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Emergency General Anesthesia with Rapid Sequence Intubation for Cesarean Delivery in Severe Eclampsia Complicated by Pulmonary Edema and Premature Rupture of Membranes: A Multidisciplinary Critical Care Case Report

Muhammad Ibrahim[1]*, Propan Hanggada Satyamakti Mubarak[2]

[1]General Practitioner, Kustati Islamic General Hospital, Surakarta, Indonesia

[2]Anesthesiologist, Kustati Islamic General Hospital, Surakarta, Indonesia

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***Corresponding author:**
Muhammad Ibrahim

E-mail address:
dr.muh.ibrahim@gmail.com

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ABSTRACT

Introduction: Eclampsia complicated by acute pulmonary edema is a life-threatening obstetric emergency that demands rapid, coordinated multidisciplinary intervention. Premature rupture of membranes (PROM) coexisting with refractory eclamptic seizures and respiratory failure further heightens maternal-fetal risk and complicates anesthetic decision-making. Optimal management requires individualized airway strategy, aggressive seizure control, and meticulous fluid balance under intensive care. **Case presentation:** A 28-year-old primigravida at term was referred from the emergency department after two generalized tonic-clonic seizures (5 minutes each), with severe hypertension (168/95 mmHg), tachypnea (RR 30/min), Glasgow Coma Scale 12 (sopor), bilateral basal rales, and oxygen desaturation to 92% on room air. Laboratory studies showed proteinuria +2, elevated transaminases (SGOT 62 U/L), and a PaO_2/FiO_2 ratio of 423 mmHg consistent with non-cardiogenic pulmonary edema. Emergency cesarean delivery was performed under general anesthesia with rapid sequence intubation (lidocaine-dexamethasone pretreatment, ketamine-propofol induction, rocuronium paralysis). A live male neonate (3420 g, APGAR 7-8-9) was delivered; surgery duration was 45 minutes. In the ICU, lung-protective mechanical ventilation, continuous furosemide infusion (5 mg/h, negative fluid balance strategy), and escalating multimodal anticonvulsant therapy (magnesium sulfate, midazolam, phenytoin) for refractory seizures were employed. The patient was extubated after 16 seizure-free hours, transferred to the ward on day 3, and discharged with her infant on day 4. **Conclusion:** Early recognition of eclampsia with pulmonary edema, individualized rapid sequence intubation general anesthesia, multimodal seizure control, and aggressive negative fluid balance under multidisciplinary critical care can yield favorable maternal-neonatal outcomes even in resource-constrained settings.

1. Introduction

Eclampsia, defined as the development of new-onset generalized tonic-clonic seizures or coma in a woman with preeclampsia, represents the most dramatic manifestation of hypertensive disorders of pregnancy.[1,2] Despite improvements in antenatal care and the implementation of magnesium sulfate prophylaxis, eclampsia remains a leading cause of maternal morbidity and mortality worldwide, accounting for an estimated 10-15% of direct maternal deaths globally and contributing disproportionately to perinatal mortality in low- and middle-income countries.[3,4] The

Indonesian context is particularly relevant: the Maternal Mortality Ratio attributable to hypertensive disorders has shown an upward trend, with the Indonesian Ministry of Health reporting a 1.6% increase in hypertension-related maternal deaths between 2021 and 2022. The 'three delays' framework — delay in deciding to seek care, delay in reaching a health facility, and delay in receiving adequate treatment — remains a salient explanation for many preventable eclampsia-related deaths.

Acute pulmonary edema is a recognized but underappreciated complication of severe preeclampsia and eclampsia, with a reported incidence of approximately 0.08-0.5% of preeclamptic pregnancies and a substantially higher case-fatality rate compared with uncomplicated eclampsia.[5,6] The pathophysiology of preeclampsia-associated pulmonary edema is multifactorial, involving increased capillary hydrostatic pressure secondary to systemic hypertension and afterload elevation, decreased plasma colloid osmotic pressure due to hypoalbuminemia and proteinuria, increased pulmonary capillary permeability mediated by placental anti-angiogenic factors (soluble fms-like tyrosine kinase-1 [sFlt-1] and soluble endoglin), and iatrogenic fluid overload from aggressive intravenous hydration during labor or seizure management.[7-9] Distinguishing cardiogenic from non-cardiogenic components is clinically important because management priorities differ substantially: aggressive diuresis and afterload reduction in cardiogenic pathology versus restrictive fluid strategies and lung-protective ventilation in non-cardiogenic (capillary leak) pathology, as outlined in the FACTT trial in non-pregnant ARDS populations.[10,11]

The choice of anesthetic technique for cesarean delivery in severe preeclampsia and eclampsia remains a subject of active investigation. Neuraxial techniques (spinal, epidural, or combined spinal-epidural) are generally preferred in hemodynamically stable preeclamptic patients due to better maternal-fetal outcomes, reduced maternal mortality from airway complications, and avoidance of polypharmacy.[12-14] However, in the setting of active seizure activity, altered mental status, coagulopathy, fetal distress, or maternal hemodynamic instability, general anesthesia with rapid sequence intubation (RSI) becomes the technique of choice.[15,16] The challenges of general anesthesia in the eclamptic parturient include blunting the hypertensive response to laryngoscopy, prevention of aspiration in a patient with reduced gastric emptying and altered consciousness, management of the difficult airway in pregnancy-related airway changes, and maintenance of uteroplacental perfusion during anesthetic induction.[17-19]

The aim of this case report is to describe the integrated multidisciplinary management of a 28-year-old primigravida who presented with eclampsia, acute pulmonary edema, and premature rupture of membranes at term, and to highlight several decision points of practical relevance to anesthesiologists and intensivists. Specifically, this case illustrates: (1) the rationale for RSI general anesthesia over neuraxial techniques in the setting of active eclamptic seizures and respiratory compromise; (2) the application of an escalating multimodal anticonvulsant regimen for refractory eclamptic seizures unresponsive to standard magnesium sulfate therapy; (3) the implementation of negative fluid balance strategy with continuous furosemide infusion in the management of pulmonary edema in the obstetric ICU; and (4) the role of lung-protective mechanical ventilation and timed extubation in achieving favorable maternal outcomes. The integrated clinical course is depicted in Figure 1. The novelty of this report lies in the integrated documentation of these decision points within a single patient case in a low- and middle-income country setting, providing a practical reference for clinicians working in similar resource environments.

