

Case Report

Fatal Cardiopulmonary Collapse, Bilateral Pleural Effusion, and Hypoxic Encephalopathy in Neglected Severe Preeclampsia: A Case Report on Antenatal Surveillance and Access Gaps

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ABSTRACT

Background: Preeclampsia is a leading cause of maternal death worldwide. Where antenatal care (ANC) is absent and women lack health insurance, a hypertensive crisis can progress quickly to fatal multi-organ failure.

Objective: To describe the clinical, hemodynamic, and laboratory course of fatal postpartum cardiopulmonary collapse after unmonitored severe preeclampsia.

Methods: Single-patient case report (CARE guideline) at Dr. Saiful Anwar General Hospital, Malang, Indonesia. Clinical findings, serial laboratory values, arterial blood gases, echocardiography, ultrasonography, and chest radiography were recorded over nine days of intensive care and presented chronologically, with a descriptive comparison with the literature.

Results: A 44-year-old uninsured primiparous woman with no ANC contacts had an emergency cesarean delivery for severe preeclampsia and left a secondary hospital against medical advice on postoperative day 2 because of financial hardship. At tertiary admission she was obtunded (Glasgow Coma Scale 13), with blood pressure 166/94 mmHg, hemoglobin 5.5 g/dL, and oliguria. Radiography showed large asymmetric bilateral pleural effusions (left ~1,250 mL) with compressive atelectasis; PaO₂/FiO₂ was 142.1 mmHg. Echocardiography showed concentric left ventricular hypertrophy, systemic vascular resistance 2,140 dyn·s/cm⁵, and E/e' 17.8 (PCWP ~24 mmHg). Despite mechanical ventilation, nicardipine, transfusions, and albumin, anuric renal failure (creatinine 5.20 mg/dL) and severe acidosis (pH 7.08, lactate 11.4 mmol/L) progressed, and cardiopulmonary arrest occurred on day 9.

Conclusion: In this patient, unmonitored severe preeclampsia was followed by fatal cardiopulmonary collapse; hydrostatic overload and endothelial hyperpermeability are proposed mechanisms that were not directly measured. Emergency coverage at private obstetric facilities may help prevent premature discharge.

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1. Introduction

In 2014 Indonesia launched Jaminan Kesehatan Nasional (JKN), described as the world's largest single-payer national health insurance scheme, which is run by the Social Security Administrator for Health (Badan Penyelenggara Jaminan Sosial Kesehatan, BPJS Kesehatan).¹ The aim was universal health coverage (UHC). Every one of more than 275 million people across the archipelago was to receive equitable and affordable medical protection.^{1,2} For

maternal and neonatal health, JKN was designed to remove the financial barriers that had long led to catastrophic out-of-pocket spending, and it covers antenatal care (ANC), delivery, postpartum surveillance, and tertiary emergency referral.^{2,3} Even so, maternal mortality remains high. Recent national demographic surveys and global analyses put the maternal mortality rate (MMR) at 177 to 189 deaths per 100,000 live births.⁴ Indonesia ranks third in Southeast Asia for maternal mortality burden, behind only Myanmar and Laos.⁴

Hypertensive disorders of pregnancy (HDP), and severe preeclampsia in particular, are the second leading cause of direct maternal death in Indonesia. They account for roughly 25% to 33% of institutional maternal deaths.^{5,6} JKN has widened access to maternal services for tens of millions of women. Gaps remain. Recent public health audits suggest that about 88 million Indonesians are uncovered, unenrolled, or intermittently inactive in the BPJS Kesehatan database, because of administrative bottlenecks, unstable informal employment, and regional differences in enrollment.^{1,6} The gap deepens socioeconomic inequity in the use of maternal care and leaves poor mothers exposed to severe outcomes.^{2,3} Without continuous insurance, pregnant women often attend antenatal visits irregularly, present late in an obstetric emergency, or decline hospital admission because they fear unaffordable bills.^{7,8}

Antenatal care is the foundation of preventive obstetrics. It allows early risk stratification, detection of subclinical hypertensive disease, and timely treatment.^{9–11} Indonesian Ministry of Health Regulation No. 97 of 2014 and the updated World Health Organization (WHO) antenatal guidelines set a minimum schedule of contacts: at least one in the first trimester, two in the second, and three or more in the third.^{10,12} At these visits clinicians measure baseline blood pressure, calculate mean arterial pressure (MAP), screen for proteinuria, and assess uterine artery Doppler waveforms. In high-risk women they can also start low-dose aspirin (150 mg daily) and calcium supplementation before 16 weeks of gestation.^{13–15} Skipping antenatal care altogether is dangerous. Hypertensive disease then advances unchecked, from asymptomatic endothelial dysfunction to fulminant multi-organ crisis, and this is most likely in high-risk women, particularly those of advanced maternal age (≥ 35 years).^{16,17}

Preeclampsia is a multisystem microvascular disorder. Its features are abnormal placentation, syncytiotrophoblast stress, and systemic angiogenic imbalance.^{18–20} In normal pregnancy, extravillous trophoblasts invade the maternal decidua and myometrium and remodel the high-resistance, low-capacity spiral arteries into low-resistance, high-capacitance vessels.^{21,22} In preeclampsia the endovascular invasion is shallow. Placental perfusion is compromised. Ischemia-reperfusion injury persists, and oxidative stress is intense.^{18,23} The stressed syncytiotrophoblast releases large amounts of anti-angiogenic factors into the maternal circulation, chiefly soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng).^{24,25} This report describes the clinical course, the laboratory and imaging findings, and the health system context of a fatal case of neglected severe preeclampsia.

2. Methods

2.1. Study design and setting

This is a single-patient case report of a postpartum woman followed for nine days. It tracks the clinical course, laboratory and imaging findings, and the dynamics of multi-organ failure in a patient with neglected severe preeclampsia complicated by fatal cardiopulmonary collapse, and it discusses the pathophysiological cascade that this course suggests. Reporting followed the CARE guideline for case reports.²⁶ The setting comprised the Emergency Department Resuscitation Unit (P1), the Intensive Care Unit (ICU), and the Division of Maternal-Fetal Medicine at Dr. Saiful Anwar General Hospital (RSSA), Malang, East Java, Indonesia. RSSA is a tertiary university-affiliated academic medical center and the main referral hub for a regional population of more than 15 million in East Java.

2.2. Case definition and diagnostic criteria

Diagnoses were made with standard clinical criteria for severe preeclampsia, hypertensive emergency, acute respiratory distress syndrome (ARDS), acute kidney injury (AKI), and severe anemia.^{27–31} Severe preeclampsia and hypertensive emergency were defined by the International Society for the Study of Hypertension in Pregnancy (ISSHP) 2021/2022 guidelines and the criteria of the American College of Obstetricians and Gynecologists (ACOG). Severe-range blood pressure (systolic ≥ 160 mmHg or diastolic ≥ 110 mmHg) with concurrent end-organ damage (renal, neurological, hepatic, or hematological) was required.^{27,28} Thresholds for the sFlt-1 to placental growth factor (PlGF) ratio were taken from recent multicenter validated cut-offs.³² ARDS followed the Berlin Definition. This requires onset within one week of a clinical insult, bilateral opacities on chest radiography not fully explained by volume overload, and hypoxemia with a $\text{PaO}_2/\text{FiO}_2$ ratio ≤ 200 mmHg at positive end-expiratory pressure (PEEP) ≥ 5 cmH₂O.³⁰ AKI was staged with the Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Doubling of serum creatinine and absolute oliguria (< 0.5 mL/kg/h for > 12 hours) were used.³¹ Severe postpartum anemia was defined by WHO as a hemoglobin concentration < 7.0 g/dL.³³

2.3. Data collection

Observation began at triage in the RSSA Emergency Department (Day 1) and continued without interruption until the terminal cardiopulmonary arrest on Day 9 of intensive care.²⁶ Monitoring was standardized and comprised:

1. Continuous hemodynamic and neurological monitoring: Invasive and non-invasive arterial blood pressure, heart rate, electrocardiographic telemetry, respiratory rate, peripheral oxygen saturation (SpO_2), Glasgow Coma Scale (GCS), and hourly diuresis through an indwelling Foley catheter connected to an electronic closed-system meter.³⁴ MAP was calculated as $\text{MAP} = \text{DBP} + 1/3(\text{SBP} - \text{DBP})$.
2. Serial arterial blood gases (ABG) and gas exchange: Samples were drawn anaerobically from an indwelling radial arterial line and analyzed at once on a calibrated point-of-care analyzer (Cobas b 123, Roche Diagnostics, Basel, Switzerland).³⁵ The variables were arterial pH, partial pressure of arterial oxygen (PaO_2), partial pressure of arterial carbon dioxide (PaCO_2), serum bicarbonate (HCO_3^-), base excess (BE), and blood lactate. The oxygenation index was the $\text{PaO}_2/\text{FiO}_2$ (P/F) ratio in mmHg.
3. Serial hematological, biochemical, and coagulation panels: Venous blood was drawn daily and processed to accredited ISO 15189 laboratory standards.³⁶ Assays covered automated complete blood counts with leukocyte differential (Sysmex XN-1000, Kobe, Japan), serum albumin (bromocresol green method), total protein, blood urea nitrogen, enzymatic serum creatinine (isotope dilution mass spectrometry-aligned), liver transaminases (AST, ALT), total and direct bilirubin, D-dimer (latex-enhanced immunoturbidimetry), prothrombin time (PT), activated partial thromboplastin time (aPTT), and plasma fibrinogen (Clauss method).
4. Diagnostic imaging and point-of-care ultrasound (POCUS): Bedside chest radiographs were taken with a high-frequency digital mobile unit (MobileArt Evolution, Shimadzu Corporation, Kyoto, Japan).³⁷ Two independent senior board-certified radiologists read the images for cardiothoracic ratio (CTR), pleural fluid meniscus depth, perihilar alveolar infiltrates, and compressive atelectasis. Pelvic and abdominal ultrasonography used a multi-

frequency convex transducer (3.5–5.0 MHz, GE Logiq S8, GE Healthcare, Chicago, IL, USA) to assess uterine dimensions, intracavitary echotexture, and dependent free fluid in the hepatorenal space (Morison's pouch), the splenorenal recess, and the pouch of Douglas.³⁸ A critical care cardiologist performed bedside transthoracic echocardiography (TTE) with a phased-array probe (1.5–4.6 MHz, Philips Affiniti 70, Philips Healthcare, Eindhoven, Netherlands). Measurements were left ventricular ejection fraction (LVEF, biplane Simpson method), left ventricular mass index (LVMI), relative wall thickness (RWT), the pulsed-wave Doppler mitral inflow early-to-late filling velocity ratio (E/A), the tissue Doppler septal mitral annular early diastolic velocity (e'), the E/e' ratio as an index of left ventricular filling pressure, tricuspid regurgitant jet velocity (TR Vmax), and estimated pulmonary artery systolic pressure (PASP).^{39–41}

2.4. Comparison with the literature

To place the findings in the wider literature, the case was compared descriptively with published cohorts of severe maternal morbidity (SMM), maternal near-miss events, and hypertensive critical care admissions.⁴² Because the report concerns one patient, no causal inference is drawn and no hypothesis test is applied.

