



## Bioscientia Medicina: Journal of Biomedicine & Translational Research

Journal Homepage: [www.bioscmed.com](http://www.bioscmed.com)

### Intensive Care Management of Eclampsia Complicated with Acute Kidney Injury: A Case Report

Liliriawati Ananta Kahar<sup>1\*</sup>, Wiwi Monika Sari<sup>2</sup>, Rizky Rivonda Bennovry<sup>3</sup>, Muhammad Ryfki Syahredi Adnani<sup>3</sup>

<sup>1</sup>Department of Anesthesiology and Intensive Care, Faculty of Medicine, Universitas Andalas, Padang, Indonesia

<sup>2</sup>Department of Intensive Care, Dr. M. Djamil General Hospital, Padang, Indonesia

<sup>3</sup>Department of Obstetrics and Gynecology, Faculty of Medicine, Universitas Andalas, Padang, Indonesia

#### ARTICLE INFO

##### Keywords:

Acute kidney injury

Eclampsia

Intensive care

##### \*Corresponding author:

Liliriawati Ananta Kahar

##### E-mail address:

[lili\\_ananta@ymail.com](mailto:lili_ananta@ymail.com)

All authors have reviewed and approved the final version of the manuscript.

<https://doi.org/10.37275/bsm.v7i10.872>

#### ABSTRACT

**Background:** Pre-eclampsia-related AKI occurs in 21% of all pregnancies, with 4% associated with severe eclampsia. The most common cause of AKI is eclampsia, which affects about 3-5% of pregnancies. **Case presentation:** The patient came to the hospital with seizures three times at home, whole body seizures, duration 1-2 minutes, and after a seizure, the patient was unconscious. The patient had a seizure in the hospital twice, 5-10 minutes. She has had a history of hypertension during pregnancy (210/112 mmHg). The patient was diagnosed with decreased consciousness due to eclampsia antepartum on Primipara 31-32 weeks of preterm pregnancy on a maintenance dose of MgSO<sub>4</sub> regimen + oligohydramnion patient was performed emergency section cesarean. After Termination, the patient was admitted to ICU. The patient was assisted by a mechanical ventilator. **Conclusion:** Critical care management is necessary for eclampsia with Acute Kidney Injury, adequate initial management, including hemodynamic stabilization, fluid balance, electrolyte correction, and possibly dialysis, is required to reduce morbidity and mortality.

#### 1. Introduction

Acute kidney injury occurring during pregnancy or postpartum is a dangerous obstetric complication. If accompanied by acute kidney injury, morbidity and mortality in mothers and infants will increase.<sup>1</sup> The most frequent cause of AKI in pregnancy is preeclampsia, exacerbated by eclampsia. However, in most cases that occur in preeclampsia, the decrease in glomerular filtration rate (GFR) is not too significant, only decreasing no more than 30% to 40%, so the increase in serum creatinine levels is also not significant.<sup>2</sup> In a study by Hassan et al., 2022, the incidence of acute kidney injury in women with

eclampsia or severe pre-eclampsia reached 42.86%.<sup>2</sup>

In developed countries, pre-eclampsia-related AKI occurs in 21% of all pregnancies, with 4% associated with severe eclampsia. The most common causes of AKI in developed countries are pre-eclampsia and eclampsia, which affect about 3-5% of pregnancies.<sup>3</sup> In developed countries, pre-eclampsia-related AKI occurs in 21% of all pregnancies, with 4% associated with severe eclampsia. The most common causes of AKI in developed countries are pre-eclampsia and eclampsia, which affect about 3-5% of pregnancies.<sup>2</sup> The most common cause of pregnancy-related acute Kidney injury is hypertension in pregnancy. The peak

incidence of hypertension-related AKI in pregnancy can occur in two most frequent times, with the first occurring at gestational ages between 8 and 16 weeks and the second peak occurring at gestational ages above 16 weeks, which will be with more severe obstetric complications such as preeclampsia and eclampsia, uterine bleeding, and placental abruption.<sup>3,4</sup>