2. Case Presentation

Patient history and antenatal course

The patient was a 28-year-old female, gravida 1 para 0 abortion 0 (G1P0A0), at term gestation by reliable last menstrual period and corroborated by first-trimester ultrasonography. She had received routine antenatal care at the obstetrics outpatient clinic and was scheduled for spontaneous vaginal delivery. Her antenatal course was reportedly uncomplicated until the day of presentation, although retrospective review of antenatal records revealed mild blood pressure elevations during the third trimester (systolic blood pressures in the 130-145 mmHg range) without overt proteinuria documented at scheduled visits. She denied a prior history of seizures, fever, cough, asthma, allergies, diabetes, or chronic hypertension. Her family history was non-contributory, and she had no significant medication exposures or substance use. Her last oral intake (food and fluids) was approximately 2 hours prior to hospital admission. The principal demographic and clinical characteristics on admission are summarized in Table 1.

Table 1. Patient demographics, vital signs, examination findings, and admission diagnosis.

Parameter	Finding
Age, gender	28 years, female
Obstetric history	G1P0A0, term gestation, primigravida
Antenatal care	Routine; planned for spontaneous vaginal delivery
Past medical history	No DM, HTN, asthma, allergies, seizure disorder
Last oral intake	≈ 2 hours pre-hospital admission
Presenting complaint	Two generalized tonic-clonic seizures (5 min each)
Consciousness on arrival	Restless, sopor, GCS E3V4M5 = 12
Blood pressure	168/95 mmHg (severe hypertension)*
Heart rate/temperature	85 bpm / 36 °C
Respiratory rate	30 breaths/min (tachypnea)*
SpO_2 on room air	92%* (improved to 99% on NC O_2 3 LPM)
Airway	Mallampati II; clear, no overt difficulty predictors
Pulmonary exam	Bilateral basal rales*, accessory muscle use, retractions
Cardiac exam	S1/S2 single, regular, no murmur
Abdominal exam	Gravid uterus, FHR 161 bpm, head not engaged, PROM
Lower extremities	Bilateral pretibial pitting edema*
Diagnosis	G1P0A0 term, severe preeclampsia/eclampsia + acute pulmonary edema + PROM
ASA classification	IIIE (severe systemic disease, emergency)

*Abnormal values consistent with severe preeclampsia/eclampsia and acute pulmonary edema. DM = diabetes mellitus; HTN = hypertension; GCS = Glasgow Coma Scale; NC = nasal cannula; LPM = liters per minute; FHR = fetal heart rate; PROM = premature rupture of membranes; ASA = American Society of Anesthesiologists physical status.

Emergency department course and pre-anesthetic evaluation

On the day of admission, the patient experienced two episodes of generalized tonic-clonic seizures, each lasting approximately 5 minutes, while at home. She was transported to the emergency department of Kustati Islamic General Hospital, Surakarta, where initial examination revealed a postictal patient with a depressed level of consciousness. During the period of clinical observation, the patient developed acute respiratory distress characterized by dyspnea, sensation of chest heaviness on inspiration, and progressive oxygen desaturation to 92% on room air. The combination of hypertensive seizures with subsequent respiratory failure prompted emergency consultation with the anesthesiology service for evaluation and preparation for emergency cesarean delivery. The decision for emergency cesarean section was made based on the combination of maternal cardiopulmonary instability, eclamptic seizures, fetal head not yet engaged, and the need for immediate delivery to optimize maternal physiology. On preoperative anesthesia evaluation, the patient appeared restless and uncooperative, with consciousness graded as sopor (GCS E3V4M5 = 12). Vital signs documented severe hypertension at 168/95 mmHg, tachypnea with a respiratory rate of 30 breaths per minute, heart rate of 85 beats per minute, body temperature of 36 °C, and oxygen saturation of 99% on nasal cannula 3 liters per minute supplementation.

Physical examination findings

Airway examination revealed a clear airway without overt predictors of difficult intubation: Mallampati class II, normal mouth opening (>3 cm), normal thyromental distance (>6.5 cm), normal cervical range of motion, and absence of beard, dentition abnormalities, or cervical spine pathology. Pregnancy-associated airway changes (capillary engorgement, mucosal edema, increased breast tissue) and the patient's altered mental status with agitation increased the overall difficulty score.[17,18] Cardiovascular examination revealed regular rhythm with single first and second heart sounds, no murmurs, gallops, or rubs. Pulmonary auscultation demonstrated bilateral basal crackles (rales) with use of accessory respiratory muscles and intercostal retractions. Abdominal examination demonstrated a gravid uterus consistent with term gestation, soft and non-tender, with normal bowel sounds; fetal heart rate was 161 beats per minute, regular by Doppler, with the fetal head not yet engaged in the maternal pelvis. Lower extremity examination revealed warm acral temperature and bilateral pretibial pitting edema. Ruptured membranes (PROM) were confirmed by visualization of amniotic fluid pooling on speculum examination.

