

CASE REPORT

Rapid Sequence Intubation, Tiered Anticonvulsant Escalation and Continuous Furosemide Infusion in Severe Eclampsia with Acute Pulmonary Edema: A Case Report

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ARTICLE INFO

Article history

Received: August 25, 2026
Revised: September 10, 2026
Accepted: September 29, 2026
Available online: October 2, 2026
Published: October 5, 2026

Keywords

Airway management
Eclampsia
Furosemide
Pulmonary edema
Rapid sequence intubation

DOI

<https://doi.org/10.37275/jacr.v7i2.968>

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ABSTRACT

Background. Eclampsia with acute pulmonary edema is a life-threatening obstetric emergency in which seizure control, airway management and fluid strategy compete.

Objective. To describe the airway, anticonvulsant, ventilatory and fluid management of one parturient with eclampsia, acute pulmonary edema and premature rupture of membranes, and her outcome.

Methods. A 28-year-old nulliparous woman at 38 weeks and 4 days presented after two generalized tonic-clonic seizures, with blood pressure 168/95 mmHg, Glasgow Coma Scale (GCS) 12, bilateral basal crackles and SpO₂ 92% on room air. Emergency cesarean delivery was done under rapid sequence general anesthesia after sevoflurane pre-oxygenation. In the intensive care unit (ICU) she received magnesium sulfate, a continuous furosemide infusion (5.0 mg/h) and low tidal volume ventilation.

Results. Intubation was uneventful, and a male neonate was delivered (3,420 g; Apgar 7, 8 and 9). Within 45 minutes of ICU arrival, three seizures occurred despite magnesium sulfate 1.0 g/h. A midazolam infusion (1.5 mg/h) and phenytoin (100 mg) were added, and no further seizures followed. The cumulative net fluid balance reached -2,800 mL over 72 hours with stable creatinine. She was extubated at hour 16 with GCS 15. Mother and neonate went home on postoperative day 4; her blood pressure was normal at six weeks.

Conclusion. In this patient, rapid sequence induction, escalation of anticonvulsants, low tidal volume ventilation and continuous loop diuresis were followed by survival of mother and neonate. A single case cannot show that any one component caused the recovery.

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1. Introduction

Hypertensive disorders of pregnancy are a leading cause of maternal and perinatal illness and death, and their burden has grown over the past three decades.¹ Eclampsia is far more frequent in low-resource settings (50–151 versus 1.6–10 per 10,000 deliveries).² Reports from Oman, Ghana and Nigeria document this burden.^{3–5} In Indonesia they account for 20–25% of maternal deaths, with a rising trend.⁶ Eclampsia is the occurrence of generalized tonic-clonic seizures in a woman with preeclampsia that cannot be attributed to another cause, so other causes of seizures in pregnancy, such as intracranial infection or local anesthetic toxicity, must be excluded.^{7,8} Preeclampsia is now understood mainly as a vascular disorder of placental origin.⁹ Poor remodeling of the uteroplacental spiral arteries leads to placental ischemia¹⁰ and to the release of anti-angiogenic factors, notably soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin, which bind vascular endothelial growth factor and placental growth factor and cause widespread endothelial dysfunction,^{11–13} including damage to the endothelial glycocalyx.¹⁴ In the brain this produces hyperperfusion and vasogenic edema of the kind seen in posterior reversible encephalopathy syndrome.^{15,16}

Acute pulmonary edema complicates roughly 0.08–1.5% of pregnancies and is hypertensive in origin in most cases.¹⁷ Pregnancy itself predisposes to it, and preeclampsia adds further risk.^{18,19} In preeclampsia it is a mixed hydrostatic and permeability edema: raised afterload, low colloid osmotic pressure after albuminuria from endothelial injury of the kidney²⁰ and a leaky alveolar-capillary membrane combine, and intravenous fluid can precipitate it.²¹ Sepsis and uterotonic drugs are other triggers.²²

Delivery is the definitive treatment of eclampsia.²³ Neuraxial anesthesia is preferred for cesarean delivery in stable severe preeclampsia,²⁴ but it is unsuitable with active seizures, depressed consciousness or respiratory distress, and general anesthesia with rapid sequence intubation is then required.^{25–27} Intubation is hazardous in pregnancy because of mucosal edema,^{28,29} a lower functional residual capacity³⁰ and delayed gastric emptying with a risk of aspiration.³¹ General anesthesia carries higher maternal morbidity than regional anesthesia, mainly through failed intubation, hypoxia and aspiration; difficult intubation occurred in 5.0% of 660 cesarean general anesthetics in one hospital.^{32,33} The pressor response to laryngoscopy can cause dangerous surges in blood pressure.³⁴

Magnesium sulfate is the standard anticonvulsant and has a large trial base,^{35,36} and its anticonvulsant and neuroprotective actions are multiple.³⁷ Breakthrough seizures despite magnesium are uncommon, and the next steps are not standardized; case reports describe thiopental, benzodiazepines and other agents.^{38–41} After delivery, critical care must address the pulmonary edema as well as the seizures. In acute lung injury, a conservative fluid strategy shortened ventilation without harming the kidneys,⁴² continuous furosemide infusion has produced greater urine output than bolus dosing in critically ill patients without differences in mortality or renal function tests,⁴³ and low tidal volume ventilation reduced mortality in the acute respiratory distress syndrome.⁴⁴ Whether these principles apply in eclampsia has not been established.

This report describes the airway, anticonvulsant, ventilatory and fluid management of one parturient with eclampsia, acute pulmonary edema and premature rupture of membranes, from emergency delivery to discharge.

2. Methods

2.1. Report design and reporting guideline

This is a single case report prepared according to the CARE guideline.⁴⁵

2.2. Setting

The patient was managed at Kustati Islamic General Hospital, Surakarta, Central Java, Indonesia, a secondary and tertiary referral hospital, in the emergency department, the operating room and the intensive care unit (ICU). Care extended from presentation to hospital discharge on postoperative day 4.

2.3. Patient information and case selection

The patient was a 28-year-old nulliparous woman (gravida 1, para 0, abortion 0), 65 kg and 162 cm, at 38 weeks and 4 days of gestation by first-trimester ultrasonography. Antenatal care at a primary clinic had been uncomplicated until the third trimester, when systolic pressures of 130–145 mmHg were noted without proteinuria or treatment. She denied epilepsy, asthma, diabetes and renal disease. Her last oral intake was about two hours before admission.

