Sriwijaya Journal of Obstetrics and Gynecology
Vol. 4, No. 2, 2026, pp. 72-86 · e-ISSN 2986-9838 *journal homepage: phlox.or.id/index.php/sjog*

Case Report

Epidural Ropivacaine, Sildenafil–Beraprost Vasodilation, and Invasive Monitoring for Emergency Cesarean Delivery in an Adolescent with Atrial Septal Defect–Induced Eisenmenger Syndrome: An Observational Analytic Case Study

Fajar Tri Mudianto[1*], Isngadi[1]

[1]*Department of Anesthesiology and Intensive Therapy, Faculty of Medicine, Universitas Brawijaya/Dr. Saiful Anwar General Hospital, Malang, Indonesia*

ARTICLE INFO

Article history:
Received: August 15, 2026
Revised: September 7, 2026
Accepted: October 3, 2026
Available Online: October 18, 2026
Published: October 30, 2026

Keywords:
Anesthesia, epidural
Atrial septal defect
Cesarean section
Eisenmenger complex
Hypertension, pulmonary

***Corresponding author:**
Fajar Tri Mudianto
E-mail: dian.fjr@gmail.com
DOI: 10.59345/sjog.v4i2.381

ABSTRACT

Background: Gravid Eisenmenger syndrome carries 30–60% maternal mortality from falling systemic vascular resistance (SVR), right ventricular (RV) failure, and shunt reversal.

Objective: To describe the cardiopulmonary course, dual oral vasodilation, and graded epidural anesthesia of an adolescent gravida with secundum atrial septal defect (ASD)–induced Eisenmenger syndrome who underwent emergency cesarean section.

Methods: This case report follows an 18-year-old woman (46.0 kg, 152 cm, BMI 19.91 kg/m^2; G2P0010Ab0010 at 34–36 weeks; ASA 5E, mWHO Class IV) across five perioperative milestones (T_0–T_4). Relative deviations (Δ%), oxygenation ratios, fluid balance, and mWHO, ASA, and CARPREG II risk scores were calculated.

Results: Echocardiography showed a large secundum ASD with balanced shunt, severe tricuspid and moderate pulmonary regurgitation, reduced RV contractility, and a left ventricular ejection fraction of 51%. She had hypoxemia (SpO_2 73% on a 15 L/min non-rebreathing mask; SpO_2/FiO_2 81.11), lung edema, hypoalbuminemia (3.10 g/dL, Δ% −11.43%), and prerenal azotemia (urea 62.3 mg/dL, Δ% +55.75%; creatinine 0.94 mg/dL). She received oral sildenafil (3 × 20 mg/day), oral beraprost (3 × 20 µg/day), and intravenous furosemide (3 × 20 mg/day; target balance −500 mL/24 h). Under arterial and central venous monitoring with milrinone and phenylephrine on standby, graded 0.75% ropivacaine epidural anesthesia was followed by a blood pressure of 104/60 mmHg (MAP 74.67 mmHg). A live infant (Apgar 7 and 8) was delivered; mother and infant survived.

Conclusion: In this patient, sildenafil–beraprost vasodilation, diuresis, invasive monitoring, and graded epidural anesthesia were followed by delivery without shunt crisis. A single case cannot establish causation.

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

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1. Introduction

Eisenmenger syndrome is the most advanced and irreversible form of pulmonary arterial hypertension associated with congenital heart disease (PAH-CHD), and it arises when an uncorrected communication between the systemic and pulmonary circulations causes progressive obliterative remodeling of the pulmonary vessels [1,2]. Early in its course, a large left-to-right intracardiac or aortopulmonary shunt exposes the pulmonary microcirculation to chronic volume and shear-stress overload. The endothelium becomes dysfunctional. The balance of vasoactive mediators shifts, and endogenous nitric oxide (NO) and prostacyclin (PGI_2) fall while endothelin-1 (ET-1) rises. Smooth muscle hypertrophy, intimal fibrosis, plexiform arteriopathy, and in situ microvascular thrombosis follow [1,2]. As pulmonary vascular resistance (PVR) approaches or exceeds systemic vascular resistance (SVR), the left-to-right shunt becomes a balanced bidirectional shunt and finally reverses to right-to-left. Deoxygenated systemic venous blood then enters the systemic arterial circulation directly. The

consequences are refractory central hypoxemia, secondary digital clubbing, progressive right ventricular (RV) systolic failure, life-threatening arrhythmias, and premature death [1,3].

