is arterial hypotension, which can occur due to nerve block-induced vasodilation. This can be a serious concern in eclamptic patients who often experience hypovolemia and arterial hypertension. Careful fluid management and the use of vasopressors may be necessary to prevent hypotension in eclamptic patients receiving regional anesthesia. Close hemodynamic monitoring is also important to ensure patient safety. Coagulopathy, a common complication in eclampsia, is another risk associated with regional anesthesia. The anticoagulant effects of local anesthetics and the risk of epidural or spinal hematoma may exacerbate pre-existing coagulopathy in eclamptic patients. Therefore, regional anesthesia should be performed with caution in eclamptic patients with coagulopathy. Preoperative coagulation checks and close monitoring during the procedure are essential. Several studies have examined the use of regional anesthesia in eclamptic patients. The results of the study suggest that regional
anesthesia may be a safe and effective option for eclamptic patients, with significant benefits in terms of minimal respiratory depression, lower transfer of the drug to the fetus, and possible spontaneous delivery. A meta-analysis study examining 14 studies with a total of 812 eclamptic patients found that regional anesthesia was associated with a lower risk of respiratory depression and higher neonatal Apgar scores compared with general anesthesia. Another study involving 60 eclamptic patients showed that regional anesthesia was associated with lower fetal blood levels of anesthetic drugs and a lower need for postoperative intubation compared with general anesthesia. This study provides scientific evidence that supports the use of regional anesthesia in eclamptic patients. However, it is important to note that this study has limitations, such as small sample size and heterogeneity in methodology. The choice of anesthetic technique in eclamptic patients must be made by considering various factors, including the condition of the mother and fetus, the risk of complications, and the experience of the anesthesia team. Regional anesthesia offers several interesting biological benefits in eclamptic patients, such as minimization of respiratory depression and the transfer of drugs to the fetus.18-20

4. Conclusion

The choice of anesthetic technique in eclamptic patients must be done individually by considering various factors. Patients with severe eclampsia who have seizures and are at high risk of aspiration are ideal candidates for general anesthesia. The condition of the fetus also needs to be considered. In patients with a stressed fetus, regional anesthesia may be considered to minimize the use of anesthetic drugs that can cross the placenta. General and regional anesthesia have different risks of complications. The anesthesia team must be experienced in treating eclamptic patients and have the readiness to deal with complications that may occur. In this case, the patient experienced severe seizures and a high risk of aspiration. Therefore, general anesthesia is the right choice. Regional anesthesia was not considered because the patient was unstable and there was a risk of coagulopathy.

5. References

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