2.5. Data presentation

Continuous variables are reported as absolute values and as percentage change from baseline ($\Delta\%$), calculated as:

$$\Delta\% = [(V_{\text{observed}} - V_{\text{baseline}}) / V_{\text{baseline}}] \times 100\%$$

Hourly urine output (mL/h and mL/kg/h) and systemic vascular resistance (SVR) were derived from standard physiological equations:

$$SVR = [(MAP - CVP) \times 80] / CO$$

Here MAP is mean arterial pressure in mmHg, CVP is central venous pressure in mmHg (estimated from inferior vena cava ultrasound), and CO is cardiac output in L/min from Doppler echocardiography. No statistical tests were performed.

2.6. Ethical considerations

CMHC Research Center Ethics Committee, Indonesia, reviewed the protocol and gave approval (Protocol Approval Number: 2026/0852). All procedures, including specimen collection, imaging, and clinical monitoring, followed the Declaration of Helsinki (revised 2013).⁴³ They also met the Good Clinical Practice guidelines of the International Council for Harmonisation of Technical Requirements for Pharmaceuticals for Human Use (ICH).⁴⁴ The patient was encephalopathic on arrival and later died. Her husband and next of kin therefore gave written informed consent. The consent covered longitudinal recording, analysis, and publication of anonymized clinical data, serial biochemical parameters, physiological waveforms, and radiographic images. In line with ICMJE and COPE publication ethics, the patient's initials, hospital record numbers, and demographic markers were removed.

3. Results

3.1. Sociodemographic profile, obstetric risk, and baseline presentation

The patient was a 44-year-old woman, primiparous (G1P1A0). She arrived at the Emergency Department of Dr.

Saiful Anwar General Hospital (RSSA) on postoperative day 3 after an emergency cesarean delivery. She was uninsured and had never been registered with the national health insurance scheme (BPJS Kesehatan / JKN). Throughout the index pregnancy she had no antenatal care contacts (0 visits) with a midwife, general practitioner, or obstetric specialist, so there was no routine surveillance at all (Table 1). Her pregnancy had been complicated by unmonitored hypertension, and she took self-purchased oral nifedipine (10 mg three times daily) intermittently, without medical supervision or dose adjustment.^{45–47}

Three days before tertiary presentation she developed severe headache, scotomata, epigastric distress, and a sharp rise in blood pressure. She went to the emergency room of a private secondary hospital in Malang. Severe preeclampsia (preeclampsia berat, PEB) with hypertensive emergency was diagnosed. An emergency lower segment cesarean section followed. The infant was a live girl weighing 2,520 grams, borderline low birth weight, in keeping with chronic placental insufficiency and the risk of advanced maternal age.^{16,23} After surgery the patient went to the general ward. On postoperative day 2 she still had dyspnea, orthopnea, and blurred vision, yet she and her family insisted on discharge against medical advice. The main reason was financial. She had no BPJS Kesehatan coverage, so emergency surgery, inpatient bed charges, and medications at the secondary hospital were catastrophic out-of-pocket costs that the family could not meet.^{1,3}

Her condition worsened sharply over the next 24 hours at home. Dyspnea progressed to severe air hunger with heavy use of accessory muscles. Blurred vision became near-total visual loss. She grew lethargic and confused, then obtunded, as expected in unmanaged hypertensive crisis.⁴⁸ Family members brought her to the Emergency Department Resuscitation Unit (P1) at RSSA. At triage she was in severe respiratory and neurological distress, with an altered sensorium and a Glasgow Coma Scale (GCS) score of 13 (Eye 4, Verbal 4, Motor 5). Blood pressure was 166/94 mmHg, a hypertensive emergency, giving a mean arterial pressure (MAP) of 118.0 mmHg; the systolic pressure is 38.3% above the upper normal limit of 120 mmHg. Heart rate was 116 beats/min and respiratory rate 38 breaths/min, with prominent intercostal and supraclavicular retractions. She was febrile at 38.2°C. Peripheral oxygen saturation was 100% on high-flow nasal cannula (HFNC) oxygen at 60 L/min with an inspired oxygen fraction (FiO₂) of 0.80 (Table 1).

On cardiovascular examination the heart sounds were distant and there was a gallop rhythm but no murmur. The jugular veins were distended to the angle of the mandible at 45 degrees. Auscultation of the lungs revealed coarse crackles through the mid and upper zones bilaterally, with greatly reduced vesicular breath sounds and stony dullness to percussion over the left hemithorax. The uterine fundus was subinvolved, palpable two fingerbreadths below the umbilicus, with firm contractions. The Pfannenstiel incision was intact, without purulent discharge. The lower abdomen was distended. Shifting dullness indicated ascites. On pelvic examination there was no active external vaginal bleeding and the cervix was closed. An indwelling Foley catheter drained less than 15 mL of dark, concentrated amber urine in the first hour, confirming critical oliguria. Diffuse anasarca was evident, with 3+ bilateral pitting pedal and pretibial edema (Table 1).

Table 1. Baseline sociodemographic and obstetric profile and clinical presentation at tertiary admission.

Clinical Parameter / Domain	Observational Finding (the patient)	Physiological Reference / Normal Baseline	Clinical Interpretation & Risk Stratification
Maternal Age, years	44	20–34 years	Advanced maternal age (AMA); independent high-risk factor for preeclampsia
Gravidity and Parity	G1P1A0 (Postpartum Day 3)	Nulliparous prior to index delivery	Primiparity combined with AMA confers synergistic preeclampsia hazard
Antenatal Care (ANC) Visits	0 visits (Complete absence of ANC)	≥ 6–8 contacts (WHO/MOH Standard)	Neglected antenatal surveillance; missed early screening and aspirin prophylaxis
Health Insurance Status	Uninsured (Non-BPJS JKN beneficiary)	Universal Health Coverage (BPJS JKN)	Out-of-pocket financial distress drove premature discharge against medical advice
Initial Emergency CS Indication	Severe preeclampsia & hypertensive crisis	Elective or normotensive labor	Acute maternal compromise at secondary community facility
Neonatal Birth Weight, grams	2,520	2,500–4,000 g	Borderline low birth weight; suggests chronic uteroplacental hypoperfusion
Discharge Timeline & Delay	Discharged against medical advice (D2 post-CS)	Minimum 72–96 h in-hospital monitoring	Outpatient gap with unmonitored symptom exacerbation
Presenting Neurological Status (GCS)	13 (E4-V4-M5), blurred vision, obtundation	15 (E4-V5-M6, Fully alert)	Acute hypertensive encephalopathy vs cytotoxic/vasogenic cerebral edema
Systolic Blood Pressure, mmHg	166	90–120 mmHg	Severe systolic hypertensive emergency (+38.3% above normal ceiling)
Diastolic Blood Pressure, mmHg	94	60–80 mmHg	Diastolic hypertension requiring intravenous vasodilatory titration
Mean Arterial Pressure (MAP), mmHg	118.0	70–105 mmHg	At or above the proposed upper limit of cerebral autoregulation (> 115 mmHg)
Heart Rate, beats/min	116 (Sinus tachycardia)	60–100 beats/min	Compensatory tachycardic response to severe anemia, hypoxia, and pain
Respiratory Rate, breaths/min	38 (Marked tachypnea / retractions)	12–20 breaths/min	Acute impending respiratory muscle exhaustion secondary to pulmonary edema
Peripheral Oxygen Saturation (SpO ₂)	100% on HFNC (60 L/min, FiO ₂ 0.80)	95–100% on room air (FiO ₂ 0.21)	Underlying hypoxemia masked by high-flow oxygen delivery
Uterine Involution & Bleeding	Fundus 2 fingerbreadths below umbilicus	Expected puerperal involution	Subinvolved uterine contour with intracavitary hematoma retention on USG
Hourly Urine Output, mL/h	< 15 mL/h (Oliguria)	> 30 mL/h (> 0.5 mL/kg/h)	Severe acute oliguric renal dysfunction (KDIGO Stage 2/3 criteria)
Systemic Edema Distribution	Bilateral pedal, presacral, and anasarca	None or minimal dependent edema	Extensive third-space fluid sequestration and hypoalbuminemic capillary leak

Notes: AMA, advanced maternal age; ANC, antenatal care; BPJS JKN, Badan Penyelenggara Jaminan Sosial Jaminan Kesehatan Nasional; CS, cesarean section; GCS, Glasgow Coma Scale; HFNC, high-flow nasal cannula; KDIGO, Kidney Disease: Improving Global Outcomes; MAP, mean arterial pressure. Measurements were documented on admission to the Emergency Department at Dr. Saiful Anwar General Hospital (RSSA). Hb, hemoglobin; E4-V4-M5, eye, verbal, and motor components of the GCS.

3.2. Thoracic imaging

A portable anteroposterior supine chest radiograph on admission showed severe cardiopulmonary decompensation (Figure 1). The heart was markedly enlarged, with a cardiothoracic ratio (CTR) of 0.62. A dense, homogeneous opacity filled the lower two-thirds of the left hemithorax. It obliterated the left hemidiaphragmatic contour and the left costophrenic angle (positive silhouette sign). The left mid-lung showed dense compressive atelectasis with air bronchograms (Figure 1). The right lung showed prominent

perihilar vascular engorgement in a "batwing" distribution, typical of alveolar-interstitial pulmonary edema, with blunting of the right lateral costophrenic sulcus. Quantitative radiologic analysis estimated the left pleural effusion at approximately 1,250 mL and the right at 400 mL (Figure 1). The trachea and upper mediastinum were displaced 4.0 mm to the right, indicating significant mass effect from the left hydrothorax. The initial Murray Lung Injury Score was 3.25. That signifies severe lung injury on the Murray scale.⁴⁹

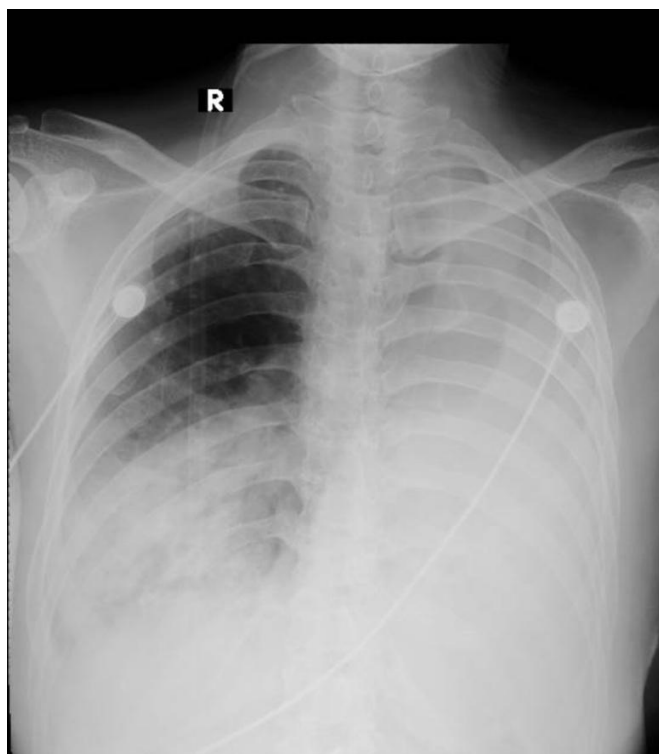


Figure 1. Chest radiograph on admission. Thorax radiology showed bilateral pleural effusion (left dominant) with suspicion of left lung atelectatic component and pneumonia. The portable anteroposterior supine chest radiograph obtained on ICU Day 1 shows severe cardiomegaly (CTR 0.62), marked perihilar batwing vascular congestion, and dense homogeneous opacification of the left middle and lower hemithoraces with positive silhouette sign, indicative of a massive left-sided pleural effusion (~1,250 mL) and compressive atelectasis. Note the 4 mm rightward contralateral mediastinal shift and the monitoring leads. Patient identifying details have been removed in line with ICMJE ethics.