When Eisenmenger syndrome coincides with pregnancy, the physiological adaptations that support fetal growth collide with a fixed, non-distensible pulmonary vascular bed. The mother falls into modified World Health Organization (mWHO) cardiovascular risk Class IV. Pregnancy is formally contraindicated there, because maternal mortality has historically ranged between 30% and 60% [4–7]. A tertiary Indonesian cohort of 18 pregnant patients with Eisenmenger syndrome recorded a maternal mortality of 38.9% (7 of 18 cases). Deaths were driven mainly by refractory postpartum cardiogenic shock, severe pulmonary hypertensive crisis, and thromboembolic decompensation in the first two weeks after delivery [8]. Large multicenter registries and contemporary single-center series report the same pattern [9]. Pregnant women with uncorrected shunt-related PAH do worse than those with idiopathic PAH or repaired congenital heart disease. They have much higher rates of acute right heart failure, severe hypoxemia, intrauterine growth restriction, preterm delivery, and fetal loss [10–12]. In normal gestation, maternal plasma volume expands by 40% to 50%, and progesterone-, estrogen-, and relaxin-mediated vasodilation lowers SVR by 20% to 30% by the late second and third trimesters [13,14]. In a patient with a large secundum atrial septal defect (ASD) and balanced Eisenmenger physiology, this fall in SVR narrows or inverts the systemic-to-pulmonary pressure gradient (SVR/PVR ratio). Right-to-left shunting accelerates, arterial desaturation worsens, uteroplacental oxygen delivery falls, and the right ventricle faces acute-on-chronic volume and pressure overload [14,15].

Ventricular interdependence within the constraining pericardial sac adds to the vulnerability of the gravid Eisenmenger heart [16]. Severe pulmonary hypertension and torrential tricuspid regurgitation (TR) dilate the thin-walled right ventricle and impair its systolic contractility. Right ventricular end-diastolic pressure then rises. It pushes the interventricular septum leftward during diastole and produces a D-shaped left ventricular cavity. This restricts left ventricular diastolic filling and reduces preload reserve, and it can cause presyncopal cerebral hypoperfusion even when the left ventricular ejection fraction (LVEF) is preserved [16]. At the same time, chronic elevation of central venous and right atrial pressures transmits backward into the pulmonary lymphatic drainage and the systemic venous beds. The results are alveolar-interstitial pulmonary edema, hepatic sinusoidal stasis, mild hypoalbuminemia, and prerenal cardiorenal venous congestion [17,18].

Emergency cesarean section is therefore among the most formidable challenges in obstetric anesthesiology and maternal-fetal intensive care. This is especially so when the parturient has decompensated Eisenmenger syndrome, acute cardiogenic pulmonary edema, severe hypoxemic respiratory failure, and fetal compromise [19–21]. The hemodynamic aim during delivery has four parts. SVR must be preserved. Any surge in PVR must be avoided, whether it comes from hypoxia, hypercarbia, acidosis, hypothermia, pain-induced catecholamine release, or positive-pressure ventilation. RV preload must stay euvolemic, and RV myocardial contractility needs protection [22,23]. Both conventional general endotracheal anesthesia and single-shot subarachnoid (spinal) block carry lethal hazards in this population. A single-shot spinal causes an abrupt, non-titratable sympathetic blockade that collapses SVR and increases right-to-left shunting. General anesthesia brings myocardial depression from induction agents, vasodilation from volatile agents, rapid apneic desaturation, atelectasis-related shunt aggravation, and a rise in intrathoracic pressure and PVR from positive-pressure ventilation [21,24].

Despite broad international agreement on multidisciplinary care, clinical uncertainty remains. Three measures are at issue. The first is preoperative combined oral pulmonary vasodilator therapy, with a phosphodiesterase type 5 (PDE-5) inhibitor (sildenafil) plus an oral prostacyclin analogue (beraprost sodium). The second is loop diuretic preload unloading (furosemide). The third is sole graded lumbar epidural anesthesia with 0.75% ropivacaine under real-time invasive arterial blood pressure (ABP) and central venous catheter (CVC) monitoring [25]. Parenteral prostanoids and inhaled nitric oxide are often advocated in high-income tertiary centers [26,27]. Resource-conscious referral hospitals in Southeast Asia frequently have to stabilize patients quickly in other ways. They use oral dual-pathway pulmonary vasodilators. These target both the NO–soluble guanylate cyclase–cyclic guanosine monophosphate (cGMP) axis and the prostacyclin IP receptor–cyclic adenosine monophosphate (cAMP) axis. They add targeted negative fluid balance protocols and titrated amide local anesthetic epidural blockade backed by standby milrinone (an inodilator) and phenylephrine (a pure α_1-adrenergic vasopressor) [28].