The case is reported because eclampsia, pulmonary edema, premature rupture of membranes and breakthrough seizures occurred together and required airway, anticonvulsant, ventilatory and fluid decisions in one patient.

2.4. Case timeline

The course is summarized in Table 1. Time is given in hours after ICU arrival, or relative to induction (0 h) for the perioperative period.

Table 1. Clinical timeline of the case from presentation to follow-up.

Time point	Clinical event	Investigation and result	Intervention
About 2 h before arrival	Two generalized tonic-clonic seizures at home (about 5 min each, 8 min apart)	—	Ambulance transfer
Emergency department	GCS 12; BP 168/95 mmHg; respiratory rate 30/min; SpO ₂ 92% on room air; basal crackles; ruptured membranes; fetal heart rate 161/min	Dipstick protein +2; AST 62 U/L; hemoglobin 11.9 g/dL; platelets 181 × 10 ³ /μL	Oxygen 3 L/min (SpO ₂ 99%); ASA IIIE; emergency cesarean under general anesthesia
Induction (0 h)	Agitated; BP 168/95 mmHg	Airway: Mallampati II; Cormack-Lehane I	Sevoflurane pre-oxygenation; lidocaine, dexamethasone, ketamine, propofol, rocuronium
Delivery (2 min after intubation)	Male neonate, 3,420 g, Apgar 7/8/9; surgery 45 min; blood loss 450 mL	Cord pH 7.31; base excess −2.4 mmol/L	Oxytocin 10 IU in 500 mL
ICU arrival	Intubated; PaO ₂ 292 mmHg on FiO ₂ 0.70	pH 7.36; PaCO ₂ 34.0 mmHg; HCO ₃ [−] 19.0 mmol/L	SIMV-PS; magnesium sulfate 1.0 g/h; fentanyl 15 mcg/h
0.20–0.65 h after ICU arrival	Three generalized seizures (3, 5 and 5 min)	—	Midazolam 1.5 mg/h after the second; phenytoin 100 mg after the third; no recurrence
Hours 1–16	GCS 10 at hour 1, 15 at hour 16; FiO ₂ 0.70 to 0.35	Breathing trial: RSBI 36.4; pH 7.42, PaO ₂ 98 mmHg, PaCO ₂ 37.5 mmHg on FiO ₂ 0.35	Extubation at hour 16 to nasal cannula 2 L/min
Hours 24–72	MAP 92.0 mmHg (24 h), 88.7 mmHg (72 h); crackles cleared by 36 h; net balance −1,750 mL (24 h), −2,800 mL (72 h)	Creatinine 0.44 mg/dL; AST 22 U/L; platelets 224 × 10 ³ /μL (day 4)	Magnesium stopped 24 h after delivery; furosemide 2.5 mg/h at 24 h, stopped at 48 h
Day 3–6 weeks	Ward on day 3; discharge on day 4; BP 118/76 mmHg at follow-up	Proteinuria negative; renal and liver function normal	No antihypertensive drugs

Notes: AST: aspartate aminotransferase; ASA: American Society of Anesthesiologists physical status; BP: blood pressure; FiO₂: fraction of inspired oxygen; GCS: Glasgow Coma Scale; ICU: intensive care unit; MAP: mean arterial pressure; RSBI: rapid shallow breathing index; SIMV-PS: synchronized intermittent mandatory ventilation with pressure support.

2.5. Clinical findings and diagnostic assessment

Eclampsia was diagnosed from two witnessed generalized tonic-clonic seizures in a previously normotensive nulliparous woman at term with severe-range systolic pressure (≥160 mmHg) and dipstick proteinuria of +2 (about 100 mg/dL), following the American College of Obstetricians and Gynecologists and International Society for the Study of Hypertension in Pregnancy criteria.^{46,47} Acute pulmonary edema was diagnosed from acute dyspnea, a respiratory rate of at least 30 breaths/min, desaturation on room air, bilateral basal crackles and intercostal retractions, with normal cardiac auscultation and no known cardiopulmonary disease. Premature

rupture of membranes was confirmed by sterile speculum examination.

Blood pressure was measured with an oscillometric cuff on the right arm (every minute during induction and delivery, every 15 minutes for the first 6 ICU hours, then hourly), and mean arterial pressure (MAP) was calculated as (SBP + 2 × DBP) / 3. Electrocardiography, pulse oximetry (SpO₂), end-tidal capnography and axillary temperature were monitored continuously. Airway assessment used the Modified Mallampati class, thyromental and inter-incisor distances, sternomental distance and neck movement. Consciousness was scored with the Glasgow Coma Scale (GCS). Arterial blood gases were analyzed immediately (Instrumentation Laboratory GEM Premier 3500).

Hematology, biochemistry and electrolytes were measured at admission, immediately after surgery, at hour 12 and hour 24, and before discharge on day 4. Urine output was measured through an indwelling Foley catheter, and intake and output were tabulated to give hourly and cumulative net balances. Changes were expressed as $\Delta\% = [(value\ at\ day\ 4 - value\ at\ admission) / value\ at\ admission] \times 100$. No inferential statistics were calculated, because $n = 1$.

2.6. Therapeutic intervention

In the emergency department the patient received oxygen by nasal cannula (3 L/min). In the operating room the airway equipment was checked with the STATICS checklist (scope, tubes, airway, tape, introducer, connector, suction). Because she was too agitated to tolerate a tight mask, pre-oxygenation was done with inhaled sevoflurane at 2.0 minimum alveolar concentration (MAC) and gentle positive-pressure mask ventilation. Ninety seconds before laryngoscopy she received lidocaine 2% 80 mg and dexamethasone 5 mg intravenously.^{29,34} Induction used ketamine 30 mg, propofol 90 mg and rocuronium 50 mg; intubation was by direct laryngoscopy (Macintosh 3 blade) with a 7.0-mm cuffed tube secured at 21 cm.³² Anesthesia was maintained with sevoflurane 1.0–1.5 MAC in oxygen/air. Furosemide was started intraoperatively at 5.0 mg/h by syringe pump. A Pfannenstiel incision and low transverse hysterotomy were used. After delivery, oxytocin 10 IU in 500 mL of crystalloid was infused, and methylergonovine was avoided because of its vasoconstrictive effect.