## 2. Case Presentation

The patient came to the hospital with seizures three times at home, whole body seizures, duration 1-2 minutes, and after a seizure, the patient was unconscious. The patient had a seizure in the hospital twice, 5-10 minutes. She has had a history of hypertension during pregnancy (210/112 mmHg) and did not take any regular medication. The history of hypertension from a previous pregnancy, diabetes mellitus type 2, heart problems, and asthmatic episodes was denied. From the physical examination, consciousness was somnolent, Blood Pressure 210/112 mmHg, HR 114 x/min, RR 24x/min, SpO<sub>2</sub> 95% with NRM 15 lpm. Body mass index 30,3 (obesity). The patient was intubated in the emergency department.

From abdominal ultrasound Gravid 29-30 weeks according to biometry, fetal alive, gemelli, intrauterine, transverse lie right head dorsoinferior-breech presentation, the laboratory in emergency room result, haemoglobin 13,0, leukocyte 25.790, trombocyte 437.000, hematocrite 37, ureum: 126, creatinin: 1,6, glucose: 72, protein urine: +3.

The patient was diagnosed with decreased consciousness due to eclampsia antepartum primipara 31-32 weeks of preterm pregnancy on a maintenance dose of MgSO<sub>4</sub> regimen from another institution + oligohydramnion; in the emergency department, the patient was intubated and transfer to operation room to performed emergency caesarean section.

After termination, the patient was admitted to ICU. The patient was intubated and controlled by a mechanical ventilator. Blood pressure on admission is

170/106 mmHg, heart rate: 98 x/m, temperature: 36,5, SpO<sub>2</sub>: 99%. From abdominal examination, uterine fundal palpated two fingers below umbilical, contraction (+), genitalia: v/u normal, vaginal bleeding (-). The patient received a nicardipine drip and methyldopa 3x500 mg. The patient received a midazolam drip in the first 24 hours in ICU. After 24 hours seizure-free, titration started to be decreased and stopped slowly.

Laboratory results in ICU: Hemoglobin 12.0, hematocrit 33, leukocytes: 22,470, platelets: 411,000, albumin 2.3, procalcitonin 3.80, from blood gas analysis pH 7.41 pCO<sub>2</sub> 34, pO<sub>2</sub> 131 HCO<sub>3</sub> 23.6, BCC - 0.8, SO<sub>2</sub>% 99.0%. The patient was diagnosed post-section caesarea a.i antepartum eclampsia primipara, preterm gestational age 31-32 weeks. The patient was planned for 20% albumin transfusion, initiation of feeding test. Then the patient was given the following therapy: ceftriaxone iv 2x1 gr, tranexamic acid iv 3x500 mg, vit k iv 3x10 mg, ketorolac iv 3x30 mg, omeprazole iv 2x40 mg, methyldopa 3x 500 mg, phenytoin 3 x 100 mg. The patient was also consulted to the renal hypertension department and given routinely crystalloid hydration fluid 100 - 150 cc every 4 hours.

From the chest X-ray examination results, infiltrates were found in both lung fields. Then the patient was consulted to the pulmonary department. From the pulmonary consultation, the patient has been assessed as community-acquired pneumonia.

While in the ICU, the target blood pressure was still not achieved with the administration of antihypertensive methyldopa 3 x 500 mg, so the patient was then given nicardipine titration according to body weight to achieve the target blood pressure.

On the second day of monitoring in the ICU, the target blood pressure was achieved and stabilized. The patient's consciousness began to be assessed, and ventilator weaning was performed. After 72 hours of being seizure-free, the patient was extubated, and consciousness was compos mentis. Vital signs were normal, blood pressure 132/86 mmHg, heart rate: 72 x/m, temperature: 36.5, SpO<sub>2</sub>: 99%, with

maintenance methyldopa 3 x 500 mg. After observation and stabilization, the patient was transferred to the obstetric HCU.