This report gives a detailed quantitative description of the perioperative cardiopulmonary, oncotic-renal, fluid-balance, and anesthetic course of an 18-year-old adolescent gravida. She had late-diagnosed secundum ASD–induced Eisenmenger syndrome, severe tricuspid regurgitation, reduced RV contractility, cardiogenic pulmonary edema, and severe hypoxemia (SpO_2 73% on a 15 L/min non-rebreathing mask; SpO_2/FiO_2 ratio 81.11). She underwent emergency lower-segment transperitoneal cesarean section under sole graded 0.75% ropivacaine epidural anesthesia. The aim of this case report is to describe the hemodynamic course, the biomarker deviations, and the maternal and neonatal outcome under integrated combined oral pulmonary vasodilation (sildenafil and beraprost), loop diuretic negative fluid balance (−500 mL/24 h), invasive ABP/CVC monitoring, and graded 0.75% ropivacaine epidural anesthesia at a tertiary Indonesian academic referral hospital.

2. Methods

2.1. Study design and clinical setting

This is a case report of the perioperative cardiopulmonary, laboratory, radiographic, echocardiographic, and anesthetic course of an adolescent gravida with decompensated Eisenmenger syndrome who underwent emergency cesarean section. It took place in January 2025 at the Department of Anesthesiology and Intensive Therapy and in the Maternal-Fetal High-Risk Obstetric Operating Theater and Intensive Care Unit of RS Dr. Saiful Anwar (Dr. Saiful Anwar Regional General Hospital), an academic tertiary referral center affiliated with the Faculty of Medicine, Universitas Brawijaya, Malang, East Java, Indonesia. The report follows the CARE guideline for case reports. Data were recorded across five perioperative milestones. T_0 (2–3 January 2025) was admission triage and the diagnostic baseline. T_1 covered preoperative pulmonary vasodilator and diuretic therapy and invasive vascular optimization. T_2 was intraoperative sole graded 0.75% ropivacaine epidural anesthesia and emergency lower-segment transperitoneal cesarean delivery. T_3 was immediate fetal extraction and neonatal transition. T_4 was postoperative intensive

care unit cardiopulmonary and autotransfusion surveillance.

2.2. Ethical considerations and consent

All clinical observations, invasive monitoring procedures, and data analyses followed the ethical principles of the Declaration of Helsinki and the privacy guidelines of the International Committee of Medical Journal Editors (ICMJE). Ethical clearance for this observational analytic investigation was granted by the CMHC Research Center Ethics Committee, Indonesia, under protocol registration number 2025/0894. Before emergency surgery and anesthesia, the patient and her legal representatives received multidisciplinary family counseling. It disclosed the American Society of Anesthesiologists (ASA) Physical Status 5E classification and the substantial risk of intraoperative Death on Table (DOT) inherent to Eisenmenger syndrome in pregnancy. Written informed consent was obtained It came from the patient and her legal guardian and covered emergency clinical management as well as anonymized scientific publication of clinical data and diagnostic imaging.

2.3. Participants and diagnostic assessment

Eligibility was narrow. It required an obstetric patient in the third trimester with echocardiographically confirmed Eisenmenger physiology from an uncorrected congenital intracardiac shunt, complicated by acute-on-chronic cardiopulmonary decompensation that required emergency cesarean delivery. Eisenmenger syndrome and acute cardiorespiratory failure were diagnosed from clinical, echocardiographic, radiographic, and laboratory criteria. Transthoracic echocardiography (2 January 2025) assessed atrial and ventricular septal integrity and shunt direction. It graded the severity of tricuspid regurgitation (TR) and pulmonary regurgitation (PR) jets and the echocardiographic probability of pulmonary hypertension. It also assessed right ventricular systolic contractility, Biplane Simpson left ventricular ejection fraction (LVEF), left ventricular diastolic filling, and global wall motion. Portable anteroposterior chest radiography (3 January 2025) assessed the cardiothoracic ratio, chamber enlargement, and pulmonary vascular and alveolar-interstitial markings. Oxygenation was quantified by continuous pulse oximetry (SpO_2) during high-flow oxygen via a non-rebreathing mask (NRBM) at 15 L/min (estimated fraction of inspired oxygen [FiO_2] ≈ 0.90). The non-invasive Rice oxygenation index (SpO_2/FiO_2 [S/F] ratio) was also used.