In the ICU the patient was ventilated in synchronized intermittent mandatory ventilation with pressure support (SIMV-PS): tidal volume 420 mL, positive end-expiratory pressure (PEEP) 6 cmH₂O, pressure support 8 cmH₂O, backup rate 14 breaths/min, inspired oxygen fraction (FiO₂) 0.70 and an inspiratory-to-expiratory ratio of 1:2. Magnesium sulfate was infused at 1.0 g/h, fentanyl at 15 mcg/h, and crystalloid was limited to 50–70 mL/h. Midazolam (1.5 mg/h by syringe pump) was added after the second breakthrough seizure and phenytoin (100 mg by slow intravenous bolus over 20 minutes, then 100 mg

every 12 hours) after the third. Furosemide was continued at 5.0 mg/h, reduced to 2.5 mg/h at hour 24 and stopped at hour 48. Extubation followed a 30-minute spontaneous breathing trial on continuous positive airway pressure with pressure support (PEEP 5, pressure support 5 cmH₂O, FiO₂ 0.35).

2.7. Outcome measures and follow-up

Outcomes were seizure recurrence, GCS, blood pressure and SpO₂, the arterial gas and PaO₂/FiO₂ ratio, laboratory values, cumulative fluid balance, time to extubation, neonatal Apgar scores and cord blood gas, and survival at discharge. Follow-up visits took place two and six weeks after discharge.

2.8. Ethics and consent

CMHC Research Center Ethics Committee, Indonesia, approved the protocol (reference 2026/0518). Written informed consent to participation and to publication of anonymized clinical data was obtained from the patient after she regained full capacity. The report follows the Declaration of Helsinki.

3. Results

3.1. Presentation and baseline findings

At triage the patient was restless and uncooperative, with a GCS of 12 (E3 V4 M5), blood pressure 168/95 mmHg (MAP 119.3 mmHg), heart rate 85 beats/min, respiratory rate 30 breaths/min with intercostal retractions, temperature 36.0 °C and SpO₂ 92% on room air, rising to 99% on oxygen 3 L/min. Both lung bases had inspiratory crackles, and both legs showed grade 2+ pitting edema. Heart sounds were normal. The airway examination was reassuring (Table 2). The uterus was term and non-tender, the fetal heart rate was 161 beats/min, the vertex was not engaged, and clear amniotic fluid was seen pooling in the posterior fornix. She was classified as ASA physical status IIIE. The two seizures at home had lasted about 5 minutes each, with 8 minutes of postictal stupor between them.

Table 2. Baseline findings at presentation to the emergency department.

Parameter	Finding	Reference value
Age; gravity and parity	28 years; G1P0A0	—
Gestational age	38 weeks + 4 days	37–41 weeks
Weight; height	65 kg; 162 cm	—
Blood pressure; MAP	168/95 mmHg; 119.3 mmHg	<120/80 mmHg; 70–100 mmHg
Heart rate; temperature	85 beats/min; 36.0 °C	60–100 beats/min; 36.5–37.5 °C
Respiratory rate; SpO ₂ (room air)	30 breaths/min; 92%	12–20 breaths/min; 95–100%
Consciousness	Restless, stuporous; GCS 12 (E3 V4 M5)	GCS 15
Airway	Mallampati II; thyromental distance > 6.5 cm; inter-incisor distance > 3.5 cm; normal neck movement	Class I–II
Lung auscultation	Bilateral basal inspiratory crackles	Clear
Obstetric findings	Term uterus; fetal heart rate 161 beats/min; ruptured membranes; vertex not engaged	110–160 beats/min; intact membranes
Peripheral edema	Grade 2+ pretibial pitting	Trace or none
ASA physical status	IIIE	—

Notes: ASA: American Society of Anesthesiologists; GCS: Glasgow Coma Scale (E: eye, V: verbal, M: motor); MAP: mean arterial pressure; SpO₂: oxygen saturation.

Dipstick proteinuria was +2, aspartate aminotransferase (AST) 62 U/L with alanine aminotransferase 14 U/L, hemoglobin 11.9 g/dL, platelets $181 \times 10^3/\mu\text{L}$ and creatinine 0.50 mg/dL (Table 3). The timeline is in Table 1.

3.2. Diagnostic findings

The first arterial blood gas, drawn after induction on FiO₂ 0.70, showed pH 7.36, PaO₂ 292 mmHg, PaCO₂ 34.0 mmHg and bicarbonate 19.0 mmol/L (Table 3). The authors report a PaO₂/FiO₂ ratio of 423 mmHg (292 / 0.70 = 417 mmHg). Because the ratio exceeds 300 mmHg, the

diagnosis of pulmonary edema rests on the clinical findings, not on impaired gas exchange.

Over the next four days, proteinuria fell from +2 to +1 at hour 12, trace at hour 24 and negative at discharge. AST fell from 62 to 22 U/L ($\Delta\% = -64.52\%$), platelets rose from 181

to $224 \times 10^3/\mu\text{L}$ ($\Delta\% = +23.76\%$), creatinine moved from 0.50 to 0.44 mg/dL ($\Delta\% = -12.00\%$), blood urea nitrogen from 21.0 to 12.8 mg/dL ($\Delta\% = -39.05\%$) and hemoglobin from 11.9 to 11.8 g/dL ($\Delta\% = -0.84\%$). Sodium, potassium and chloride stayed between 139 and 144, 3.8 and 4.2, and 102 and 107 mmol/L (Table 3).

Table 3. Laboratory and arterial blood gas results from admission to day 4.