3.3. Longitudinal laboratory dynamics and multiorgan dysfunction

Serial laboratory tests over the nine-day stay documented progressive multi-organ failure. The features were severe anemia, pronounced leukocytosis, hypoalbuminemia, acute kidney injury, hepatic congestion, and uncorrectable metabolic acidosis (Table 2).

At baseline (Day 1) the patient had life-threatening normocytic normochromic anemia, with a hemoglobin of 5.5 g/dL (normal: 12.0–15.5 g/dL; Δ -54.2%), a hematocrit of 16.8%, and severe leukocytosis. The white blood cell (WBC) count was $24,390/\text{mm}^3$, with 88.0% neutrophils (absolute neutrophil count $21,460/\text{mm}^3$) (Table 2). The platelet count was $182 \times 10^3/\text{mm}^3$. She received three units of leukoreduced packed red cells (PRC) between Day 1 and Day 3. Hemoglobin rose briefly to 8.4 g/dL (+52.7% above nadir), then fell to 8.1 g/dL on Day 5, 7.5 g/dL on Day 7, and 6.8 g/dL shortly before death on Day 9. Platelets declined steadily, a pattern of consumptive thrombocytopenia: $182 \times 10^3/\text{mm}^3$ at admission, $148 \times 10^3/\text{mm}^3$ on Day 3, $126 \times 10^3/\text{mm}^3$ on Day 5, $94 \times 10^3/\text{mm}^3$ on Day 7. The nadir was $58 \times 10^3/\text{mm}^3$ on Day 9 (-68.1%). Plasma D-dimer was already high at admission, 13.3 $\mu\text{g/mL}$ FEU (normal: < 0.50 $\mu\text{g/mL}$ FEU; 26.6 times the upper reference limit). This reflects intravascular fibrin formation with secondary fibrinolysis (Table 2). It fell briefly to 8.2 $\mu\text{g/mL}$ FEU on Day 5 after initial stabilization. Then it rose to 14.6 $\mu\text{g/mL}$ FEU on Day 7 and peaked at 22.4 $\mu\text{g/mL}$ FEU on Day 9 (+68.4% above admission), suggesting consumptive coagulopathy.⁵⁰

Serum protein chemistry showed severe capillary leak and loss of oncotic pressure. Albumin was low at admission, 2.75 g/dL (normal: 3.50–5.00 g/dL), reflecting

extensive transcapillary protein loss (Table 2). Daily infusions of 20% human albumin (100 mL) and oral concentrated VIP albumin extract (*Ophiocephalus striatus*) did not help. Albumin reached only 2.92 g/dL on Day 5, then dropped to 2.40 g/dL on Day 7 and 1.95 g/dL on Day 9 (-29.1% from admission). Total protein followed the same course, falling from 5.10 g/dL to 4.10 g/dL (Table 2).

Renal markers showed rapidly progressive acute kidney injury meeting KDIGO Stage 3. Creatinine was 1.84 mg/dL at admission (estimated GFR $31.4 \text{ mL/min/1.73 m}^2$). It rose to 2.45 mg/dL on Day 3, 2.90 mg/dL on Day 5, 3.85 mg/dL on Day 7, and a terminal 5.20 mg/dL on Day 9 (+182.6%; eGFR $9.4 \text{ mL/min/1.73 m}^2$) (Table 2). Blood urea nitrogen climbed in parallel from 38.5 mg/dL to 96.5 mg/dL (+150.6%). Urine output stayed severely reduced. It rose briefly from 18.5 mL/h to 30.5 mL/h on Day 3 after furosemide and albumin were given together, then fell to 25.0 mL/h on Day 5 and 12.0 mL/h on Day 7. In the final 24 hours urine output was 4.2 mL/h (about 100 mL/24 h; < 0.08 mL/kg/h), at the threshold for anuria.

Transaminases pointed to secondary ischemic and congestive liver injury. Aspartate aminotransferase (AST) rose from 68 U/L at admission to 118 U/L on Day 7 and then to a peak of 245 U/L (+260.3%). Alanine aminotransferase (ALT) rose from 52 U/L to 182 U/L (+250.0%) (Table 2). Total bilirubin increased late, from 1.24 mg/dL at baseline to 3.42 mg/dL on Day 9.

Arterial blood gases showed uncorrectable, fatal metabolic acidosis with respiratory failure. On admission arterial pH was 7.30 and PaO_2 was 113.7 mmHg on HFNC (FiO_2 0.80). The resulting $\text{PaO}_2/\text{FiO}_2$ ratio of 142.1 mmHg established moderate-to-severe ARDS (Table 2). PaCO_2 was 32.1 mmHg, serum bicarbonate 15.4 mEq/L, base excess -9.2 mEq/L, and arterial lactate 3.4 mmol/L (normal: 0.5–1.6

mmol/L). After endotracheal intubation and lung-protective ventilation on Day 2, pH and oxygenation improved for a time. By Day 5, pH was 7.38, PaO₂/FiO₂ 182.0 mmHg, bicarbonate 21.2 mEq/L, and lactate 2.1 mmol/L. Then came a severe secondary septic and cardiopulmonary deterioration on Day 7. pH fell to 7.24, PaO₂/FiO₂ to 112.5 mmHg, and base excess to -11.6 mEq/L, while lactate rose to 5.8 mmol/L. Shortly before death on Day 9, acidemia and

hypoxic exhaustion were severe: pH 7.08, PaO₂/FiO₂ ratio 54.2 mmHg at its lethal nadir (severe ARDS; Δ -61.9% from admission), PaCO₂ 58.2 mmHg because of dead-space ventilation, bicarbonate 9.8 mEq/L, base excess -19.4 mEq/L, and lactate 11.4 mmol/L (+235.3% above admission). These values suggest severe tissue hypoperfusion and circulatory failure (Table 2).

Table 2. Longitudinal laboratory biomarkers, arterial blood gas values, and multiorgan function over nine days.

Biomarker / Assay (Units)	Day 1 (RSSA admission)	Day 3 (Post-PRC Tx)	Day 5 (ICU Plateau)	Day 7 (Septic Surge)	Day 9 (Terminal Collapse)	Reference Interval	Analytical Assay Platform
Hemoglobin (Hb), g/dL	5.5	8.4	8.1	7.5	6.8	12.0–15.5 g/dL	Automated SLS-Hemoglobin Photometry
Hematocrit (Hct), %	16.8	25.2	24.5	22.8	20.4	36.0–46.0 %	Automated Cumulative Pulse Height
White Blood Cells (WBC), /mm ³	24,390	18,200	16,400	28,600	34,200	4,500–11,000 /mm ³	Flow Cytometry Semiconductor Laser
Absolute Neutrophil Count, /mm ³	21,460	15,100	13,450	25,170	30,780	1,800–7,700 /mm ³	Automated Leukocyte Differential
Platelet Count, $\times 10^3$ /mm ³	182	148	126	94	58	150–450 $\times 10^3$ /mm ³	Direct Impedance Aperture Count
Serum Albumin, g/dL	2.75	2.85	2.92	2.40	1.95	3.50–5.00 g/dL	Bromocresol Green (BCG) Colorimetry
Serum Total Protein, g/dL	5.10	5.45	5.60	4.80	4.10	6.40–8.30 g/dL	Biuret Alkaline Copper Method
Serum Creatinine, mg/dL	1.84	2.45	2.90	3.85	5.20	0.50–1.10 mg/dL	Enzymatic Jaffe-Compensated Assay
Blood Urea Nitrogen (BUN), mg/dL	38.5	46.2	54.8	72.4	96.5	7.0–20.0 mg/dL	Urease-Glutamate Dehydrogenase UV
Estimated GFR, mL/min/1.73 m ²	31.4	22.8	18.6	13.2	9.4	> 90 mL/min/1.73 m ²	CKD-EPI Creatinine Equation 2021
Aspartate Aminotransferase (AST), U/L	68	54	62	118	245	10–35 U/L	IFCC Kinetic UV without Pyridoxal
Alanine Aminotransferase (ALT), U/L	52	44	48	86	182	10–35 U/L	IFCC Kinetic UV without Pyridoxal
Total Bilirubin, mg/dL	1.24	1.10	1.18	1.85	3.42	0.20–1.20 mg/dL	Diazo Sulfanilic Acid Photometry
D-Dimer, μ g/mL FEU	13.3	9.8	8.2	14.6	22.4	< 0.50 μ g/mL FEU	Particle-Enhanced Immuno-turbidimetric
Prothrombin Time (PT), seconds	12.8	13.0	13.4	16.8	21.5	11.0–14.0 s	Electromechanical Clotting Detection
Activated PTT (aPTT), seconds	32.4	33.1	34.0	42.5	56.8	25.0–35.0 s	Optical Clotting Sensor Platform
Plasma Fibrinogen, mg/dL	420	380	340	220	145	200–400 mg/dL	Clauss Quantitative Thrombin Clotting
Arterial pH	7.30	7.35	7.38	7.24	7.08	7.35–7.45	Direct Potentiometric Blood Gas Analyzer
Arterial PaO ₂ , mmHg	113.7	115.6	109.2	78.8	48.8	80.0–100.0 mmHg	Clark Polarographic Electrode
Arterial PaCO ₂ , mmHg	32.1	34.5	36.2	44.5	58.2	35.0–45.0 mmHg	Severinghaus Potentiometric Electrode
Serum Bicarbonate (HCO ₃ ⁻), mEq/L	15.4	18.8	21.2	14.2	9.8	22.0–26.0 mEq/L	Calculated Henderson-Hasselbalch
Base Excess (BE), mEq/L	-9.2	-5.4	-2.8	-11.6	-19.4	-2.0 to +2.0 mEq/L	Calculated Siggaard-Andersen Curve
Arterial Lactate, mmol/L	3.4	2.6	2.1	5.8	11.4	0.5–1.6 mmol/L	Enzymatic Amperometric Biosensor
PaO ₂ /FiO ₂ (P/F Ratio), mmHg	142.1	165.2	182.0	112.5	54.2	> 400 mmHg	Physiological Gas Exchange Calculation

Notes: FEU, fibrinogen equivalent units; GFR, glomerular filtration rate; IFCC, International Federation of Clinical Chemistry; PaO₂/FiO₂, ratio of arterial oxygen tension to fraction of inspired oxygen; PRC, packed red cells; SLS, sodium lauryl sulfate. All assays were processed under accredited ISO 15189 clinical laboratory protocols at Dr. Saiful Anwar General Hospital.