2.4. Perioperative protocol

At baseline (T_0) the patient had a structured six-system physical examination. The systems were B1 airway and breathing, B2 blood and cardiovascular circulation, B3 brain and neurological function, B4 bladder and renal output, B5 bowel and uteroplacental obstetric status, and B6 bone, musculoskeletal, and peripheral integumentary signs. A standardized venous laboratory panel was drawn on 3 January 2025. It included complete blood count (hemoglobin, hematocrit, leukocyte count, platelet count) and serum electrolytes (sodium [Na^+], potassium [K^+], chloride [Cl^-]). Hepatic transaminases (aspartate aminotransferase [AST], alanine aminotransferase [ALT]), serum albumin, and random blood glucose were also measured. Renal function was assessed with serum urea, calculated blood urea nitrogen [BUN = Urea / 2.1428], serum creatinine, and 2021 CKD-EPI estimated glomerular filtration rate [eGFR]. Inflammatory biomarkers were C-reactive protein [CRP] and procalcitonin. During preoperative stabilization (T_1) she received combined oral pulmonary vasodilator therapy. This was sildenafil citrate (Revatio) 3 × 20 mg/day (60 mg/24 h) and beraprost sodium (Dorner) 3 × 20 µg/day (60 µg/24 h). She also received intravenous furosemide 3 × 20 mg/day (60 mg/24 h) and a quantitative fluid restriction. The restriction allowed intravenous 0.9% NaCl at 1,000 mL/24 h plus oral intake of 800 mL/24 h. The target was a net negative cumulative fluid balance of −500 mL/24 h. During preoperative fasting from 09:00 WIB, Ringer's lactate was infused at 100 mL/h. Before anesthetic induction (T_2), three lines were placed: an 18-gauge peripheral venous line, a radial arterial cannula for continuous beat-to-beat ABP monitoring, and a central venous catheter (CVC). Intravenous milrinone and phenylephrine were prepared on immediate standby. Surgical anesthesia for emergency lower-segment transperitoneal cesarean section (SCTP) was achieved with sole graded lumbar epidural anesthesia using 0.75% ropivacaine. Neonatal Apgar assessment at 1 and 5 minutes followed (T_3), then postoperative ICU management (T_4).

2.5. Risk stratification

To place the clinical course in context, maternal values at each observation point were compared with physiological third-trimester reference intervals and perioperative safety thresholds. The thresholds were a platelet count of at least 100 × 10^3/µL for neuraxial epidural catheterization, an S/F ratio below 150 for severe acute respiratory distress syndrome (ARDS), a BUN-to-creatinine ratio above 20:1 for prerenal azotemia, and the De Ritis ratio (AST/ALT) for hepatic congestion. Maternal cardiovascular risk was stratified with three validated instruments. These were the Modified World Health Organization (mWHO) Maternal Cardiovascular Risk Classification (Classes I–IV), the ASA Physical Status Classification, and the Cardiac Disease in Pregnancy II (CARPREG II) risk score. Adverse events were recorded as observed. The three risk instruments are listed in Table 4.

2.6. Calculated indices

Values were calculated from the recorded measurements to describe cardiopulmonary, renal-oncotic, and fluid-balance findings. Anthropometric indices were Body Mass Index (BMI = weight [kg] / height [m]^2) and Mosteller Body Surface Area (BSA = √[(height [cm] × weight [kg]) / 3,600]). Composite cardiovascular indices were Pulse Pressure (PP = SBP − DBP), Mean Arterial Pressure (MAP = [SBP + 2(DBP)] / 3), Shock Index (SI = HR / SBP), and Rate-Pressure Product (RPP = SBP × HR). Oxygenation was summarized by the Rice non-invasive ratio (S/F = SpO_2 / FiO_2). Cardiorenal and hepatobiliary congestion were quantified by Blood Urea Nitrogen conversion (BUN = Urea / 2.1428) and the Urea-to-Creatinine and BUN-to-Creatinine ratios. The race-free 2021 CKD-EPI creatinine equation gave the estimated glomerular filtration rate (eGFR), and the De Ritis transaminase ratio (AST / ALT) was also calculated. Every drug regimen was normalized to maternal body weight (mg/kg/day or µg/kg/day). Deviations from reference limits were expressed as relative percentage deltas. The formulas were Δ% = [(T_n − T_0) / T_0] × 100% and Δ% = [(Observed − Reference Limit) / Reference Limit] × 100%.