Analyte	Admission	After surgery	Hour 12	Hour 24	Day 4	Reference	$\Delta\%$
Hemoglobin (g/dL)	11.9	11.2	11.0	11.3	11.8	12.0–16.0	-0.84
Hematocrit (%)	35.2	33.4	33.0	33.8	35.0	36.0–46.0	-0.57
White blood cells ($\times 10^3/\mu\text{L}$)	8.32	11.40	10.20	8.90	7.60	4.00–11.00	-8.65
Platelets ($\times 10^3/\mu\text{L}$)	181	172	178	195	224	150–400	+23.76
Random glucose (mg/dL)	142	136	118	104	96	70–140	-32.39
Urine protein (dipstick)	+2	+2	+1	Trace	Negative	Negative	—
AST (U/L)	62	58	46	34	22	0–40	-64.52
ALT (U/L)	14	16	15	14	13	0–40	-7.14
Urea nitrogen (mg/dL)	21.0	22.4	18.6	15.2	12.8	7.0–20.0	-39.05
Creatinine (mg/dL)	0.50	0.54	0.48	0.46	0.44	0.60–1.10	-12.00
Sodium (mmol/L)	144	142	140	139	140	135–145	-2.78
Potassium (mmol/L)	3.9	3.8	3.9	4.1	4.2	3.5–5.0	+7.69
Chloride (mmol/L)	107	105	104	102	103	98–108	-3.74
Arterial pH	7.36	7.38	7.40	7.42	7.41	7.35–7.45	—
PaO ₂ (mmHg) [FiO ₂]	292 [0.70]	278 [0.65]	192 [0.40]	98 [room air]	96 [room air]	80–100	—
PaCO ₂ (mmHg)	34.0	34.8	36.2	37.5	38.0	35.0–45.0	—
Bicarbonate (mmol/L)	19.0	20.5	22.4	23.8	24.2	22.0–26.0	—

Notes: $\Delta\% = (\text{day 4} - \text{admission}) / \text{admission} \times 100$. The first gas was drawn after induction on the FiO₂ shown. ALT: alanine aminotransferase; AST: aspartate aminotransferase.

3.3. Treatment course and outcome

Intubation succeeded at the first attempt with a Cormack-Lehane grade I view, and the tube position was confirmed by capnography. Systolic pressure fell from 168 mmHg before induction to 142 mmHg after it (Figure 1). Surgery

lasted 45 minutes. A vigorous male neonate was born two minutes after intubation, weighing 3,420 g, with Apgar scores of 7, 8 and 9 at 1, 5 and 10 minutes and a cord blood pH of 7.31 (base excess -2.4 mmol/L). Blood loss was estimated at 450 mL, and the uterus contracted well.

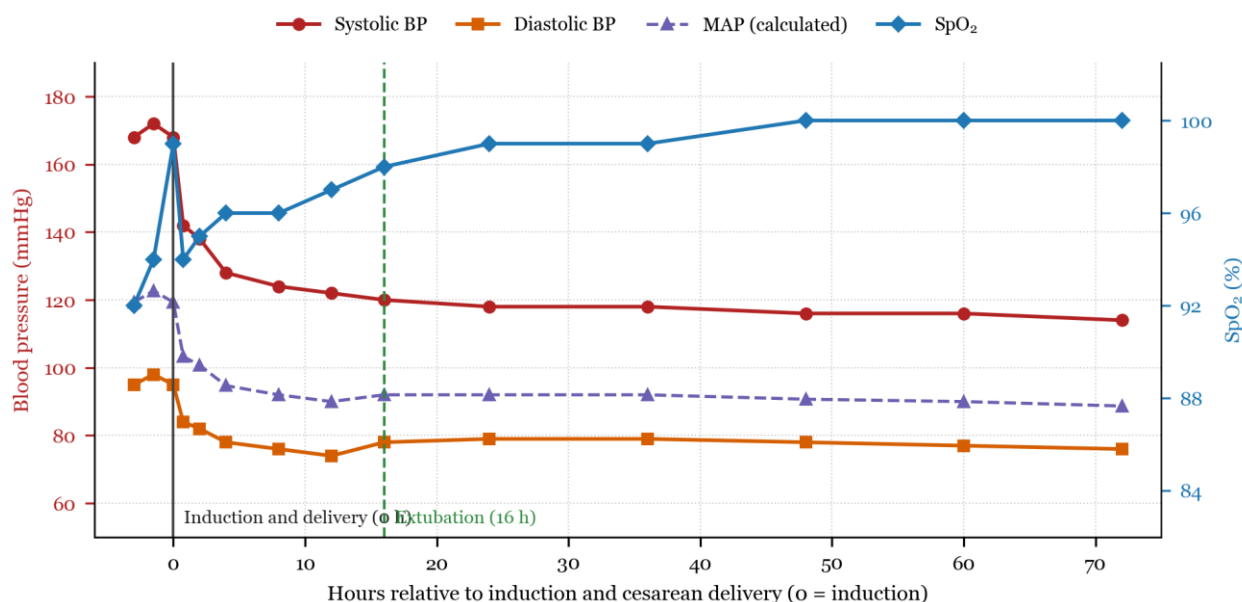


Figure 1. Blood pressure and oxygen saturation. Systolic (red circles), diastolic (orange squares) and calculated mean arterial pressure (dashed purple triangles) are plotted with SpO₂ (blue diamonds) from before induction to 72 hours. The solid vertical line marks induction and delivery and the dashed line marks extubation. Values were read from the authors' original figure.

In the ICU, with magnesium sulfate running at 1.0 g/h, three generalized tonic-clonic seizures occurred at 0.20, 0.45 and 0.65 hours after arrival and lasted 3, 5 and 5 minutes. Midazolam was started after the second seizure and phenytoin was given after the third (Table 4). The

bolus is reported as complete at 0.75 hours, and no seizure recurred afterward. The GCS rose from 10 at hour 1 to 11, 12, 13 and 14 at hours 2, 4, 8 and 12 and to 15 at hour 16, with no focal deficit (Figure 2).

Table 4. Anesthetic, anticonvulsant, diuretic and ventilator regimen.