3.4. Pelvic and abdominal ultrasonography

Bedside pelvic and abdominal ultrasonography showed disease in several compartments (Figure 2). The puerperal uterus was enlarged and subinvolved, measuring 14.8 $\times$ 9.2 $\times$ 8.4 cm. The endometrial cavity was distended by a heterogeneous mass of mixed hypoechoic and hyperechoic texture measuring 5.2 $\times$ 3.6 cm, suggestive of retained intrauterine coagula and hematoma (hematometra) (Figure 2A). Myometrial color Doppler showed raised vascular resistance (resistance index RI = 0.78). Point-of-care ultrasound found extensive anechoic fluid in the abdomen: a 1.8 cm stripe in Morison's pouch (hepatorenal recess), a 1.2 cm

rim in the splenorenal space, and about 320 mL of free ascites in the pouch of Douglas (Figure 2B). The bladder was collapsed (internal volume < 15 mL), in keeping with the oliguria already recorded. Lung ultrasound (POCUS) after intubation showed confluent, coalescent B-lines (> 6 lines per rib interspace, "lung rockets") across the anterior and lateral zones. The left posterolateral base showed a prominent "quad sign" and "sinusoid sign", indicating a large left pleural effusion with adjacent compressive consolidation. Inferior vena cava (IVC) ultrasound showed a diameter of 2.3 cm with a collapsibility index below 15%, suggesting raised central venous pressure and volume overload (Figure 2B).^{41,51}

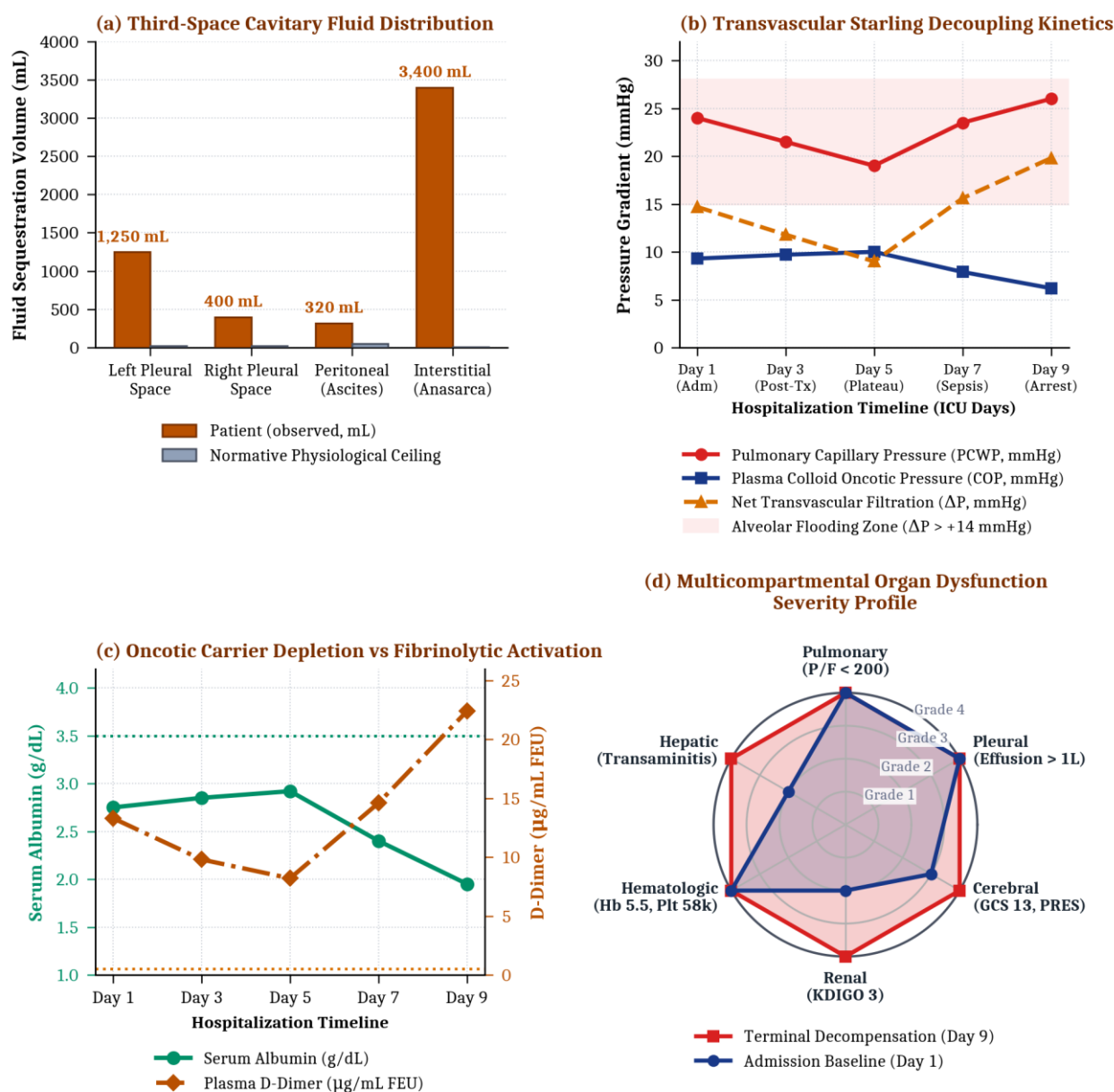


Figure 2. Multicompartmental third-space fluid sequestration, transvascular Starling forces, and organ dysfunction. (a) Third-space cavity fluid distribution: massive pleural effusion (left 1,250 mL, right 400 mL), peritoneal ascites (320 mL), and interstitial anasarca (~3,400 mL) versus physiological baselines. (b) Transvascular Starling kinetics: the widening gradient between elevated pulmonary capillary wedge pressure (PCWP 24 mmHg) and reduced plasma colloid oncotic pressure (COP nadir 9.3 mmHg). (c) Inverse relationship between serum albumin (2.75 to 1.95 g/dL) and D-dimer (peak 22.4 $\mu\text{g/mL FEU}$). (d) Organ dysfunction severity profile across the nine-day intensive care course.

3.5. Bedside echocardiography and hemodynamics

Bedside transthoracic echocardiography on Day 3 showed a preserved left ventricular ejection fraction (LVEF 58.4%, biplane Simpson method) with marked concentric remodeling and severe diastolic dysfunction (Figure 3). Interventricular septal thickness in diastole (IVSd) was 13.8 mm (normal: 6–10 mm), posterior wall thickness (PWd) 12.6 mm, and relative wall thickness (RWT) 0.54, indicating concentric left ventricular remodeling (Figure 3A). Pulsed-wave Doppler of mitral inflow gave an E/A ratio of 0.65 with an E-wave deceleration time of 260 ms. Tissue Doppler of the septal mitral annulus showed a severely reduced e' velocity of 4.2 cm/s. The E/ e' ratio was therefore high at 17.8 (normal: < 14), corresponding to an estimated

pulmonary capillary wedge pressure (PCWP) of about 24 mmHg (Figure 3B). Continuous-wave Doppler of the tricuspid regurgitant jet gave a peak velocity (TR Vmax) of 3.4 m/s. Estimated pulmonary artery systolic pressure (PASP) was 61.2 mmHg ($4 \times [3.4]^2 + \text{estimated RAP } 15 \text{ mmHg}$), indicating raised pulmonary artery systolic pressure, most likely post-capillary. Calculated systemic vascular resistance (SVR) was 2,140 $\text{dyn}\cdot\text{s}/\text{cm}^5$ (normal: 900–1,400 $\text{dyn}\cdot\text{s}/\text{cm}^5$), indicating raised systemic vascular resistance. Repeat echocardiography on Day 8 showed acute right ventricular dilatation and septal flattening ("D-shaped" left ventricle in systole and diastole). LVEF had collapsed to 32.0%, suggesting acute right heart strain and myocardial dysfunction.⁴¹

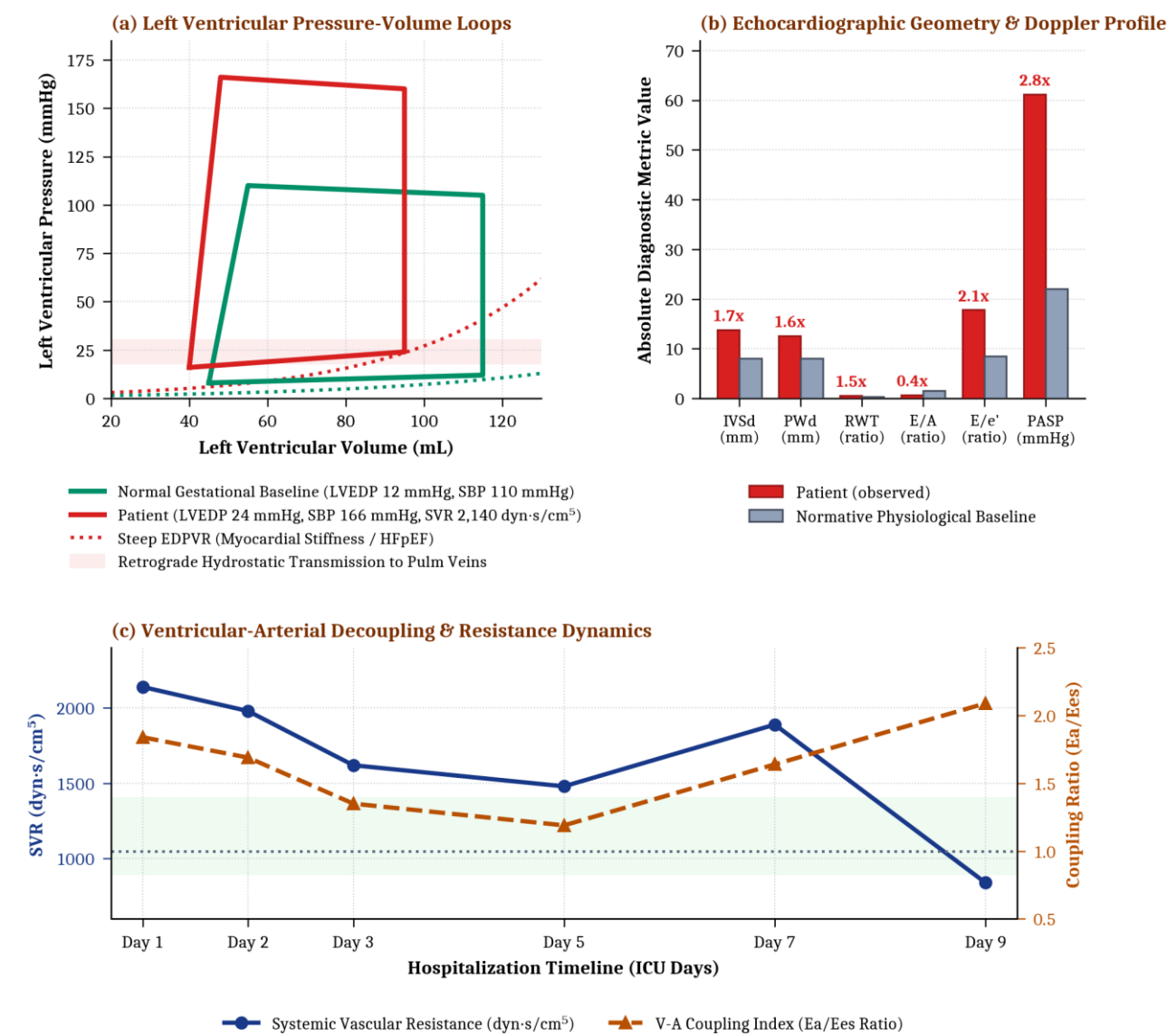


Figure 3. Ventricular-arterial mechanics, diastolic impairment, and vascular resistance. (a) Left ventricular pressure-volume loop reconstruction illustrating concentric remodeling, a steep end-diastolic pressure-volume relationship (EDPVR), and elevated LVEDP (24 mmHg), as in heart failure with preserved ejection fraction (HFpEF), compared with a normotensive gestational baseline. (b) Echocardiographic geometry and Doppler profile (IVSd 13.8 mm, PWd 12.6 mm, RWT 0.54, E/A 0.65, E/e' 17.8, PASP 61.2 mmHg). (c) Ventricular-arterial coupling ratio (Ea/Ees) and systemic vascular resistance (SVR 2,140 dyn-s/cm⁵ on Day 1) across the intensive care course. IVSd, interventricular septal thickness in diastole; PWd, posterior wall thickness in diastole; RWT, relative wall thickness; PASP, pulmonary artery systolic pressure; LVEDP, left ventricular end-diastolic pressure.