3. Results

3.1. Baseline presentation (T_0)

An 18-year-old Indonesian woman (body weight 46.0 kg, height 152 cm [1.52 m]) presented to our tertiary referral hospital at 34–36 weeks of gestation (Gravida 2, Para 0, Abortus 1 [G2P0010Ab0010]). She needed emergency lower-segment transperitoneal cesarean section (SCTP) for acute-on-chronic maternal cardiopulmonary

decompensation and acute fetal compromise. Her first pregnancy had ended in a spontaneous first-trimester abortion (50.0% prior pregnancy loss rate). That was her only loss. For two days she had noticed worsening shortness of breath. It came and went, became much worse with walking or minimal exertion, and eased with rest and supplemental oxygen. The exertional dyspnea came with episodes of presyncopal dizziness and lightheadedness, which point to transient reductions in systemic cardiac output and cerebral perfusion pressure. Congenital heart disease had first been identified at a secondary regional hospital (dr. Iskak Regional General Hospital) in September 2024. She had never had cardiac surgical repair. She had no history of chronic systemic hypertension, diabetes mellitus, bronchial asthma, or chronic kidney disease, and no known drug or food allergies. Her weight (46.0 kg) and stature (1.52 m) gave a BMI of 19.91 kg/m^2. This is +7.62% above the 18.50 kg/m^2 lower normal threshold. The Mosteller BSA was 1.39 m^2, as shown in Table 1.

The six-system examination at baseline (T_0) showed the main features of decompensated Eisenmenger physiology and cardiogenic pulmonary edema. Airway and respiration (B1): the airway was patent. Mouth opening was three fingers, thyromental distance 7 cm, dentition intact without loose prostheses, and cervical spine flexion and extension full. She breathed spontaneously at 20 breaths/min. Her peripheral oxygen saturation (SpO_2) was only 73% despite high-flow oxygen at 15 L/min via a non-rebreathing mask ($FiO_2 \approx 0.90$). That is a relative SpO_2 deficit of −23.96% below the 96% lower normal limit. The non-invasive SpO_2/FiO_2 (S/F) ratio was 81.11, which is −74.25% below the 315 normal threshold and well under 150, a level that indicates severe hypoxemia. The term ARDS is not used here because the edema is cardiogenic. She was severely hypoxemic. Auscultation found bilateral vesicular breath sounds with diffuse bilateral pulmonary rales/crackles (+/+) and no wheezing (−/−), in keeping with acute cardiogenic pulmonary edema on top of right-to-left intracardiac shunting. Cardiovascular findings (B2): the extremities were warm, dry, and erythematous, with a capillary refill time < 2 seconds. Blood pressure was 104/60 mmHg (PP = 44 mmHg; MAP = 74.67 mmHg) and the radial pulse was regular and strong at 88 beats/min. The Shock Index was 0.85, the Rate-Pressure Product 9,152 mmHg·beats/min, and the heart sounds were a single regular S_1/S_2. Neurological status (B3): she was fully conscious (Glasgow Coma Scale 15 [$E_4V_5M_6$]), with symmetrical 3 mm/3 mm reactive pupils and no focal lateralization. Renal monitoring (B4) through an indwelling Foley catheter showed clear yellow urine at 200 mL over 4 hours (50.0 mL/h, or 1.09 mL/kg/h; equivalent to 1,200 mL/24 h). Obstetric abdominal examination (B5) found normoactive bowel sounds and a fundal height of 26 cm. The fetal lie was longitudinal, the estimated fetal weight 2,015 g, and the baseline fetal heart rate 125 beats/min. Musculoskeletal inspection (B6) found bilateral lower-extremity pitting edema (+) and pronounced digital clubbing (+).