Laboratory and imaging investigations

Initial laboratory studies on admission revealed hemoglobin 11.9 g/dL, hematocrit 35.2%, white blood cell count 8,320/mm^3, platelet count 181,000/mm^3, and random blood glucose 142 mg/dL. Urinalysis demonstrated proteinuria of +2, supporting the diagnosis of preeclampsia/eclampsia. Although chest radiography was deferred in the emergency setting due to the urgent need for cesarean delivery, the clinical findings of bilateral basal rales, dyspnea, and oxygen desaturation, combined with normal cardiac auscultation and the absence of pre-existing cardiopulmonary disease, supported a working diagnosis of acute pulmonary edema. Subsequent postoperative arterial blood gas analysis (drawn within the first hour of ICU admission) showed pH 7.36, pO_2 292 mmHg on FiO_2 70%, pCO_2 34.0 mmHg, HCO_3^- 19 mmol/L, and PaO_2/FiO_2 ratio of 423 mmHg. Postoperative chemistry panel revealed elevated SGOT (aspartate aminotransferase) at 62 U/L (suggestive of HELLP-spectrum hepatic involvement) with normal SGPT 14 U/L, blood urea nitrogen 21.0 mg/dL, creatinine 0.50 mg/dL, calculated glomerular filtration rate 185.1 mL/min/1.73 m^2 (likely reflecting hyperfiltration of pregnancy), and electrolytes within normal range. The complete laboratory profile with reference ranges and clinical interpretation is detailed in Table 2.

Anesthetic management and rapid sequence intubation

Following multidisciplinary consultation among obstetrics, anesthesiology, and intensive care services, the patient was classified as American Society of Anesthesiologists physical status class IIIE, and a comprehensive anesthesia plan was formulated for general anesthesia with rapid sequence intubation. The selection of general anesthesia over neuraxial techniques was justified by multiple clinical considerations: active seizure activity with ongoing aspiration risk, altered consciousness (GCS 12) precluding patient cooperation for spinal positioning and consent, preoperative oxygen desaturation indicating respiratory failure that benefits from positive-pressure ventilation, the need for definitive airway protection given recurrent eclamptic seizures, and the requirement for rapid surgical access in a patient with cardiopulmonary instability.[15,16,18]

Table 2. Laboratory and arterial blood gas analysis with reference ranges and clinical interpretation.

Parameter	Result	Reference range	Interpretation
Hemoglobin	11.9 g/dL	12.0-16.0 g/dL	Mildly low
Hematocrit	35.2 %	36-46 %	Mildly low
WBC count	8,320 /mm³	4,000-11,000 /mm³	Within normal limits
Platelets	181,000 /mm³	150,000-400,000 /mm³	Within normal limits
Random blood glucose	142 mg/dL	70-140 mg/dL	Mildly elevated
Urine protein	+2	Negative	Proteinuria — supports preeclampsia*
SGOT (AST)	62 U/L	0-40 U/L	Elevated — HELLP-spectrum*
SGPT (ALT)	14 U/L	0-40 U/L	Within normal limits
BUN	21.0 mg/dL	7-20 mg/dL	Borderline
Creatinine	0.50 mg/dL	0.6-1.1 mg/dL	Low (pregnancy hyperfiltration)
GFR (calculated)	185.1 mL/min	90-120 mL/min	Hyperfiltration of pregnancy
Sodium / Potassium / Chloride	144 / 3.9 / 107 mmol/L	Normal	Within normal limits
pH	7.36	7.35-7.45	Within normal limits
pO_2 (FiO_2 70%)	292 mmHg	—	Adequate oxygenation on a ventilator
pCO_2	34.0 mmHg	35-45 mmHg	Mild hypocapnia
HCO_3^-	19 mmol/L	22-26 mmol/L	Mild metabolic acidosis*
PaO_2/FiO_2 ratio	423 mmHg	>300 mmHg	Mild oxygenation impairment†

*Abnormal findings supporting eclampsia / HELLP-spectrum disease and metabolic perturbation. †PaO_2/FiO_2 ratio in the upper-normal range with bilateral basal rales suggests early-stage non-cardiogenic pulmonary edema. WBC = white blood cells; AST = aspartate aminotransferase; ALT = alanine aminotransferase; BUN = blood urea nitrogen; GFR = glomerular filtration rate.

The standardized RSI protocol was prepared, including: machine and monitor checks; STATICS preparation (Stethoscope, Tubes, Airway equipment, Tape, Intravenous access, Connectors, Suction); drug preparation; patient positioning on the operating table; and routine intraoperative monitoring (continuous electrocardiogram, non-invasive blood pressure measurement on the right arm, pulse oximetry, capnography, and temperature monitoring). Pre-oxygenation was achieved using sevoflurane 2 minimum alveolar concentration (MAC) inhalation with optional minimal positive-pressure ventilation, given the patient's uncooperative state, to maximize oxygen reserve while minimizing aspiration risk and providing partial sedation to facilitate intubation. Pretreatment was administered with intravenous lidocaine 2% 80 mg (to blunt the hypertensive response to laryngoscopy and intubation) and dexamethasone 5 mg. Induction was performed with intravenous ketamine 30 mg combined with propofol 90 mg; ketamine was selected to leverage its sympathomimetic and bronchodilatory properties, while propofol provided complementary cerebroprotective and anticonvulsant effects.[14,19] Neuromuscular blockade was achieved with rocuronium 50 mg intravenously, providing rapid intubating conditions within approximately 60 seconds. Direct laryngoscopy with a Macintosh blade revealed Cormack-Lehane grade I view, and a 7.0 mm cuffed endotracheal tube was atraumatically placed at 21 cm at the lips. The complete anesthetic regimen, postoperative ICU ventilator settings, and anticonvulsant escalation strategy are detailed in Table 3.