Phase	Intervention	Dose or setting
Pre-oxygenation	Sevoflurane by mask with gentle positive pressure	2.0 MAC
Pretreatment (90 s before laryngoscopy)	Lidocaine 2%; dexamethasone	80 mg (1.2 mg/kg); 5 mg
Induction	Ketamine; propofol; rocuronium	30 mg (0.46 mg/kg); 90 mg (1.38 mg/kg); 50 mg (0.77 mg/kg)
Airway	Macintosh 3 blade, Cormack-Lehane I; 7.0-mm cuffed tube	21 cm at the lips
Maintenance	Sevoflurane in oxygen/air; furosemide infusion	1.0–1.5 MAC; 5.0 mg/h
After delivery	Oxytocin infusion; methylergonovine avoided	10 IU in 500 mL
ICU sedation and analgesia	Fentanyl infusion	15 mcg/h
Anticonvulsants	Magnesium sulfate; midazolam (after second seizure); phenytoin (after third seizure)	1.0 g/h; 1.5 mg/h; 100 mg over 20 min, then 100 mg every 12 h
Ventilation	SIMV-PS	Tidal volume 420 mL (7.7 mL/kg predicted body weight, 54 kg); PEEP 6 then 5 cmH ₂ O; pressure support 8 cmH ₂ O; rate 14/min; FiO ₂ 0.70 to 0.35; I:E 1:2
Diuresis and fluids	Furosemide infusion; crystalloid	5.0 mg/h, 2.5 mg/h from 24 h, stopped at 48 h; 50–70 mL/h
Breathing trial	CPAP with pressure support, 30 min	PEEP 5, pressure support 5 cmH ₂ O, FiO ₂ 0.35

Notes: Doses per kilogram use the admission weight of 65 kg. MAC: minimum alveolar concentration; PEEP: positive end-expiratory pressure; I:E: inspiratory-to-expiratory ratio; SIMV-PS: synchronized intermittent mandatory ventilation with pressure support.

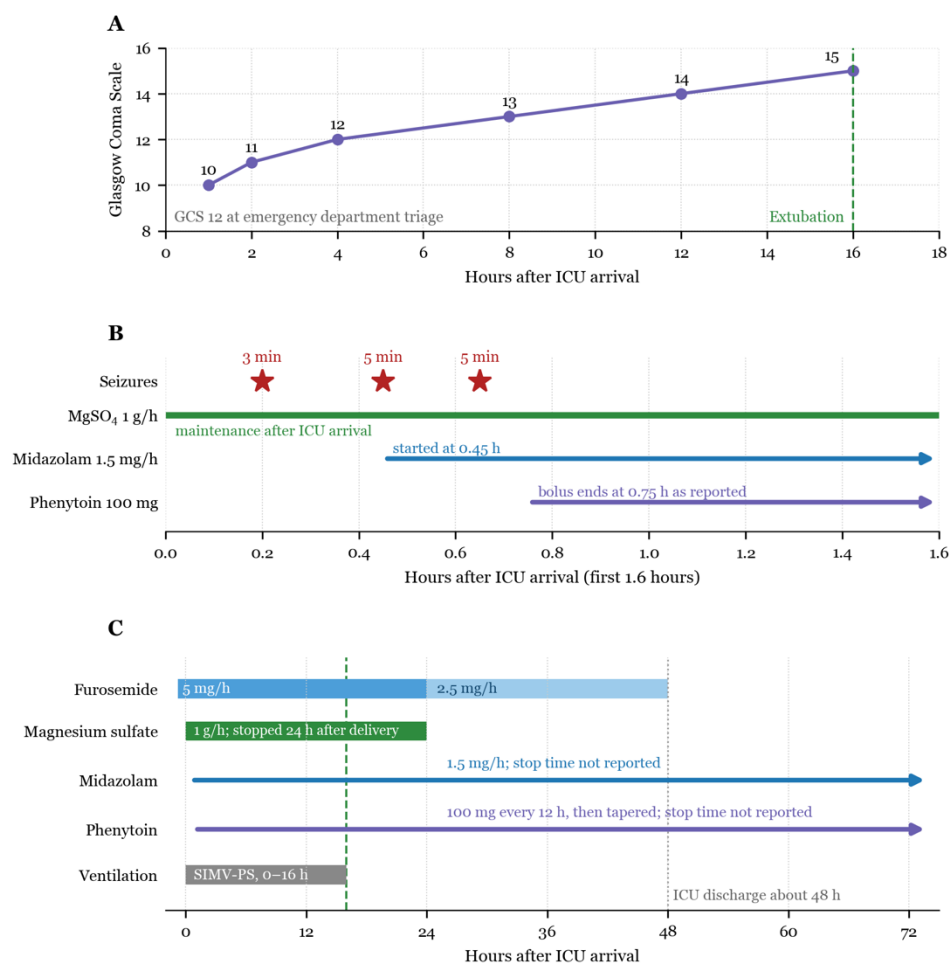


Figure 2. Neurological course and anticonvulsant timeline. (A) Glasgow Coma Scale after ICU arrival. (B) Three seizures (stars; durations shown) and the start of midazolam and phenytoin in the first 1.6 hours. (C) Duration of the main infusions, ventilation and ICU stay; arrows mean the stop time was not reported.

The FiO₂ was weaned from 0.70 to 0.65 at hour 4, 0.55 at hour 8, 0.45 at hour 12 and 0.35 at hour 16, and PEEP was lowered from 6 to 5 cmH₂O after 6 hours. Peak airway pressure stayed below 24 cmH₂O and plateau pressure below 18 cmH₂O. The authors report a PaO₂/FiO₂ ratio rising from 423 mmHg to 495 mmHg before extubation

(Figure 3). At hour 16 the rapid shallow breathing index was 36.4 breaths/min/L (16 / 0.44), and the arterial gas on FiO₂ 0.35 showed pH 7.42, PaO₂ 98 mmHg, PaCO₂ 37.5 mmHg and bicarbonate 23.8 mmol/L. She was extubated to a nasal cannula at 2 L/min with SpO₂ 99–100%.