3.6. Multimodal imaging timeline

Table 3 sets out the course of radiographic, sonographic, and echocardiographic findings during intensive care. It traces the progression from flash pulmonary edema and

compressive hydrothorax on Day 1, through secondary bacterial superinfection on Day 7, to biventricular myocardial exhaustion on Day 8. The timeline gives an objective anatomical correlate for the patient's physiological decline (Table 3).

Table 3. Imaging timeline: radiography, point-of-care ultrasound, and echocardiography.

Timepoint / Modality	Organ / Compartment Investigated	Key Imaging & Geometric Findings	Severity & Pathophysiological Correlation
Day 1 (Admission) Bedside Chest X-Ray	Thorax (Lungs, Pleura, Mediastinum)	Asymmetric bilateral pleural effusion; massive opacity in left middle/lower zones; perihilar batwing congestion; CTR = 0.62; mild rightward tracheal deviation	Severe flash pulmonary edema, bilateral effusion (Left > Right ~1,250 mL), and compressive left lung atelectasis (Murray Score: 3.25)
Day 1 (Admission) Pelvic Ultrasound	Postpartum Uterus & Pelvis	Enlarged puerperal uterus (14.8 × 9.2 × 8.4 cm); distended cavity with heterogeneous echoes (5.2 × 3.6 cm coagula); collapsed bladder	Subinvolved puerperal uterus with intrauterine clot retention (hematometra) and an empty bladder in keeping with oliguria
Day 1 (Admission) Abdominal FAST USG	Hepatorenal, Splenorenal, Pelvic Pockets	Positive anechoic fluid stripe in Morison's pouch (1.8 cm depth), splenorenal recess (1.2 cm), and pouch of Douglas (~320 mL free fluid)	Multicompartmental third-space ascites secondary to severe endothelial hyperpermeability and hypoalbuminemic oncotic collapse
Day 2 (Post-Intubation) Lung POCUS	Bilateral Hemithoraces (12-Zone Protocol)	Zone 1–4: Confluent B-lines (> 6 per intercostal space) with lung rockets; Zone 5–6: Quad sign and sinusoid sign on left base with massive anechoic space	Sonographic profile confirms combined cardiogenic alveolar flooding (B-profile) and massive exudative-transudative pleural effusions
Day 3 (ICU Day 2) Transthoracic Echo	Cardiac Chambers, Myocardium, Valves	Concentric LV hypertrophy (IVS 13.8 mm, PWd 12.6 mm, RWT 0.54); LVEF = 58.4%; Mitral E/A = 0.65; Septal E/e' = 17.8; TR Vmax = 3.4 m/s (PASP 61.2 mmHg)	Grade III severe diastolic dysfunction with elevated LV filling pressures (PCWP ~24 mmHg), secondary pulmonary hypertension, and preserved LVEF
Day 5 (ICU Day 4) Serial Chest X-Ray	Thorax (Mechanical Ventilation Day 4)	Marginal clearance of right upper zone; persistent dense opacity across left hemithorax; endotracheal tube properly positioned 3.0 cm above carina	Static compressive atelectasis resistant to positive end-expiratory pressure (PEEP 10–12 cmH ₂ O) due to heavy compressive hydrothorax
Day 7 (ICU Day 6) Repeat Chest Radiograph	Thorax (Suspected Secondary Sepsis)	Bilateral diffuse patchy confluent alveolar infiltrates ('white-out' lung); air bronchograms prominent; persistent left basal fluid blunting	Rapid progression from cardiogenic congestion to probable secondary ventilator-associated pneumonia and refractory ARDS (P/F ratio: 112.5 mmHg)
Day 8 (Pre-Terminal) Bedside Echo Re-evaluation	Cardiac Chambers & Vena Cava	Depressed biventricular systolic performance (LVEF dropped to 32.0%); dilated right ventricle with D-shaped septum; IVC dilated 2.6 cm with zero collapse	Suggests acute right heart strain on cardiogenic shock; possible myocardial stunning related to sustained hypoxemia, acidosis, and sepsis

Notes: CTR, cardiothoracic ratio; FAST, focused assessment with sonography for trauma; IVS, interventricular septal thickness; LVEF, left ventricular ejection fraction; PASP, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; PEEP, positive end-expiratory pressure; POCUS, point-of-care ultrasound; PWd, posterior wall thickness in diastole; RWT, relative wall thickness; TR, tricuspid regurgitation.

3.7. Longitudinal trajectories across nine days

Figure 4 tracks the integrated multiorgan trajectory over time. It shows how hemodynamic control, gas exchange, renal excretion, and coagulopathic markers evolved together. Panel A shows nicardipine infusion titration (5.0 to 12.5 mg/h) and the blood pressure response. MAP fell from 118.0 mmHg to 96.7 mmHg by Day 5, before circulatory collapse on Day 9 (Figure 4A). Panel B shows

the PaO₂/FiO₂ ratio with arterial pH. Mechanical ventilation brought temporary stabilization, followed by catastrophic respiratory failure (P/F ratio 54.2 mmHg, pH 7.08; Figure 4B). Panel C shows the progression of KDIGO Stage 3 acute kidney injury, with creatinine rising from 1.84 to 5.20 mg/dL as hourly diuresis fell (Figure 4C). Panel D shows hemoglobin after transfusion, persistent hypoalbuminemia, and a progressive rise in D-dimer to a peak of 22.4 µg/mL FEU (Figure 4D).

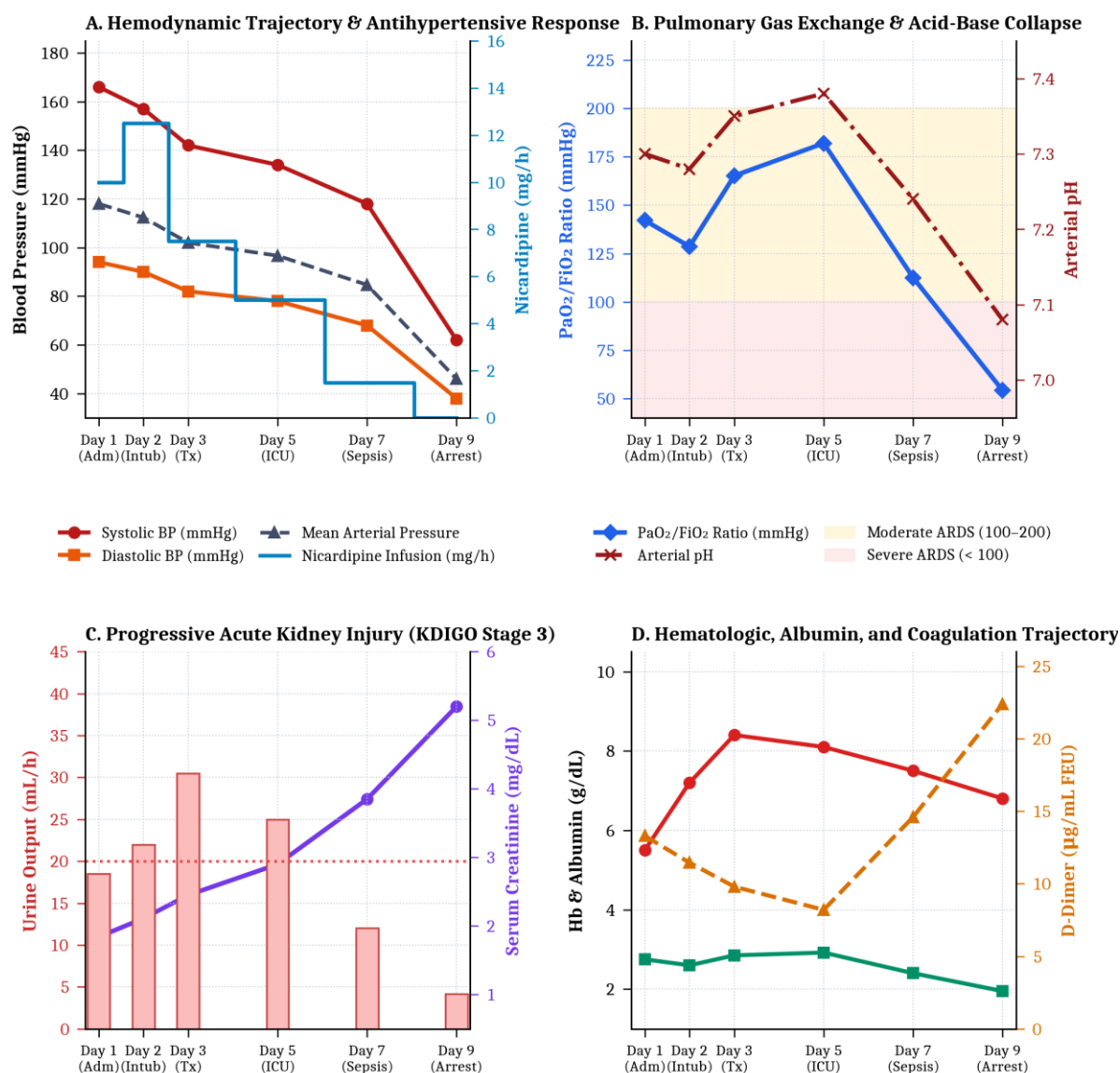


Figure 4. Longitudinal trajectories of multiorgan parameters, gas exchange, and biomarkers across nine days of intensive care. (A) Systolic and diastolic blood pressure with mean arterial pressure (MAP) and intravenous nicardipine infusion (5.0 to 12.5 mg/h). (B) Oxygenation index (PaO₂/FiO₂ ratio) and arterial pH, down to pH 7.08. (C) Progressive acute kidney injury (KDIGO Stage 3): creatinine rising to 5.20 mg/dL with severe oliguria/anuria (< 15 mL/h). (D) Hemoglobin after transfusion, collapsing serum albumin (nadir 1.95 g/dL), and D-dimer rising to 22.4 μg/mL FEU.

3.8. Therapeutic interventions and critical care measures

Table 4 summarizes the treatment given in four clinical phases. Phase 1 (Hours 0–6) comprised airway protection with rapid sequence intubation and a nicardipine infusion for vasodilation. Magnesium sulfate was given for seizure prophylaxis (4.0 g IV bolus then 1.0 g/h maintenance) (Table 4). Phase 2 (Days 2–4) included lung-protective ventilation (tidal volume 6.0 mL/kg PBW, PEEP 10–12 cmH₂O), 3 units of packed red cells, and intravenous tranexamic

acid (1.0 g every 8 hours). It also included oncotic support with 20% human albumin and oral VIP albumin extract, and furosemide diuresis (Table 4). In Phase 3 (Days 5–8), secondary septic shock and ventilator-associated pneumonia were treated with broad-spectrum antimicrobials (moxifloxacin and ceftriaxone, escalated to meropenem). Vasopressor support was norepinephrine (0.05 to 0.45 μg/kg/min) and vasopressin (0.03 units/min). Phase 4 (Day 9) consisted of five cycles of Advanced Cardiovascular Life Support (ACLS) resuscitation for electromechanical dissociation and terminal asystole (Table 4).