### 3. Discussion

Eclampsia is one of the main reasons for obstetric patients to be admitted to the ICU in women. Possible complications such as acute kidney injury and lung edema will aggravate and complicate the patient's condition.<sup>5</sup> Acute kidney injury is characterized by an increase in serum creatinine of about 1.5 times the normal value when urine output decreases to less than 400 mL for more than 6 hours or both. In eclampsia, generalized seizures or loss of consciousness occur during pregnancy or postpartum.<sup>3</sup>

The patient's urinalysis results showed a urine protein of +3. Kidney injury in eclampsia can occur due to disturbances in glomerular endothelins, which can reduce the glomerular filtration rate and renal blood flow. Increased vascular resistance due to hypertension, especially in the kidneys, predisposes to acute kidney injury characterized by proteinuria.<sup>2</sup>

In pregnancy, there will be physiological changes in kidney function, namely an increase in the glomerular filtration rate, so that in normal pregnancy, there will be a decrease in serum creatinine. The physiological changes that occur will mask mild changes in kidney function. In addition, during pregnancy, there can be a decrease in GFR of 30-40% without a significant increase in serum creatinine. The American College of Obstetricians and Gynecologists (ACOG) defines renal impairment in pregnancy hypertension as a serum creatinine level of more than 1.1 mg/dL or twice the normal serum creatinine concentration without a history of previous kidney disease. Hypertension in pregnancy, accompanied by other organ disorders, is one of the leading causes of patients being treated in intensive care units.<sup>1,6</sup>

The definitive treatment for eclampsia is termination of pregnancy. Hypertension in pregnancy will cause an increase in the permeability of the blood-brain barrier, which can disrupt cerebral blood flow due to impaired autoregulation. Most women who experience multiple seizures due to eclampsia will

show signs of cerebral infarction and HELLP syndrome. In eclampsia, Complications can occur are acute kidney failure, pulmonary edema, coagulation disorders, and postpartum hemorrhage.<sup>7</sup>

Endothelial dysfunction in preeclampsia will increase peripheral vascular resistance and disrupt vascular permeability, and this pathophysiological occurrence will result in a state of relative intravascular hypovolemia. Fluid therapy in cases of eclampsia with kidney injury may be a considerable challenge in its management. Giving fluids will increase the volume, which can trigger fluid overload and pulmonary edema, but fluid restrictions can also worsen tissue hypoperfusion and increase the risk of acute kidney injury. In addition, cardiac disorders that may occur also play an essential role in the decision to treat preeclampsia or eclampsia with acute kidney injury.<sup>8</sup>

Preeclampsia, eclampsia, and cardiovascular disease are usually associated with several predisposing factors such as obesity, sedentary lifestyle, smoking, diabetes, chronic kidney disease, chronic hypertension, and abnormal serum lipid profile. In preeclampsia and eclampsia, the renin angiotensin-aldosterone system regulation is dysfunctional. In preeclampsia and eclampsia, there is also vascular hypersensitivity to angiotensin II. Due to imbalance thing, so hypertension and increased peripheral arterial resistance will occur.<sup>9,10</sup>

Strict fluid restriction may result in ischemic renal lesions in eclampsia with renal impairment and cardiovascular disorders. At the same time, excess fluid will also increase hydrostatic pressure in pulmonary capillaries, causing pulmonary edema.<sup>8,9</sup> Eclampsia with multisystem organ disorders requires multidisciplinary management. Mechanical ventilation support and close follow-up monitoring are critical interventions in their management. In particular, eclamptic patients with impaired consciousness require interventions to maintain good oxygenation while minimizing metabolic activity in other organs.<sup>5</sup>