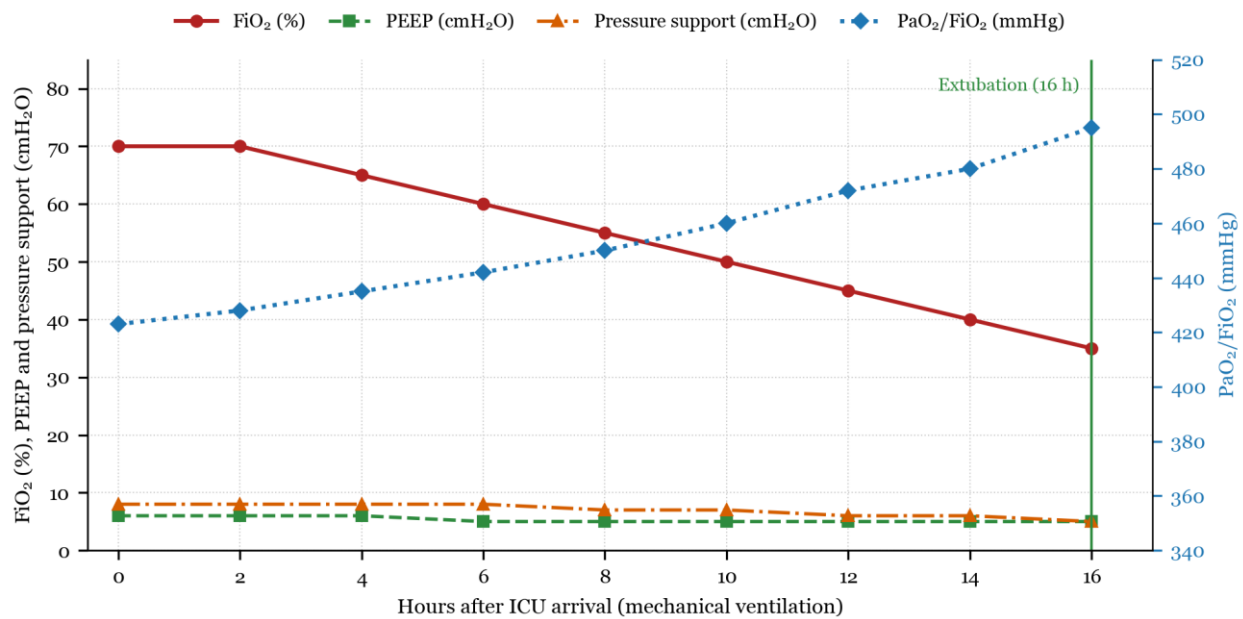


Figure 3. Ventilator weaning and oxygenation to extubation at hour 16. FiO₂ (red), PEEP (green dashed), pressure support (orange) and the PaO₂/FiO₂ ratio reported by the authors (blue dotted). Values were read from the authors' original figure.

Cumulative net fluid balance was −370 mL at 6 hours, −900 mL at 12 hours, −1,350 mL at 18 hours, −1,750 mL at 24 hours, −2,150 mL at 36 hours and −2,800 mL at 72 hours, with total intake of 3,850 mL and urine output of 6,650 mL (Table 5, Figure 4). Urine output was 2.10, 2.38 and 2.05

mL/kg/h in the first three 6-hour intervals. The basal crackles had almost cleared by hour 36. MAP fell from 119.3 mmHg to 92.0 mmHg at hour 24 and 88.7 mmHg at hour 72.

Table 5. Fluid intake, urine output and net balance after ICU arrival.

Interval after ICU arrival	Intake (mL)	Urine (mL)	Interval balance (mL)	Cumulative balance (mL)	Urine rate (mL/kg/h)	Clinical status and furosemide
0–6 h	450	820	−370	−370	2.10	5 mg/h; crackles persistent
6–12 h	400	930	−530	−900	2.38	5 mg/h; crackles resolving; FiO ₂ 50%
12–18 h	350	800	−450	−1,350	2.05	Extubated at 16 h; SpO ₂ 98% on room air
18–24 h	350	750	−400	−1,750	1.92	BP 118/79 mmHg; respiratory rate 18/min
24–36 h	650	1,050	−400	−2,150	1.35	2.5 mg/h; GCS 15; oral fluids started
36–48 h	600	850	−250	−2,400	1.09	Furosemide stopped; rales resolved
48–60 h	550	750	−200	−2,600	0.96	Transferred to ward
60–72 h	500	700	−200	−2,800	0.90	Mother and neonate rooming in

Notes: Urine rate was calculated for 65 kg. Balances were measured with an indwelling catheter and a closed urometer.

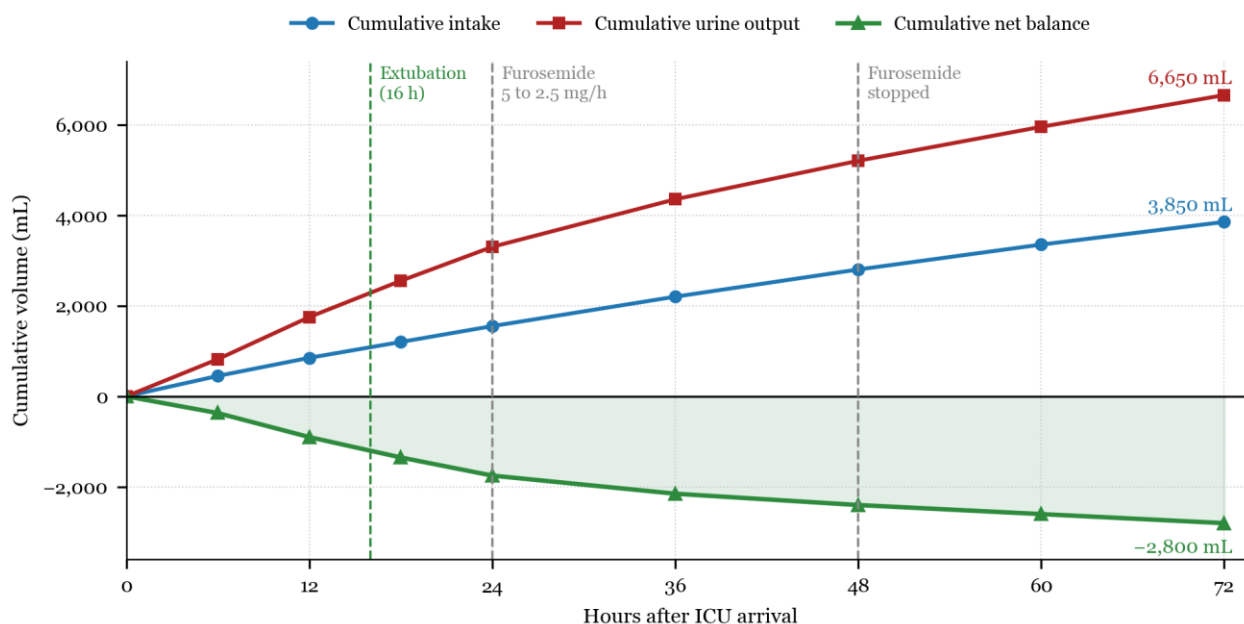


Figure 4. Cumulative intake (blue circles), urine output (red squares) and net fluid balance (green triangles, shaded) after ICU arrival, from Table 5. The final net balance was $-2,800$ mL.