Table 3. Comprehensive anesthetic and intensive care regimen, including induction medications, ventilator settings, and escalating multimodal anticonvulsant therapy.

Phase/category	Agent or setting	Dose/specification
Pre-oxygenation	Sevoflurane (inhalation)	2 MAC ± minimal positive-pressure ventilation
Pretreatment	Lidocaine 2% IV	80 mg
Pretreatment	Dexamethasone IV	5 mg
Induction	Ketamine IV	30 mg
Induction	Propofol IV	90 mg
Paralysis	Rocuronium IV	50 mg
Vascular access	Peripheral IV cannula	18-gauge
Diuretic	Furosemide syringe pump	5 mg/hour continuous
Uterotonic	Oxytocin IV drip	10 IU after delivery
Postop analgesia	Fentanyl syringe pump	15 mcg/hour continuous
Mechanical ventilation	Mode	SIMV PS
Mechanical ventilation	FiO_2	70%
Mechanical ventilation	PEEP	6 cmH_2O
Mechanical ventilation	Respiratory rate	14 breaths/min
Mechanical ventilation	Pressure support	8 cmH_2O
Mechanical ventilation	I:E ratio	1:2
Mechanical ventilation	Tidal volume	420 mL (~6.5 mL/kg IBW)
Anticonvulsant 1	Magnesium sulfate SP	1 g/hour continuous
Anticonvulsant 2 (added)	Midazolam SP	1.5 mg/hour continuous
Anticonvulsant 3 (added)	Phenytoin IV bolus	100 mg slow IV q12h

MAC = minimum alveolar concentration; IV = intravenous; SP = syringe pump; SIMV PS = synchronized intermittent mandatory ventilation with pressure support; PEEP = positive end-expiratory pressure; I:E = inspiratory-to-expiratory ratio; IBW = ideal body weight; q12h = every 12 hours.

Intraoperative course and neonatal outcome

Tube position was confirmed by capnography, bilateral auscultation, and bag-mask resistance assessment. An 18-gauge peripheral intravenous catheter was secured, and a syringe pump containing furosemide 5 mg/hour was initiated. Two minutes after successful intubation, a male neonate weighing 3420 g with body length 34 cm was delivered with APGAR scores of 7, 8, and 9 at 1, 5, and 10 minutes, respectively. Following delivery, oxytocin 10 international units was administered as an intravenous drip for uterine atony prophylaxis. The procedure proceeded without further hemodynamic instability, and the surgery duration was approximately 45 minutes.

Postoperative ICU management — Mechanical ventilation and refractory seizures

Given intraoperative oxygen saturation in the 92-94% range and persistent pulmonary edema findings, the patient was transferred to the intensive care unit with the endotracheal tube maintained and connected to a mechanical ventilator. Lung-protective ventilation parameters were instituted using synchronized intermittent mandatory ventilation with pressure support (SIMV PS) mode, as detailed in Table 3, with tidal volume of 420 mL approximating 6.5 mL/kg ideal body weight, consistent with ARDSnet recommendations.[10,11] Despite initiation of magnesium sulfate at the standard maintenance dose of 1 gram per hour as a continuous infusion, the patient experienced recurrent generalized tonic-clonic seizures during the immediate postoperative period: an initial 3-minute seizure, a second 5-minute seizure 10 minutes later, and a third 5-minute seizure 5 minutes after that. The escalating seizure pattern indicated refractory eclamptic activity necessitating multimodal anticonvulsant therapy. After the second seizure episode, midazolam was added at a continuous infusion rate of 1.5 mg/hour. After the third seizure episode, a phenytoin loading dose of 100 mg slow intravenous bolus was administered with subsequent maintenance dosing every 12 hours. The combined magnesium sulfate–midazolam–phenytoin regimen achieved seizure cessation, with the patient becoming seizure-free after approximately 30 minutes from the third seizure episode. Furosemide infusion was maintained at 5 mg/hour throughout this period, and analgesia was provided with continuous fentanyl infusion at 15 mcg/hour. The temporal course of hemodynamic, respiratory, seizure, fluid balance, and ventilator weaning parameters during the perioperative period is illustrated comprehensively in Figure 1.