Table 4. Therapeutic interventions and critical care measures by phase.

Clinical Phase / Time-line	Intervention Category	Specific Pharmacological / Mechanical Regimen	Target Endpoint & Clinical Response
Phase 1: Emergency Triage (RSSA ED, Hours 0–6)	Oxygenation & Airway	High-Flow Nasal Cannula (HFNC) at 60 L/min, FiO ₂ 0.80, humidified at 37.0°C; escalated to rapid sequence intubation (RSI) with ETT 7.5 mm	Initial SpO ₂ 100%; failed non-invasive support due to refractory tachypnea (RR 38) and deteriorating GCS (13 → 10)
Phase 1: Emergency Triage (RSSA ED, Hours 0–6)	Vasodilator therapy	Nicardipine hydrochloride continuous IV infusion initiated at 5.0 mg/h, titrated up to 12.5 mg/h based on MAP monitoring	Gradual reduction of blood pressure from 166/94 mmHg to 142/82 mmHg within 4 hours; no intracranial hemorrhage was diagnosed
Phase 1: Emergency Triage (RSSA ED, Hours 0–6)	Anticonvulsive Neuroprotection	Magnesium sulfate (MgSO ₄) 4.0 g IV loading dose over 20 min, followed by continuous maintenance infusion of 1.0 g/h for 24 h	Seizure prophylaxis; patellar reflexes and serum magnesium (target 4.8–8.4 mg/dL) monitored
Phase 2: ICU Stabilization (Days 2–4)	Mechanical Ventilation	Volume-controlled lung-protective ventilation: Tidal volume 6.0 mL/kg PBW (360 mL), PEEP 10–12 cmH ₂ O, plateau pressure < 28 cmH ₂ O	Mitigated barotrauma; stabilized PaO ₂ /FiO ₂ between 140–182 mmHg despite heavy left lung compressive atelectasis
Phase 2: ICU Stabilization (Days 2–4)	Hemotherapy & Transfusion	Transfusion of 3 units of leukoreduced Packed Red Cells (PRC, ~750 mL total); IV Tranexamic Acid 1.0 g every 8 hours	Elevated hemoglobin from critical nadir of 5.5 g/dL to 8.4 g/dL; curtailed ongoing occult intrauterine microvascular oozing
Phase 2: ICU Stabilization (Days 2–4)	Oncotic & Diuretic Support	Human Albumin 20% (100 mL IV daily) combined with oral VIP albumin extract (<i>Ophiocephalus striatus</i>); Furosemide infusion (5–10 mg/h)	Raised serum albumin to 2.92 g/dL; augmented diuretic response temporarily (urine output increased to 30.5 mL/h on Day 3)
Phase 3: Septic Shock & Decompensation (Days 5–8)	Empirical Antimicrobial Therapy	Intravenous Moxifloxacin 400 mg once daily plus Ceftriaxone 2.0 g IV every 12 hours; switched to Meropenem 1.0 g q12h on Day 7	Targeted hospital-acquired and aspiration-associated pneumonia; unable to reverse septic surge (WBC spiked to 28,600/mm ³)
Phase 3: Septic Shock & Decompensation (Days 5–8)	Vasoactive Inotropic Support	Norepinephrine infusion titrated from 0.05 to 0.45 µg/kg/min; Vasopressin added at 0.03 units/min as refractory shock ensued	Maintained perfusion pressure temporarily; peripheral vasoconstriction worsened lactic acidosis (lactate surged to 5.8 mmol/L)
Phase 4: Terminal Cardiopulmonary Arrest (Day 9, Hours 184–192)	Advanced Cardiac Life Support	CPR initiated per ACLS protocol (5 cycles of chest compressions, 100% FiO ₂ , IV Epinephrine 1.0 mg every 3–5 min, IV Calcium Chloride)	Refractory asystole / electromechanical dissociation; deceased declared by anesthesiology/critical care team in presence of family

Notes: ACLS, Advanced Cardiovascular Life Support; ETT, endotracheal tube; HFNC, high-flow nasal cannula; ICU, intensive care unit; MAP, mean arterial pressure; PBW, predicted body weight; PEEP, positive end-expiratory pressure; PRC, packed red cells; RSI, rapid sequence intubation. Pharmacotherapy followed the guidelines of the Indonesian Obstetric and Gynecological Society (POGI) and international critical care guidelines.

3.9. Proposed multi-tier pathophysiological and health system cascade

Figure 5 sets out the proposed multi-tier pathophysiological and health system cascade. The mechanisms are inferred from the literature and were not directly measured in this patient. Tier 1 covers defective trophoblast invasion, chronic uteroplacental ischemia, and the anti-angiogenic surge, with sFlt-1 and sEng oversecretion and PlGF suppression (Figure 5). Tier 2 covers systemic endothelial glycocalyx shedding, loss of nitric oxide bioavailability, severe vasoconstriction (SVR 2,140 dyn·s/cm⁵), and hypertensive crisis (166/94 mmHg). Tier 3A covers the

cardiopulmonary consequences: severe left ventricular diastolic failure (E/e' 17.8), raised hydrostatic PCWP (24 mmHg), and capillary hyperpermeability leading to massive asymmetric bilateral pleural effusions (~1,250 mL left) and severe respiratory failure (Figure 5). Tier 3B models the breakdown of cerebral autoregulation, hypoxic encephalopathy, and renal glomeruloendotheliosis leading to KDIGO Stage 3 AKI. Tier 4 captures the health system determinants. These are no BPJS Kesehatan coverage, 0 antenatal care visits, and premature discharge against medical advice because of catastrophic out-of-pocket costs. The result was critical care exhaustion, secondary septic shock, and fatal cardiopulmonary arrest (Figure 5).

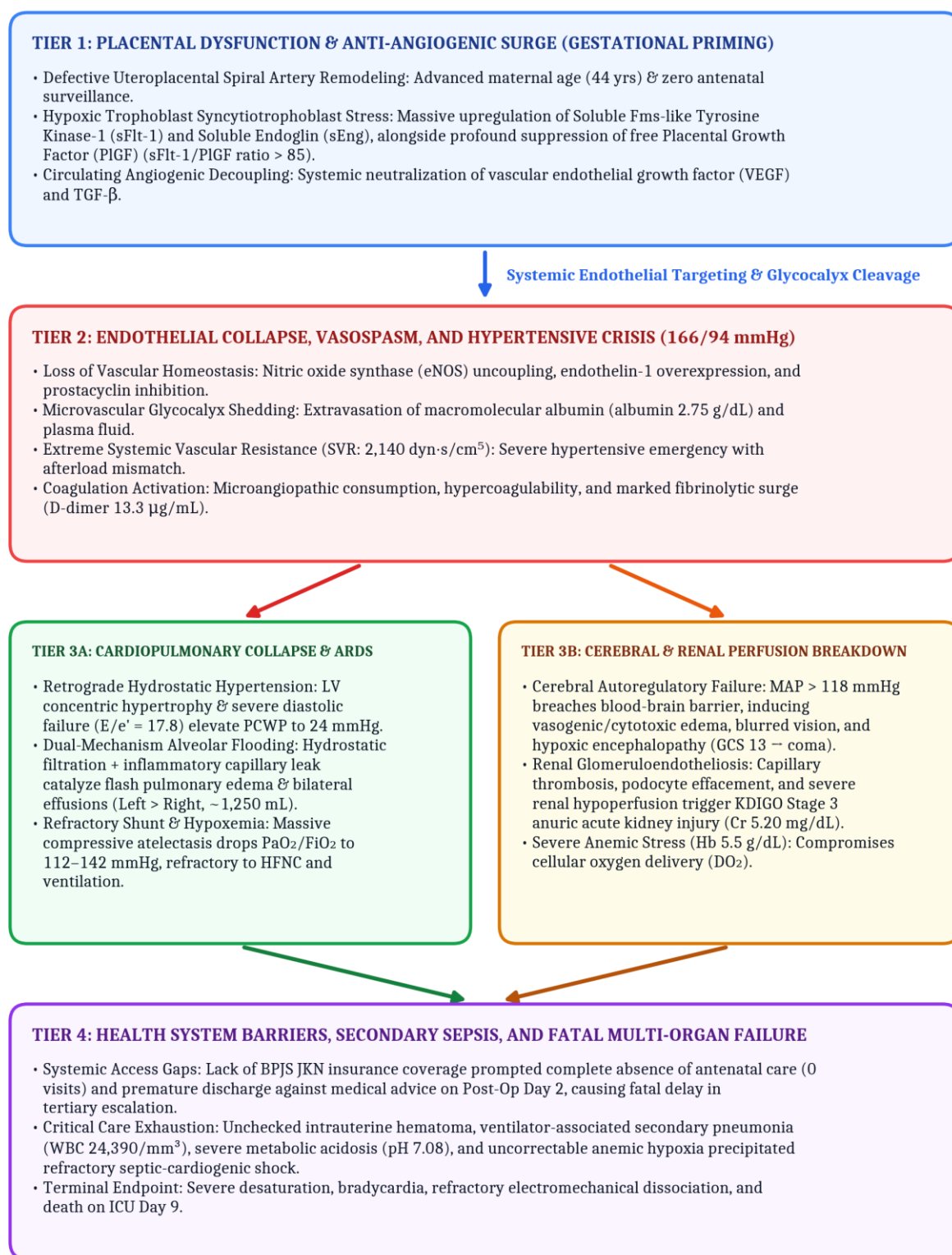


Figure 5. Multi-tier diagram of the proposed pathophysiological and health system cascade in fatal preeclamptic cardiopulmonary collapse. Tier 1: defective spiral artery remodeling, syncytiotrophoblast hypoxia, and anti-angiogenic factors (sFlt-1/PlGF elevation). Tier 2: systemic endothelial cell activation, glycocalyx degradation, and severe vasoconstriction (SVR 2,140 dyn-s/cm⁵). Tier 3A: left ventricular diastolic dysfunction ($E/e' = 17.8$), raised PCWP (24 mmHg), and capillary hyperpermeability with massive asymmetric bilateral pleural effusions (~1,250 mL left) and severe respiratory failure. Tier 3B: blood-brain barrier disruption, hypertensive encephalopathy, and renal glomeruloendotheliosis (KDIGO 3 AKI). Tier 4: health system barriers (lack of BPJS JKN insurance, 0 ANC visits, premature discharge against medical advice due to out-of-pocket costs) leading to diagnostic delay, ventilator-associated pneumonia, septic shock, and fatal cardiopulmonary arrest.

3.10. Comparison with published literature

Table 5 compares the findings of this case with published reports, domain by domain, without causal grading. The patient had no antenatal care, and her blood pressure

rose suddenly to 166/94 mmHg. Unbooked pregnancies are reported to carry a 4.8- to 8.2-fold higher risk of severe maternal outcomes in comparable low- and middle-income settings⁸ (Table 5). Flash pulmonary edema and respiratory failure are repeatedly reported as complications of

neglected severe preeclampsia in international registries.³⁷ The organ findings were oliguria with creatinine of 5.20 mg/dL, hypoalbuminemia (2.75 g/dL), and a D-dimer of 13.3 µg/mL, features described in preeclamptic microangiopathy.^{17,19} The sequence was absent antenatal care, unmanaged preeclampsia that needed emergency surgery, discharge against medical advice on postoperative day 2, and an interval out of hospital in which cardiopulmonary and neurological decompensation developed (Table 5).