Magnesium sulfate was stopped 24 hours after delivery and phenytoin was tapered. The patient moved to the obstetric ward on postoperative day 3, and mother and infant went home on postoperative day 4. At two and six weeks, her blood pressure was 118/76 mmHg without antihypertensive drugs, proteinuria was negative, renal and liver function were normal, and the infant was growing normally.

4. Discussion

4.1. Principal lesson of the case

In a parturient with eclampsia, pulmonary edema and a depressed level of consciousness, a rapid sequence general anesthetic, an ordered escalation of anticonvulsants, low tidal volume ventilation and a continuous negative fluid balance were followed by survival of mother and neonate and by full neurological recovery. Each step was chosen at the bedside, and none can be credited with the outcome on its own.

4.2. Comparison with the published literature

Magnesium sulfate is the drug of choice. In the Collaborative Eclampsia Trial it lowered recurrent convulsions by 52% compared with diazepam and by 67% compared with phenytoin,³⁵ and in the Magpie Trial it lowered the risk of eclampsia by 58% compared with placebo (Table 6).³⁶ Both trials compared single agents and did not define treatment after breakthrough seizures. Recurrence on maintenance magnesium is rarely reported. Two Indonesian women had seizures after cesarean delivery despite magnesium and were controlled with

thiopental or with repeat magnesium and midazolam,⁴⁰ and a postpartum patient who did not respond to magnesium needed alternative therapy.⁴¹ Our patient followed the same pattern, with seizures despite magnesium and control after two added agents, but the reported doses of midazolam and phenytoin are lower than usual regimens, and a magnesium loading dose is not documented (Section 2.6).

For pulmonary edema, a Brazilian obstetric ICU series of 50 women found a hypertensive cause in 62%, fluid overload in the preceding 24 hours in 34% and death in 4%.¹⁷ In cesarean delivery, propofol, thiopental and ketamine appear clinically comparable as induction agents, with low to moderate certainty of evidence.⁴⁸ In a randomized trial of 84 patients with left ventricular dysfunction, the ketamine–propofol combination used here (ketofol) produced larger falls in blood pressure and more ephedrine use than etomidate,⁴⁹ so the stable pressure in our patient (168 to 142 mmHg) should not be credited to the combination. Dennis and Solnordal recommend restricted fluid administration and venodilators and stress the aspiration and airway risks of pregnancy.²¹ In our patient the intake of 3,850 mL over 72 hours was well below the urine output of 6,650 mL. A randomized trial of 100 women with severe preeclampsia found more, but readily treated, hypotension with spinal than with epidural anesthesia for cesarean delivery;²⁴ that trial concerned regional techniques, which seizures, stupor and hypoxemia ruled out here. General anesthesia has been reported for severe preeclampsia with pulmonary edema^{25,26,50} and for refractory seizures.²⁷

Table 6. Comparison with published trials, reviews and cases.

Source	Design and size	Population	Main finding	Relevance to this case
Collaborative Eclampsia Trial ³⁵	Randomized trial, 1,687 women	Eclampsia	Magnesium sulfate lowered recurrent convulsions by 52% versus diazepam and 67% versus phenytoin	Supports magnesium first; no plan after breakthrough seizures
Magpie Trial ³⁶	Randomized trial, 10,141 women	Preeclampsia	58% lower risk of eclampsia with magnesium sulfate than with placebo	Supports magnesium prophylaxis
Visalyaputra et al. ²⁴	Randomized trial, 100 women	Severe preeclampsia, cesarean delivery	Spinal anesthesia caused more hypotension than epidural (51% versus 23%), readily treated	Regional techniques; excluded here by seizures and stupor
Dennis and Solnordal ²¹	Systematic review	Acute pulmonary edema in pregnancy	Classification by hypertension; restricted fluids and venodilators; airway and aspiration risks	Basis for fluid restriction and airway plan
Pordeus et al. ¹⁷	Case series, 50 women	Obstetric ICU, Brazil	Hypertensive cause 62%; fluid overload 34%; mortality 4%	Context for pulmonary edema outcome
Harsono et al. ⁴⁰	Two cases	Eclampsia, Indonesia	Seizures recurred on magnesium; thiopental, or repeat magnesium and midazolam, controlled them	Similar breakthrough seizures
Kalmegh et al. ⁴¹	One case	Postpartum eclampsia	No response to magnesium; alternative therapy needed	Similar breakthrough seizures
This case	One case	Eclampsia with pulmonary edema	Three seizures on magnesium, none after midazolam and phenytoin; net balance -2,800 mL; extubation at 16 h	—

Notes: GCS: Glasgow Coma Scale.

4.3. Pathophysiological or mechanistic interpretation

Figure 5 summarizes the proposed pathophysiology and the management. What was observed is a sequence: seizures that persisted on magnesium for the first 45 minutes of intensive care and stopped after midazolam and phenytoin, crackles that cleared by hour 36 during a negative balance of about 2 L, and extubation at hour 16. What is inferred is that the added anticonvulsants stopped the seizures and that diuresis cleared the lung. Neither

inference can be separated from the effect of delivery, which removes the placental source of anti-angiogenic factors, from continued magnesium, or from the natural course of eclampsia, because no serum magnesium level, electroencephalogram or neuroimaging was obtained. The PaO₂/FiO₂ ratio of 423 mmHg after induction on FiO₂ 0.70 does not meet criteria for acute lung injury, so the picture is better described as hydrostatic and permeability pulmonary edema with mild impairment of gas exchange.