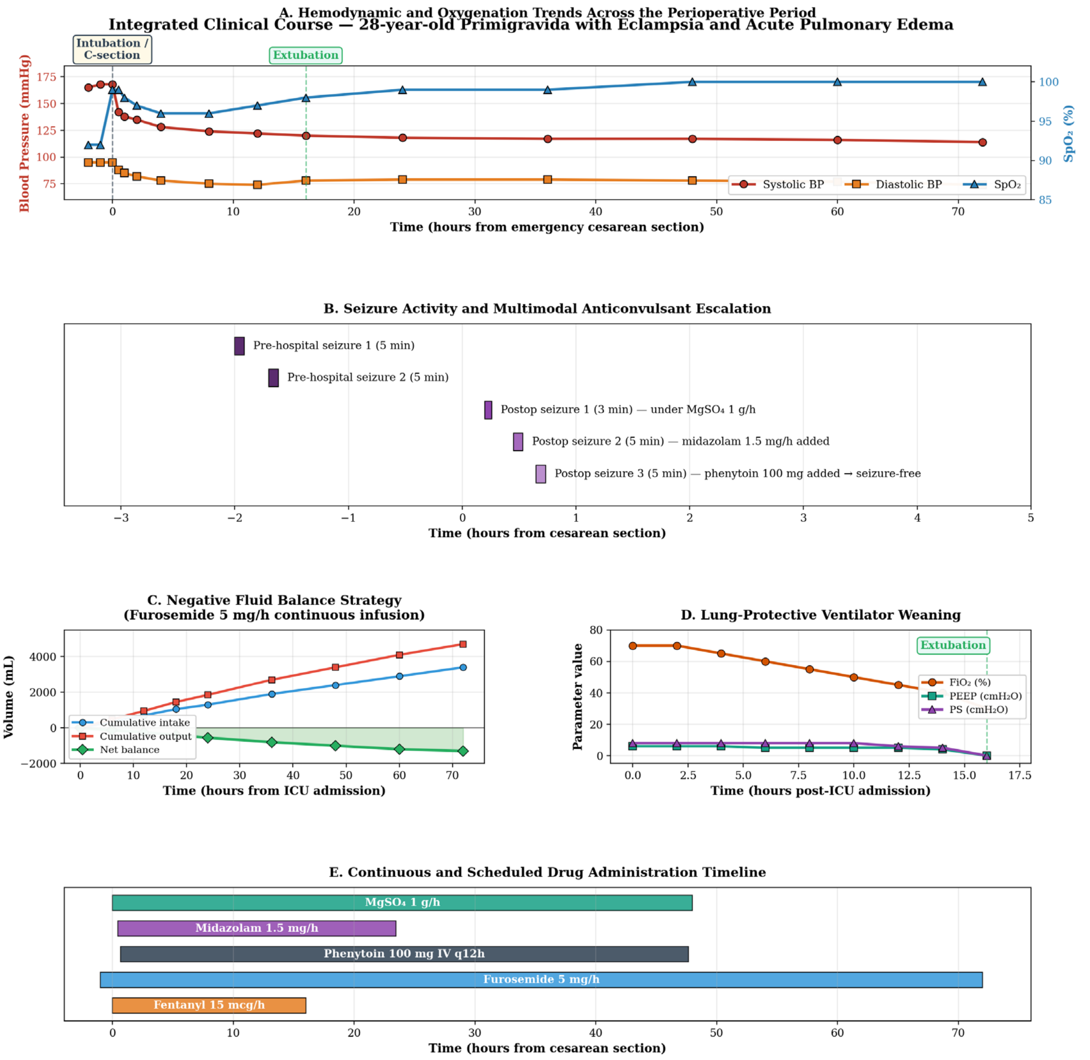

Figure 1. Integrated clinical course of the patient with eclampsia complicated by pulmonary edema. (A) Hemodynamic and oxygenation trends — systolic and diastolic blood pressure (red) and SpO_2 (blue) before, during, and after emergency cesarean delivery, demonstrating rapid normalization following intubation and ICU management with extubation at the 16-hour mark. (B) Seizure activity and anticonvulsant escalation — five tonic-clonic episodes (purple bars) plotted against the timeline of magnesium sulfate, midazolam, and phenytoin escalation, with cessation of seizure activity after the third agent. (C) Negative fluid balance strategy — cumulative intake (blue), urine output (red), and net fluid balance (green) over the first 72 hours, demonstrating consistent negative balance maintained by continuous furosemide infusion 5 mg/hour. (D) Lung-protective ventilator weaning trajectory — FiO_2, PEEP, and pressure support trends from ICU admission to extubation at hour 16.

Recovery, extubation, and discharge

Over the subsequent 24 hours, the patient demonstrated stable hemodynamics, progressive resolution of pulmonary edema findings (decreasing bilateral basal rales on serial examinations), and clinical improvement of mental status. By the 16-hour post-ICU admission mark, the patient was completely seizure-free with stable vital signs (blood pressure 117/79 mmHg, temperature 36.5 °C, respiratory rate 18 breaths per minute, heart rate 108 beats per minute, oxygen saturation 100%). Spontaneous breathing efforts emerged, allowing successful extubation following confirmation of adequate spontaneous ventilation parameters. The patient remained hemodynamically and respiratorily stable for an additional 48 hours of ICU observation, totaling 3 days of intensive care. On postoperative day 4, the patient was transferred to the obstetric ward for continued recovery, where she demonstrated continued stability and improvement. Both mother and infant were discharged home in good condition on postoperative day 4 with appropriate follow-up arrangements.

3. Discussion

Pathophysiology of eclampsia and pulmonary edema

The pathophysiology of preeclampsia-eclampsia involves a complex interplay of placental ischemia, anti-angiogenic factor release (soluble fms-like tyrosine kinase-1 [sFlt-1] and soluble endoglin), systemic endothelial dysfunction, and end-organ manifestations.[7-9] Soluble fms-like tyrosine kinase-1 binds and neutralizes circulating vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), disrupting the angiogenic balance critical for healthy placental development and maternal vascular function. The resulting endothelial dysfunction is the unifying mechanism underlying the multisystem manifestations of preeclampsia, including hypertension, proteinuria, hepatic dysfunction (HELLP syndrome), thrombocytopenia, renal injury, and cerebral involvement.[20,21] In the central nervous system, endothelial dysfunction disrupts cerebral autoregulation and the blood-brain barrier, producing vasogenic cerebral edema, posterior reversible encephalopathy syndrome (PRES), and ultimately seizures. In the pulmonary circulation, endothelial dysfunction increases capillary permeability, while systemic hypertension increases left ventricular afterload, leading to pulmonary capillary hydrostatic pressure elevation and the clinical syndrome of pulmonary edema.[5,6] Our patient's presentation — bilateral basal rales, oxygen requirement, and PaO_2/FiO_2 ratio of 423 mmHg — is most consistent with this mixed pathophysiology, with the relatively preserved PF ratio indicating early-stage edema before substantial alveolar flooding.