Table 5. Findings of this case compared with published literature

Domain	Evaluated component	This case	Published benchmark	Interpretation (descriptive)
Antenatal care and risk	Zero ANC and uncontrolled gestational hypertension	Complete absence of antenatal care (0 visits) culminated in BP 166/94 mmHg, flash edema, and death	Unbooked pregnancies exhibit 4.8-fold to 8.2-fold increased risk of severe maternal mortality ⁸	Consistent with reports that unmonitored hypertension is associated with severe maternal outcomes; causality cannot be judged from one patient
Clinical phenotype	Cardiopulmonary collapse in severe preeclampsia	Bilateral effusion, ARDS, and concentric LV diastolic dysfunction documented on chest X-ray and echo	Reported across near-miss cohorts: flash pulmonary edema occurs in 2.9–5.8% of severe PE ³⁷	The phenotype resembles that reported in other settings
Organ involvement	Organ damage directly attributable to preeclampsia	Suspected glomeruloendotheliosis (oliguria, Cr 5.2 mg/dL), severe hypoalbuminemia (2.75 g/dL), and D-dimer surge (13.3 µg/mL)	Characteristic preeclamptic microangiopathy and capillary leak triad ^{17,19}	The organ findings fit the described preeclamptic pattern; the mechanisms were not measured
Sequence of events	Progression sequence from delivery to fatal collapse	Uncontrolled pregnancy → Emergency CS → Premature discharge D2 → Home deterioration → RSSA D3 → MODS D9	Inadequate postpartum surveillance is the leading temporal predictor of late maternal near-miss ⁵³	Sequence in this patient: no antenatal care, emergency surgery, discharge on day 2, decompensation; the contribution of discharge cannot be quantified
Severity and organ dysfunction	Severity of blood pressure and biomarker elevation	MAP 118 mmHg, Hb 5.5 g/dL, D-dimer 13.3 µg/mL directly correlated with GCS 13 and P/F ratio 142	Relationship reported between sFlt-1/PlGF ratio, MAP, and multi-organ morbidity ^{32,54}	Higher afterload and greater fluid loss coincided with worse organ function in this patient
Fluid mechanics of the effusion	Dual hydrostatic-permeability mechanism of edema	Elevated PCWP (24 mmHg) + capillary hyperpermeability + hypoalbuminemia (2.75 g/dL) drove 1,250 mL effusion	Starling equation breakdown in preeclampsia: high capillary pressure plus low oncotic pressure ^{37,52}	A combined hydrostatic and permeability mechanism is a plausible explanation for the effusion and atelectasis
Local epidemiology	Concordance with clinical trials and registry data	Patient's fatal outcome aligns with Indonesian JKN maternal mortality trends in referral centers	Persisting maternal mortality in severe PE despite JKN expansion has been documented ^{3,6}	The outcome is in line with reports of persisting maternal deaths despite JKN expansion
Respiratory failure	Frequency of acute respiratory failure / ARDS	Severe hypoxemia requiring mechanical ventilation and PEEP (P/F ratio nadir: 54.2 mmHg)	ARDS complicates 1.5–3.2% of ICU-admitted severe preeclampsia cases, with mortality ranging from 28% to 45% ³⁵	The respiratory course was at the severe end of the reported range
Neurological involvement	Hypoxic encephalopathy and altered sensorium	Presenting GCS 13 with blurred vision, progressing to obtundation and irreversible coma	Posterior Reversible Encephalopathy Syndrome (PRES) occurs in up to 90% of preeclamptic encephalopathies ⁵⁵	Altered cerebral autoregulation during blood pressure surges is a proposed mechanism; no neuroimaging is available
Health insurance and care-seeking	Impact of health insurance on maternal survival	Patient lacked BPJS coverage, paid out-of-pocket, and requested discharge against medical advice	Uninsured mothers in Indonesia have 2.3-fold higher odds of incomplete ANC and delayed emergency care ^{1,10}	Financial barriers are a likely contributor to delayed and discontinued care in this case

Notes: ANC, antenatal care; ARDS, acute respiratory distress syndrome; CS, cesarean section; GCS, Glasgow Coma Scale; JKN, Jaminan Kesehatan Nasional; MAP, mean arterial pressure; MODS, multi-organ dysfunction syndrome; PCWP, pulmonary capillary wedge pressure; PE, preeclampsia; P/F, PaO₂/FiO₂ ratio; SMM, severe maternal morbidity. Benchmark values are quoted as printed in the submission

4. Discussion

4.1. Principal findings

This case report describes the clinical and hemodynamic course of neglected severe preeclampsia that ended in fatal cardiopulmonary collapse, massive bilateral pleural effusion, and hypoxic encephalopathy. We followed the patient through nine days of tertiary intensive care. Her death was probably multifactorial. A multi-tier pathophysiological cascade is proposed. The cascade began with early placental malperfusion and was accelerated by health system and socioeconomic failures (Figure 5).^{18,23}

4.2. Biological plausibility and the proposed multi-tier cascade

The first event in the biological hierarchy (Tier 1) is defective trophoblast invasion. Remodeling of the maternal spiral arteries is impaired in early gestation.^{21,22} In normal pregnancy, extravillous trophoblasts replace the muscular and elastic media of the spiral arterioles. The vessels become dilated, tortuous, and low in resistance. They can

Higher MAP (118 mmHg), extreme SVR (2,140 dyn·s/cm⁵), and high left ventricular filling pressure (E/e' 17.8) coincided with greater pleural fluid loss (~1,250 mL) and hypoxemia (PaO₂/FiO₂ 142.1 to 54.2 mmHg). This describes one patient and is not an estimate of dose-response. Starling mechanics and the anti-angiogenic sFlt-1/PlGF paradigm offer a plausible explanation for these findings (Table 5).^{32,52}

carry a 10-fold rise in uteroplacental blood flow without vasoconstrictor responsiveness.²² In our patient, advanced maternal age (44 years) and primiparity together raised the risk of defective endovascular invasion.^{16,56} The persistent high-resistance placental circulation is thought to produce repeated ischemia-reperfusion cycles and syncytiotrophoblast oxidative stress. Heavy secretion of anti-angiogenic mediators into the maternal circulation followed.^{23,24} The leading mediators are soluble fms-like tyrosine kinase-1 (sFlt-1), a splice variant of the VEGF receptor-1 that lacks the transmembrane and cytoplasmic domains, and soluble endoglin (sEng), a TGF-β coreceptor.^{24,25} Circulating sFlt-1 acts as a molecular trap. It binds free vascular endothelial growth factor (VEGF) and placental growth factor (PlGF) with high affinity and removes them from circulation.^{24,54} Normally, VEGF and PlGF signaling maintains endothelial fenestrations, basal nitric oxide synthase (eNOS) activity, and vascular barrier integrity in every microvascular bed. When it is lost, endothelial cells undergo apoptosis and detachment. Systemic vasoconstriction follows (Tier 2).^{18,54}

4.3. Endothelial glycocalyx breakdown and capillary leak mechanics

A key finding in our patient was the rapid development of fluid sequestration in several compartments. There were large bilateral pleural effusions (mainly left-sided, ~1,250 mL), ascites (Morison's pouch 1.8 cm, pouch of Douglas 320 mL), and diffuse anasarca. Hypoalbuminemia accompanied them, with albumin 2.75 g/dL on admission falling to 1.95 g/dL (Table 1, Table 2, and Figure 2). This fluid loss reflects breakdown of the endothelial glycocalyx and vascular hyperpermeability.^{54,57} The glycocalyx is a negatively charged, carbohydrate-rich meshwork of proteoglycans (syndecans, glypicans) and glycosaminoglycans (heparan sulfate, chondroitin sulfate, hyaluronic acid) on the luminal surface of healthy endothelium.⁵⁷ When intact, it is the main semipermeable barrier to macromolecules, keeping plasma proteins such as albumin in the vessel and maintaining the transvascular colloid oncotic gradient described by the revised Starling principle.⁵²

In severe preeclampsia, systemic inflammation, circulating anti-angiogenic factors, and high mechanical shear stress increase endothelial-bound matrix metalloproteinases, heparanases, and hyaluronidases. These enzymes cleave the glycocalyx.^{54,57} The loss of this barrier exposes the endothelial intercellular junctions (vascular endothelial-cadherin) to inflammatory disruption. Albumin then leaves the intravascular compartment quickly for the interstitial and serosal spaces.^{52,57} The resulting hypoalbuminemia lowers plasma oncotic pressure from a normal ~22–25 mmHg to < 14–16 mmHg.⁵² Oncotic pressure collapses. Fluid moves into low-resistance serosal cavities (pleura, peritoneum) at an accelerating rate, large exudative-transudative effusions form, and the adjacent lung is compressed (Table 3).^{37,58}

4.4. Hydrostatic overload, diastolic dysfunction, and flash pulmonary edema

On top of this increased permeability came marked hydrostatic afterload mismatch (Tier 3A). It turned localized pulmonary congestion into flash pulmonary edema and acute respiratory distress syndrome (Figure 3 and Figure 4).^{35,37} Transthoracic echocardiography showed concentric left ventricular hypertrophy (IVS 13.8 mm, PWd 12.6 mm, RWT 0.54) and a high systemic vascular resistance (SVR 2,140 dyn·s/cm⁵), together with severe grade III diastolic dysfunction (mitral E/A 0.65, septal E/e' 17.8) despite a preserved ejection fraction (LVEF 58.4%) (Table 3 and Figure 3). This is the classic profile of heart failure with preserved ejection fraction (HFpEF). Acute hypertensive crisis in pregnancy can trigger it.^{40,59}

Extreme systemic vasoconstriction, driven by endothelin-1 overexpression, eNOS uncoupling, and loss of prostacyclin synthesis, forces the left ventricle to eject against very high resistance.^{45,54} Under sustained, unmonitored gestational hypertension the maternal myocardium remodels concentrically. It becomes stiffer, with impaired active relaxation in diastole.^{40,59} When blood pressure rises acutely in a hypertensive crisis (MAP 118.0 mmHg), left ventricular end-diastolic pressure climbs steeply. The pressure is transmitted backward into the left atrium (dilated LAVI 44 mL/m²) and the pulmonary veins.^{40,59} The E/e' ratio of 17.8 corresponds to a pulmonary capillary wedge pressure (PCWP) of about 24 mmHg, well above the threshold for hydrostatic pulmonary edema (~18 mmHg) (Table 3).^{37,60} Now consider a hydrostatic capillary pressure of 24 mmHg acting on a damaged, highly permeable alveolar-capillary membrane, with oncotic collapse (albumin 2.75 g/dL). The alveoli flood very quickly.^{37,52}