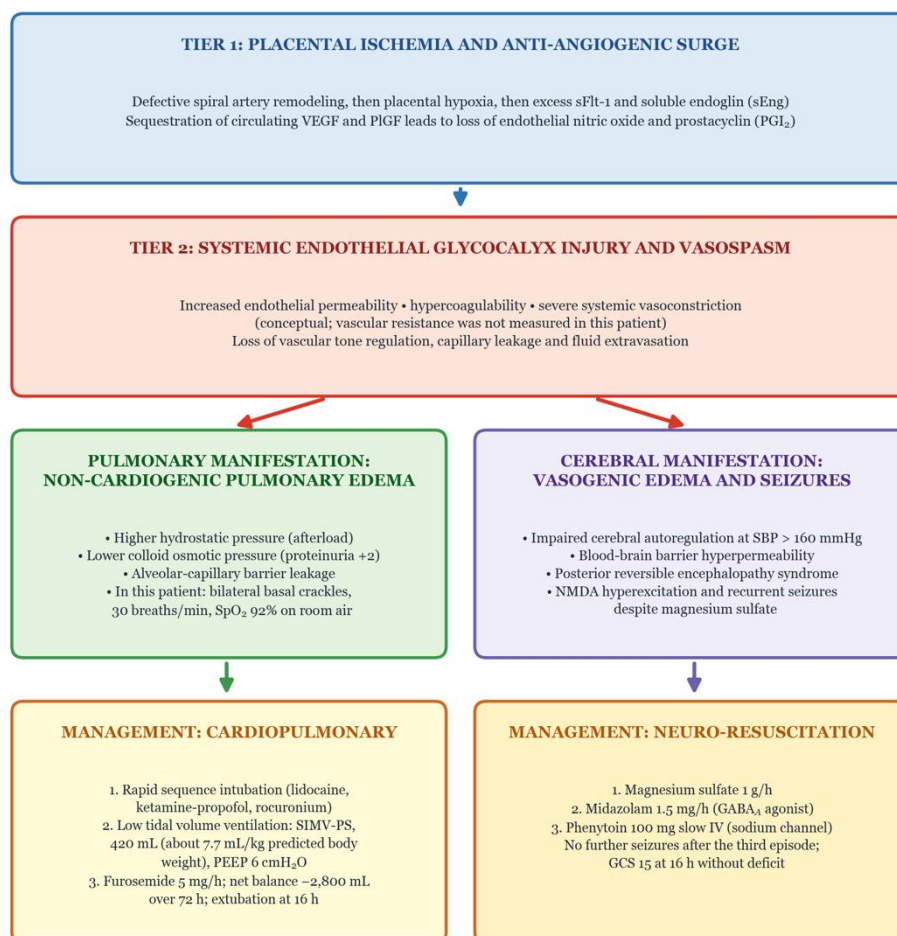


Figure 5. Conceptual summary of the pathophysiology and management. The diagram is illustrative; vascular resistance, magnesium level and cerebral imaging were not measured in this patient.

4.4. Implications for practice

A clinician facing a similar presentation can take four points. First, plan the airway early, because neuraxial block is not possible in a convulsing, drowsy and hypoxemic patient; pre-oxygenation by sevoflurane mask ventilation is a departure from standard rapid sequence practice and needs an explicit aspiration plan. Second, before escalating anticonvulsants, confirm that magnesium was adequately loaded and maintained and look for other causes of seizures.^{7,8} Third, limit intravenous fluid and use diuresis while monitoring renal function. Fourth, rely on a rehearsed, multidisciplinary emergency response, which has been associated with better outcomes,⁵¹ and on training in obstetric emergencies.⁵² In a Malawian review of maternal deaths, delays at the facility were the most frequent type of delay, so the quality of in-hospital care matters as much as transport.⁵³ Severe preeclampsia can deteriorate to cardiac arrest within hours,⁵⁴ so early recognition matters. These points come from one patient and may not transfer to other settings.

4.5. Strengths

The record is detailed: timed hemodynamics, serial laboratory values, an hourly fluid balance, ventilator settings and a defined extubation checklist are all documented, follow-up reached six weeks, and consent and ethics approval are stated.

4.6. Limitations

This is one patient, so causation, prevalence and effect cannot be established. No serum magnesium level, electroencephalogram or neuroimaging was available to confirm or classify the seizures, and no echocardiography, chest imaging or advanced hemodynamic monitoring was available to confirm non-cardiogenic edema or to measure vascular resistance. Antihypertensive treatment is not described. Several reported values required correction or remain unverified, and the timepoints were assembled from the clinical record.

4.7. Recommendations for future work

A multicenter registry of eclampsia with pulmonary edema could record magnesium dosing and levels, seizure recurrence, fluid balance and ventilator-free days, and trials of second-line anticonvulsants after magnesium failure are needed. Women who have had eclampsia should have their blood pressure followed after delivery, because postpartum trajectories vary.⁵⁵

5. Conclusion

In this patient with eclampsia, acute pulmonary edema and premature rupture of membranes, rapid sequence general anesthesia, escalation from magnesium sulfate to midazolam and phenytoin for breakthrough seizures, low tidal volume ventilation and a continuous furosemide infusion were followed by extubation at 16 hours and discharge of mother and neonate on day 4. The transferable lesson concerns the order of decisions: early airway control, a check that magnesium was adequate and that no other cause was present before escalation, and fluid restriction with diuresis. One case cannot show that any step caused the recovery.

6. Declarations

Ethical approval and consent to participate. This case report was approved by CMHC Research Center Ethics Committee, Indonesia, under reference number 2026/0518. Written informed consent to participation was obtained from the patient.

Consent for publication. Written informed consent for publication of this case and anonymized clinical data was obtained from the patient. A copy is available for review by the editor on request.

Availability of data and materials. The data supporting this case report are contained in the article. Further information is available from the corresponding author on reasonable request.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tool was used in the preparation of this manuscript.

Author contribution statement. M.I.: Conceptualization, Investigation, Data curation, Formal analysis, Writing – original draft. P.H.S.M.: Methodology, Validation, Supervision, Writing – review & editing. Both authors approved the final manuscript.

Funding statement. This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Conflict of interest (competing interests) statement. The authors declare that they have no competing interests.

Acknowledgements. The authors thank the medical, nursing and respiratory therapy staff of the Department of Anesthesiology and Intensive Care and the Department of Obstetrics and Gynecology, Kustati Islamic General Hospital, Surakarta, for their care of the patient.

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