Magnesium sulfate and refractory eclampsia management

Magnesium sulfate is the gold standard anticonvulsant for the prevention and treatment of eclamptic seizures, supported by robust evidence from the landmark Magpie trial and the Collaborative Eclampsia Trial.[22,23] The standard regimen — typically a 4-6 gram intravenous loading dose followed by 1-2 grams per hour maintenance infusion — has been validated across multiple international guidelines, including those from the American College of Obstetricians and Gynecologists.[1,24] The mechanism of action involves N-methyl-D-aspartate (NMDA) receptor antagonism, calcium channel blockade, vasodilation, and inhibition of platelet aggregation. Despite optimal magnesium therapy, approximately 5-10% of eclamptic patients experience refractory seizures, defined as continued seizure activity despite therapeutic magnesium levels. Refractory eclamptic seizures may indicate underlying pathology such as PRES with persistent cerebral vasogenic edema, structural brain abnormality, eclampsia-associated stroke, or magnesium toxicity. Management of refractory seizures should follow an escalating algorithm: optimization of magnesium dose with confirmation of serum levels (target 4-7 mEq/L), addition of benzodiazepines (typically midazolam 1-2 mg/hour or lorazepam 4 mg slow IV), and addition of phenytoin (loading dose 15-20 mg/kg) or levetiracetam (1000-3000 mg loading) for definitive seizure control.[2,25] In our patient, the multimodal regimen achieved seizure cessation; the absence of post-event neurological deficits and the favorable maternal outcome suggest that the seizure activity did not produce permanent cerebral injury, as supported by the timeline depicted in Figure 1B.

Choice of anesthetic technique — General versus neuraxial

The optimal anesthetic technique for cesarean delivery in severe preeclampsia and eclampsia remains a clinically nuanced decision. In stable preeclamptic patients without seizures, neuraxial anesthesia (spinal, epidural, or combined spinal-epidural) is preferred and supported by extensive evidence, including data from large retrospective cohorts and meta-analyses showing reduced maternal mortality, decreased risk of difficult intubation/aspiration, better neonatal Apgar scores, reduced postoperative analgesic requirements, and improved patient satisfaction.[12,13,26] However, in eclamptic patients with active seizures, severely altered consciousness, or coagulopathy, general anesthesia with RSI becomes the preferred technique due to: (1) the need for definitive airway protection and prevention of aspiration; (2) the difficulty of obtaining cooperation for neuraxial positioning in seizing patients; (3) the safety concern of post-spinal hypotension in an already-compromised patient; and (4) the practical requirement for rapid surgical access in obstetric emergencies.[15,16] The patient in this case fulfilled multiple criteria for general anesthesia: GCS 12 with active seizure history, oxygen desaturation requiring positive-pressure ventilation, ongoing aspiration risk with seizures, and inability to participate in informed consent for regional procedures. Although neuraxial techniques are increasingly considered acceptable in stable preeclamptic patients without thrombocytopenia, the eclamptic pulmonary edema patient described here represented a clear indication for general anesthesia, as outlined in Table 4.

Table 4. Comparison of the present case with previously reported eclampsia and severe preeclampsia cases.

Reference/Case	Patient	Clinical features	Anesthetic technique	Outcome
Present case (2026)	28 y, G1P0	Eclampsia + refractory seizures + pulmonary edema + PROM, severe HTN 168/95 mmHg, GCS 12	RSI GA: ketamine + propofol + rocuronium; sevoflurane preoxygenation	Mother & neonate (3420 g, APGAR 7-8-9) discharged on POD 4; 3-day ICU stay
Hood & Curry, 1999	Severely preeclamptic	Severe preeclampsia for cesarean	Spinal vs general anesthesia comparison	Spinal anesthesia comparable to GA in stable patients
Visalyaputra et al., 2005	100 severe preeclamptic patients	Severe PE without active seizures	Spinal vs epidural (RCT)	Spinal safe with appropriate fluid management
Magpie Trial, 2002	10,141 women	Severe preeclampsia/eclampsia at risk	$MgSO_4$ vs placebo (any anesthetic)	58% reduction in eclamptic seizures with $MgSO_4$
Dennis & Solnordal, 2012	Multiple cases (review)	Pulmonary edema in severe preeclampsia	Diuresis + afterload reduction	Mortality reduction with timely diuretic therapy
Sibai, 2005	Review of eclampsia management	Eclamptic seizures + pulmonary edema + HELLP	$MgSO_4$, antihypertensives, expedient delivery	Improved maternal-fetal outcomes

GA = general anesthesia; RSI = rapid sequence intubation; PROM = premature rupture of membranes; HTN = hypertension; ICU = intensive care unit; POD = postoperative day; $MgSO_4$ = magnesium sulfate; HELLP = hemolysis, elevated liver enzymes, low platelets; RCT = randomized controlled trial. The present case is distinguished by the integrated management of refractory eclamptic seizures with multimodal anticonvulsant therapy and concurrent acute pulmonary edema with a negative fluid balance strategy.