The asymmetry of the pleural effusion also deserves comment (Figure 1). Fluid occupied two-thirds of the left hemithorax (~1,250 mL) and caused complete compressive atelectasis of the left lung. Preeclamptic effusions are often bilateral, but asymmetry can arise from differences in regional lymphatic drainage, gravitational positioning during prolonged bedrest, or localized inflammatory pleuritis.^{37,58} In our patient the large left hydrothorax acted as a rigid space-occupying lesion. It shifted the mediastinum to the right (4 mm). It also compressed the left pulmonary vascular bed. The compressive atelectasis created a large intrapulmonary right-to-left shunt, because mixed venous blood passed through unventilated left pulmonary capillaries. This probably contributed to her severe, refractory hypoxemia (PaO₂/FiO₂ 142.1 mmHg at presentation, 54.2 mmHg shortly before death) despite high-flow oxygen and ventilation with high PEEP (10–12 cmH₂O) (Table 2 and Figure 4).³⁵

4.5. Cerebral autoregulatory failure and hypoxic encephalopathy

The neurological presentation was an initial GCS of 13, severe visual blurring, and rapid progression to obtundation and coma. It exemplifies the neurological sequelae of preeclamptic hypertensive encephalopathy (Tier 3B) (Table 1 and Figure 5).^{53,55} In healthy adults, cerebral blood flow stays near 50 mL/100 g/min across a mean arterial pressure of 60 to 140 mmHg. Myogenic vasoconstriction of precapillary arterioles maintains it (the autoregulatory curve).⁶¹ Pregnancy changes this. The autoregulatory plateau shifts to the left, and the upper limit falls to a MAP of about 115–120 mmHg.⁶¹

When the patient's MAP reached 118.0 mmHg (systolic 166 mmHg), the upper limit of cerebral autoregulation may have been exceeded (Table 1).⁶¹ Forced arteriolar dilatation followed and exposed fragile downstream capillaries to high hydrostatic pressure and shear stress.^{53,55} At the same time, circulating sFlt-1 and pro-inflammatory cytokines disrupted blood-brain barrier tight junctions (claudin-5, occludin), allowing serum proteins and fluid into the cerebral interstitium.⁵⁵ The result is vasogenic and cytotoxic cerebral edema, mainly in the parieto-occipital white matter, which has sparse sympathetic perivascular innervation. This entity is called Posterior Reversible Encephalopathy Syndrome (PRES).^{53,55} The patient's visual blurring (scotomata) may have reflected occipital or retinal involvement; no neuroimaging or ophthalmologic examination is reported.^{53,55} Over the following days, persistent tissue dysoxia, severe anemic hypoxia (Hb nadir 5.5 g/dL), and repeated shock converted reversible vasogenic edema to irreversible hypoxic-ischemic neuronal necrosis. The end point may have been hypoxic-ischemic encephalopathy (Table 2 and Table 4).⁶²

4.6. Severe anemic hypoxia, renal glomeruloendotheliosis, and multi-organ collapse

Severe normocytic anemia (hemoglobin 5.5 g/dL on admission) was an underappreciated contributor to the terminal decline (Table 2). Severe maternal anemia greatly impairs systemic oxygen delivery (DO₂), defined by:

$$DO_2 = CO \times (Hb \times 1.34 \times SaO_2 + [0.003 \times PaO_2])$$

At a hemoglobin of 5.5 g/dL, arterial oxygen-carrying capacity was roughly halved. The hypertrophied left ventricle was already under strain. It had to raise cardiac output to meet baseline metabolic demand (Table 1 and Table 2).^{42,63} Severe hypoxemia from ARDS and atelectasis was then added to this anemia. Oxygen delivery to the myocardium, brain, and kidneys fell at the same time (Figure 4).⁶³

The pathological hallmark of severe preeclampsia in the kidney is glomeruloendotheliosis. Glomerular endothelial cells swell, endothelial fenestrae are lost, and capillary lumina are occluded by swollen cytoplasm and subendothelial fibrinoid deposits.^{18,64} Circulating sFlt-1 neutralizes podocyte vascular endothelial growth factor (VEGF) signaling, which leads to podocyte foot process effacement and slit diaphragm disruption and drives heavy proteinuria.^{24,64} In our patient, glomeruloendotheliosis and acute ischemic tubular necrosis (ATN) are possible contributors; no biopsy or urinary sediment is reported. Its causes were prolonged out-of-hospital hypovolemia, third-space fluid loss, and systemic vasoconstriction (Table 2 and Table 5).³¹ The double insult produced rapid, anuric acute kidney injury (KDIGO Stage 3). Creatinine rose steadily from 1.84 mg/dL to 5.20 mg/dL, and BUN reached 96.5 mg/dL (Figure 4C). Failing kidneys could no longer excrete free water and sodium. Intravascular volume overload, refractory pulmonary edema, and terminal metabolic acidosis worsened (lactate 11.4 mmol/L, base excess -19.4 mEq/L) (Table 2 and Table 4).^{31,34}

4.7. Health system governance, out-of-pocket expenditure, and premature discharge

The microvascular and hemodynamic mechanisms above explain the physiological collapse. This case cannot, however, be separated from the health system determinants and socioeconomic barriers that governed the patient's course (Tier 4) (Figure 5).^{1,2} She had no antenatal care contact during the entire pregnancy. She was also uninsured. She falls within the estimated 88 million Indonesians who currently lack active BPJS Kesehatan / JKN coverage (Table 1).^{1,6}

Antenatal care is the proven first line of defense against maternal death. Routine first- and second-trimester contacts allow baseline blood pressure measurement and the identification of high-risk women from history and uterine artery Doppler screening. They also allow the start of low-dose aspirin prophylaxis (150 mg daily before 16 weeks), which lowers the incidence of preterm preeclampsia by up to 62%.^{13,65} This patient skipped antenatal care entirely. Her chronic hypertension stayed unmonitored and untreated. Placental ischemia and angiogenic imbalance progressed until a fulminant hypertensive crisis forced emergency delivery (Table 5).^{10,29}

The turning point in this patient's course was her premature discharge against medical advice (pulang paksa) on postoperative day 2 from the secondary private hospital (Table 1 and Table 4).^{6,8} In Indonesia, an uninsured patient who has an emergency cesarean section at a private facility faces a medical bill that becomes a catastrophic out-of-pocket liability.^{1,2} Fear of mounting charges often leads families to request discharge against medical advice, putting short-term financial survival ahead of clinical safety.^{6,8} In this case, going home with early dyspnea and blurred vision introduced a 48-hour delay in diagnosis and treatment (Table 1).^{8,12} During that unmonitored interval, untreated hypertensive emergency, developing flash pulmonary edema, and third-space fluid sequestration passed the point of reversibility. A potentially salvageable postpartum complication may have become an irreversible multi-organ crisis (Table 5).^{7,8}

4.8. Clinical and research implications: integrating private obstetric facilities into universal health coverage

This case points to urgent policy needs for maternal health governance in Indonesia. The same applies to other low- and middle-income countries that run universal

health insurance schemes.^{1,2} BPJS Kesehatan covers maternal care comprehensively at public community health centers (Puskesmas) and contracted public hospitals. Private obstetric clinics and private hospitals often operate outside the national reimbursement network. Some also impose complex administrative referral hurdles (Table 5).^{1,6} In urban and peri-urban Indonesia, many obstetric emergencies present first to private facilities. They are close, and they are perceived to offer fast emergency access.^{5,66}

To end the practice of financial discharge against medical advice, national health insurance frameworks should create mandatory mechanisms for emergency integration (Table 4).^{1,8} Under such a policy, any uninsured pregnant woman in hypertensive emergency would receive immediate emergency JKN enrollment at a private facility. The same would apply if she needed emergency surgical delivery. Enrollment would follow an expedited bedside social welfare verification, with full reimbursement guaranteed to the treating facility.^{2,8} Discharge criteria after delivery in severe preeclampsia should also be formalized in law. They should prohibit discharge against medical advice in mothers with unresolved tachypnea, severe hypertension, or neurological symptoms. The exception is when emergency escalation and inter-facility ambulance transfer to a tertiary academic medical center are arranged (Table 4 and Table 5).^{5,6} One more step is simple. Point-of-care lung ultrasound has been validated in recent critical care trials, and adding it to routine obstetric triage would help detect subclinical pulmonary edema before discharge is considered.³⁸

4.9. Strengths and limitations

The main strength of this report is its serial documentation. It gives serial quantification of hemodynamic parameters (SVR, PCWP, E/e'), gas exchange (P/F ratio, base excess, lactate), laboratory biomarkers (D-dimer, albumin, creatinine), and multimodal imaging (radiography, POCUS, echocardiography) across nine consecutive days of intensive care (Table 2, Table 3, and Table 4).^{34,39,40,51} There are two limitations. Baseline serum sFlt-1 and PlGF concentrations are lacking, because these assays are not routinely reimbursed in emergency tertiary settings in Indonesia. No autopsy was done. The family declined for socio-cultural and religious reasons. Further limitations are the single patient, the absence of neuroimaging and of tissue diagnosis, and the lack of a comparison group, so that the mechanisms described above remain proposals. Even so, the clinical, radiographic, echocardiographic, and laboratory findings allowed objective diagnostic ascertainment (Tables 1 to 5 and Figures 1 to 5).

5. Conclusion

This case report describes the clinical and hemodynamic course of neglected severe preeclampsia in a 44-year-old uninsured woman. She died. Death followed from the convergence of endothelial glycocalyx degradation, capillary leak syndrome, and severe hydrostatic afterload mismatch, which produced massive asymmetric bilateral pleural effusions, compressive atelectasis, severe ARDS, and hypoxic encephalopathy. Two factors accelerated the biological crisis. One was the complete absence of antenatal care. The other was premature discharge against medical advice, driven by catastrophic out-of-pocket medical costs.

These findings have clinical and health policy implications. For clinicians, the lesson is clear. Obstetric critical care teams should recognize that postpartum severe preeclampsia can cause sudden flash pulmonary edema with preserved left ventricular ejection fraction, and that

severe diastolic failure with high systemic vascular resistance explains it. Management calls for firm blood pressure control with intravenous vasodilators (nicardipine), cautious fluid restriction, oncotic support, and early point-of-care lung and cardiac ultrasound. For public health and governance, closing the coverage gap of the 88 million uninsured citizens under Indonesia's BPJS Kesehatan should be a priority. Universal health coverage frameworks should legally mandate emergency obstetric coverage at all private facilities. They should provide bedside rapid insurance enrollment. They should also enforce strict clinical non-discharge protocols for postpartum preeclamptic mothers. These measures may help reduce avoidable maternal morbidity and mortality.

Declarations

Declarations

Use of Generative Artificial Intelligence. The author did not use any generative artificial-intelligence tools in writing, analyzing, or preparing this manuscript. All content was produced and verified by the author, who takes full responsibility for its integrity and accuracy.

Author Contributions. Y.A.E.: conceptualization, methodology, investigation, data curation, formal analysis, writing – original draft, writing – review & editing. The author read and approved the final manuscript.

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Conflict of Interest. The author declares no competing financial, professional, or personal interests that could have influenced the design, execution, or interpretation of this work.

Ethical Approval. The study was approved by CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Number: 2026/0852), and was conducted in accordance with the Declaration of Helsinki.

Patient Consent. Written informed consent was obtained from the patient's legal representative and next of kin (husband) for the publication of this case report, including all accompanying clinical data, laboratory trajectories, and de-identified radiographic images.

Data Availability. All data generated or analyzed during this study are included in this published article and its accompanying longitudinal serial tables (Tables 1 to 5). De-identified primary laboratory datasets are available from the corresponding author upon reasonable academic request.

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