Acute renal failure can be reversed by dialysis, and pulmonary edema can be treated. If pulmonary edema

has occurred, morbidity and mortality in preeclampsia and eclampsia are high. Research shows that increasing fluid volume will increase the risk of complications in women in the peripartum period. Total fluid administration of about 80 ml/hour in patients without cardiac problems reduces the risk of further renal impairment and pulmonary edema.<sup>5</sup>

Reduced intravascular volume in preeclampsia and eclampsia can cause acute kidney injury, vasoconstriction, and activation of inflammatory and coagulation cascades due to preeclampsia. Endothelial dysfunction occurs due to angiogenic mechanisms and antiangiogenic factors. Most renal functional damage in preeclampsia is mild and improves after delivery. The clinical picture of patients with eclampsia is usually more severe and can be accompanied by complications such as coma, pulmonary edema, and renal failure.<sup>11,12</sup>

The management of AKI is determined mainly by the cause of AKI and at what stage AKI is found. Suppose it is found in the prerenal and initiation stages. In that case, the efforts that can be made are optimal management of the underlying disease to prevent the patient from falling into the next stage of AKI. These efforts include rehydration if the cause of AKI is prerenal/hypovolemia, sepsis therapy, and discontinuing nephrotoxic substances. Closed monitoring of fluid intake and output should be done. Management of AKI in pregnancy requires a multidisciplinary team, including specialists in fetal-maternal medicine. Three crucial aspects of managing AKI in pregnancy are treating the underlying disease, maintaining renal function, and preparing for early dialysis. Early management of AKI generally involves optimizing the hemodynamic status of fluid deficit correction. This will have a good effect on the kidneys and can prevent further kidney damage. The use of drugs must be following existing kidney function.<sup>11,13</sup>

Signs and symptoms of eclampsia include seizures, extreme restlessness, and impaired consciousness. Most clinical features before seizures are nausea and vomiting, epigastric discomfort, abdominal pain,

headache, swelling of the hands and face, and visual difficulties. Signs of eclampsia include blood pressure over 160/110 mmHg, serum creatinine over 1.1 mg/dl, liver transaminases at least double the average, and lung edema.<sup>7</sup>

In eclampsia with impaired consciousness, the patient's airway and tongue should be protected first. Administration of MgSO<sub>4</sub> intravenously 4 grams and 10 grams IM as a loading dose based on Pritchard's regimen should be given, followed by 5 grams every four to 24 hours after delivery. Blood pressure should be lowered, with a target diastolic blood pressure of 90-100 mmHg. It can be lowered with IV labetalol, hydralazine, or nifedipine if blood pressure  $\geq$ 160/110. Delivery is recommended for eclampsia at any gestational age when both mother and fetus are stable.<sup>7</sup>

Blood pressure should continue to be closely monitored for at least 72 hours after delivery due to the risk of recurrence of severe hypertension. Vigilance against the development of complications such as pulmonary edema and stroke needs to be watched.<sup>14</sup>

An uncontrolled increase in blood pressure will cause disturbances in the cerebrovascular system in the form of hypertensive encephalopathy with a massive increase in intracranial pressure, resulting in brain edema or intracranial hemorrhage. To avoid this, antihypertensive drugs (IV bolus dose of 5-10 mg hydralazine given over 2 minutes or IV bolus dose of 20-80 mg labetalol over 2 minutes or oral dose of 10-20 mg nifedipine) are used to maintain the target blood pressure. Second-line alternatives include labetalol or nifedipine infusion.<sup>15</sup>

In cases of eclampsia with acute kidney injury, adequate initial management, including hemodynamic stabilization, fluid balance, electrolyte correction, and possibly dialysis, is required to reduce morbidity and mortality. Fluid management may also be complicated by vascular permeability and the possibility of impending renal damage.<sup>15,16</sup>

#### 4. Conclusion

Critical care management is necessary for eclampsia with acute kidney injury, adequate initial management, including hemodynamic stabilization, fluid balance, electrolyte correction, and possibly dialysis, is required to reduce morbidity and mortality.

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