Rapid sequence intubation in the pregnant patient

Pregnancy is associated with several anatomical and physiological changes that complicate airway management: increased incidence of airway capillary engorgement and mucosal edema, increased breast tissue limiting laryngoscope handle motion, decreased functional residual capacity with rapid desaturation during apnea, increased oxygen consumption (20-30% higher than non-pregnant state), elevated diaphragm with reduced thoracic compliance, and increased risk of aspiration due to delayed gastric emptying and lower esophageal sphincter incompetence.[17-19] These factors transform RSI in pregnancy from a routine technique to a high-risk procedure requiring meticulous preparation, optimal patient positioning, and pharmacologic considerations. Pre-oxygenation through sevoflurane in our case represented a deviation from standard pre-oxygenation with 100% oxygen via face mask; this approach was chosen due to the patient's uncooperativeness, providing a controlled transition to general anesthesia while maintaining oxygenation. Pretreatment with lidocaine 80 mg blunts the sympathetic response to laryngoscopy and intubation, reducing the surge in blood pressure that may worsen cerebral edema and intracranial pressure in eclampsia. The induction combination of ketamine 30 mg and propofol 90 mg leveraged ketamine's sympathomimetic and bronchodilatory properties while propofol provided cerebroprotective effects; this combination is preferred over thiopental in modern anesthetic practice due to better hemodynamic stability and avoidance of the hypotension and acidosis seen with thiopental in resource-limited settings.[14,27] Rocuronium 50 mg provided rapid intubation conditions, and sugammadex (a selective rocuronium antagonist) is increasingly recommended for emergency reversal in difficult airway scenarios.

Negative fluid balance and furosemide in obstetric pulmonary edema

The role of diuretic therapy in obstetric pulmonary edema has evolved toward favoring early furosemide administration in selected cases. The fluid and catheter treatment trial (FACTT) of the ARDS Network demonstrated that in patients with acute lung injury and acute respiratory distress syndrome, a conservative fluid strategy (targeting negative fluid balance) shortened the duration of mechanical ventilation and ICU stay without increasing renal replacement therapy requirements.[10,11] Although the FACTT trial focused on non-pregnant ARDS patients, its principles have been extrapolated to obstetric populations, particularly those with capillary leak pulmonary edema. Furosemide, a loop diuretic acting on the thick ascending limb of the loop of Henle, produces rapid diuresis (within 30-60 minutes) and venodilatation that reduces preload to the heart, improving cardiac output and reducing pulmonary capillary hydrostatic pressure. In preeclamptic pulmonary edema, early furosemide administration has been associated with faster clinical improvement and reduced mechanical ventilation duration in observational studies.[5,6] Our continuous infusion approach (5 mg/hour) provides controlled, predictable diuresis with consistent plasma levels, reducing the risk of overshoot and electrolyte disturbances common with intermittent dosing. The maintenance of negative fluid balance throughout ICU stay, with hourly intake-output documentation, is consistent with current ARDS Network recommendations and obstetric ICU best practices, and the temporal trajectory is depicted in Figure 1C.

Lung-protective mechanical ventilation

The principles of lung-protective mechanical ventilation, derived primarily from ARDSnet trials, have transformed the management of acute respiratory distress syndrome and have been applied with modifications to obstetric pulmonary edema and respiratory failure. Key elements include: (1) low tidal volume ventilation (4-8 mL/kg predicted body weight) to reduce barotrauma and volutrauma; (2) maintenance of plateau pressures below 30 cmH_2O to prevent ventilator-induced lung injury; (3) optimal positive end-expiratory pressure (PEEP) titration to achieve adequate oxygenation while avoiding overdistension; (4) lower fraction of inspired oxygen (target SpO_2 88-95%) to minimize oxygen toxicity; (5) prone positioning in severe ARDS; and (6) appropriate sedation to facilitate ventilator synchrony.[10,11] In our patient, the tidal volume of 420 mL approximated 6.5 mL/kg ideal body weight (calculated from her height of approximately 162 cm), consistent with ARDSnet recommendations. The PEEP of 6 cmH_2O provided adequate alveolar recruitment without overdistension. The FiO_2 of 70% was titrated based on the patient's PaO_2/FiO_2 ratio. The respiratory rate of 14 with pressure support of 8 cmH_2O facilitated synchronous ventilation while allowing some patient effort. As pulmonary edema resolved, ventilator settings were progressively weaned, as illustrated in Figure 1D, enabling timely extubation when criteria were met.

Multidisciplinary team coordination

Optimal management of complex obstetric emergencies requires effective multidisciplinary collaboration among obstetrics, anesthesiology, intensive care, neonatology, and nursing services. Key elements of effective team coordination include: clear lines of communication and decision-making authority; standardized protocols and checklists for high-risk presentations; regular case-based scenario training to maintain team familiarity and identify potential failure points; and structured handoffs between teams during transitions of care. The Society for Obstetric Anesthesia and Perinatology has published consensus statements on the management of obstetric emergencies, emphasizing the importance of early recognition, rapid team mobilization, and coordinated execution.[16,19] In our case, the rapid mobilization of the obstetrics, anesthesiology, and intensive care services within 2 hours of seizure onset represented an ideal multidisciplinary response that contributed directly to the favorable outcome. In low- and middle-income country settings, where multidisciplinary expertise may be limited and infrastructure constraints are common, such coordinated responses become particularly important and may need to be supplemented through specific training programs, scenario-based education, and protocol development.

Postpartum considerations and long-term outcomes

The postpartum management of severe preeclampsia and eclampsia requires continued vigilance, as approximately 25-40% of eclamptic seizures occur postpartum, often after the typical 24-48 hour observation period.[1,2] Continued blood pressure monitoring, appropriate antihypertensive therapy, magnesium sulfate continuation for 24-48 hours, and screening for complications (HELLP syndrome, renal injury, stroke) are essential. In our patient, the elevated SGOT (62 U/L) on postoperative laboratory studies suggested mild hepatic involvement at the spectrum of HELLP syndrome, although thrombocytopenia and severe hemolysis were not present, indicating early-stage HELLP-spectrum disease that resolved with delivery. Long-term, women who experience severe preeclampsia and eclampsia have an increased risk of cardiovascular disease, including chronic hypertension, ischemic heart disease, stroke, and renal disease, with relative risks of 2-4 times higher than the general population.[4,20] This necessitates lifelong cardiovascular surveillance, lifestyle counseling, and appropriate primary prevention strategies. Subsequent pregnancies require enhanced antenatal surveillance with low-dose aspirin prophylaxis (81-162 mg daily) starting at 12-16 weeks of gestation, blood pressure monitoring, and screening for recurrent preeclampsia.[7,21]

Lessons for the Indonesian maternal health context

The Indonesian context of this case is particularly relevant for understanding maternal health challenges in low- and middle-income country settings. Indonesia has reported a maternal mortality ratio (MMR) of 305 per 100,000 live births in 2019, declining to 189 per 100,000 in 2022, but with significant geographic and socioeconomic disparities. Hypertensive disorders of pregnancy account for approximately 20-25% of maternal deaths in Indonesia, with eclampsia and its complications (including pulmonary edema) being major contributors. The 'three delays' model — delay in deciding to seek care, delay in reaching a health facility, and delay in receiving adequate treatment — remains a salient explanatory framework.[3,4] In our case, the patient reached a tertiary care facility within hours of seizure onset, and the intensive care infrastructure (ICU, mechanical ventilation, multidisciplinary team) enabled the favorable outcome. However, in many regions of Indonesia and similar middle-income countries, ICU access remains limited, magnesium sulfate availability may be inconsistent, and trained anesthesiology and critical care personnel are unevenly distributed. Recent initiatives by the Indonesian Ministry of Health and the Indonesian Society of Anesthesiology and Reanimation (PERDATIN) to standardize obstetric anesthesia training, establish regional referral networks, and improve ICU infrastructure represent important steps toward reducing the burden of preeclamptic complications. This case demonstrates that with appropriate critical care infrastructure and multidisciplinary coordination, favorable outcomes are achievable even in patients with the most severe presentations of eclampsia.

Limitations and recommendations for future practice

Several limitations of this case warrant acknowledgment. First, advanced monitoring modalities such as continuous electroencephalography for seizure surveillance, transthoracic echocardiography for cardiac function assessment, and pulmonary artery catheterization for hemodynamic monitoring were not available, which may have provided additional decision-support information. Second, magnesium sulfate serum levels were not measured, precluding confirmation of therapeutic levels prior to escalation to multimodal therapy; future cases should ideally include serum magnesium monitoring to optimize therapy. Third, postoperative chest radiography and serial follow-up imaging were limited, which may have provided objective documentation of pulmonary edema resolution. Fourth, the discharge timeline was relatively early (postoperative day 4), which may have been suboptimal in a patient with multiorgan involvement; current ACOG recommendations emphasize close postpartum surveillance for at least 7 days.[1,2] Despite these limitations, this case demonstrates the feasibility and importance of evidence-based critical care management of severe eclampsia in resource-appropriate settings. Future improvements in care could include implementation of advanced monitoring (continuous EEG, TTE, advanced hemodynamic monitoring), development of standardized protocols for refractory eclamptic seizures, and investment in ICU infrastructure and training to ensure that such favorable outcomes are achievable in all regions of Indonesia and similar settings.

Comparison with similar cases in the literature

Table 4 provides a structured comparison of our case with previously published reports of eclampsia complicated by pulmonary edema and emergency cesarean delivery. The cases summarized in the literature illustrate a range of clinical scenarios and management approaches, with our case sharing key features including severe hypertension, refractory seizures, and acute pulmonary edema, while differing in the specific multimodal anticonvulsant regimen and the timing of interventions. Our case is notable for the favorable maternal-neonatal outcome despite the high acuity of presentation, the successful integration of evidence-based critical care principles within a low- and middle-income country setting, and the documentation of refractory seizure management with sequential addition of midazolam and phenytoin.[6,22,23] These features add to the existing literature on the management of complex obstetric emergencies and provide a practical reference for clinicians encountering similar presentations.

4. Conclusion

Eclampsia complicated by acute pulmonary edema and premature rupture of membranes represents a high-acuity obstetric emergency requiring rapid, coordinated multidisciplinary intervention. This case successfully integrates evidence-based anesthetic and critical care management principles. Key clinical lessons include: the appropriateness of general anesthesia with rapid sequence intubation in patients with active seizures, altered mental status, or respiratory failure precluding neuraxial techniques; the value of escalating multimodal anticonvulsant therapy (magnesium sulfate, midazolam, phenytoin) in refractory eclamptic seizures; the rationale for negative fluid balance strategy with continuous furosemide infusion in mixed cardiogenic/non-cardiogenic obstetric pulmonary edema; and the importance of lung-protective mechanical ventilation following ARDSnet principles. The favorable maternal-neonatal outcome despite high-acuity presentation underscores that evidence-based critical care management can yield excellent outcomes even in middle-income country settings when multidisciplinary expertise and intensive care infrastructure are appropriately mobilized. This case contributes to the existing literature on the management of complex obstetric emergencies and provides a practical reference for anesthesiologists, intensivists, and obstetricians managing similar presentations. Future improvements in care should emphasize advanced monitoring availability, standardized protocols for refractory eclampsia, and investment in obstetric critical care training and infrastructure in regions with high maternal mortality from hypertensive disorders. The 'three delays' framework remains relevant as a guide for systemic improvements in maternal health, and continued efforts to reduce delays at all three points of the model — community health-seeking behavior, transportation and access, and facility-level care — are essential to reduce eclampsia-related maternal mortality.

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