Table 1. Baseline maternal anthropometry, six-system examination, transthoracic echocardiography (2 January 2025), chest radiography, and laboratory profile (3 January 2025) against reference intervals.

Clinical / Diagnostic Domain & Parameter	Patient Baseline Value (T_0)	Normative Gestational Reference Interval	Relative Deviation (Δ%) / Calculated Index	Pathophysiological & Perioperative Interpretation
Maternal Age, Gravidity & Gestational Age	18 years; Gravida 2, Para 0, Abortus 1 (G2P0010Ab0010) at 34–36 weeks gestation	Low-risk reproductive age: 20–34 years; Term: 37–40 weeks	Adolescent high-risk gravida; 1 prior spontaneous abortion (50.0% prior pregnancy loss)	Late third-trimester plasma volume peak (~40–50% expansion) precipitates acute right ventricular decompensation and fetal compromise
Anthropometry (Weight, Height, BMI, BSA)	Weight: 46.0 kg; Height: 152 cm (1.52 m); BMI: 19.91 kg/m^2; Mosteller BSA: 1.39 m^2	Normal BMI: 18.50–24.90 kg/m^2; no reference interval for BSA	BMI = 46.0 / (1.52)^2 = 19.91 kg/m^2 (+7.62% above 18.50 kg/m^2 lower cutoff)	Small maternal body habitus (BSA 1.39 m^2) necessitates strict weight-normalized fluid and local anesthetic dosing
B1 (Airway & Breathing) & Oxygenation	Patent airway (TM 7 cm, 3-finger opening); RR 20 breaths/min; SpO_2 73% on O_2 NRBM 15 L/min; bilateral rales (+/+)	RR: 12–20 breaths/min; SpO_2: 96–100% (room air); S/F ratio: > 315	SpO_2 Δ% = −23.96% vs 96% LLN; S/F ratio = 73 / 0.90 = 81.11 (−74.25% vs 315 cutoff)	Refractory arterial hypoxemia from balanced/right-to-left ASD shunting compounded by cardiogenic alveolar-interstitial edema (severe hypoxemia from shunt and cardiogenic edema)
B2 (Cardiovascular Hemodynamics)	BP: 104/60 mmHg; HR: 88 beats/min (regular, strong); CRT < 2 s; S_1S_2 single regular	BP: 90–120 / 60–80 mmHg; MAP: 70–95 mmHg; HR: 60–100 beats/min	PP = 44 mmHg; MAP = 74.67 mmHg; Shock Index (SI) = 0.85; RPP = 9,152 mmHg·bpm	Borderline systemic arterial pressure where any anesthesia-induced SVR drop risks immediate catastrophic right-to-left shunt reversal
B3 (Neurological) & B4 (Renal Output)	GCS 15 ($E_4V_5M_6$), pupils 3/3 mm reactive; Urine: 200 mL / 4 h via Foley catheter	GCS: 15; Urine output: ≥ 0.5–1.0 mL/kg/h (≥ 23.0–46.0 mL/h)	Hourly urine output = 50.0 mL/h (1.09 mL/kg/h; 1,200 mL/24 h baseline)	Preserved cerebral perfusion after presyncopal episodes; urine output within the expected range
B5 (Obstetric) & B6 (Musculoskeletal)	fundal height 26 cm, EFW 2,015 g, FHR 125 bpm; Bilateral leg edema (+), digital clubbing (+)	FHR: 110–160 bpm; Edema: absent; Digital clubbing: absent (−)	EFW 2,015 g (intrauterine growth restriction risk from chronic maternal hypoxemia)	Digital clubbing is in keeping with a longstanding unoperated cyanotic or bidirectional shunt (proposed megakaryocyte–PDGF/VEGF mechanism)
Transthoracic Echocardiography (2 Jan 2025)	Large secundum ASD (balanced shunt); Severe TR (high PH probability); Moderate PR; Reduced RV contractility; LVEF 51% (Biplane); Reduced LV diastolic function	Intact atrial septum; TR velocity ≤ 2.8 m/s; Normal RV systolic & LV diastolic function; LVEF ≥ 55%	LVEF 51% (−7.27% vs 55% threshold); High PH probability with RV contractile impairment	Eisenmenger physiology with severe right heart pressure/volume overload and interventricular septal shift impairing LV diastolic filling
Thoracic Radiography (CXR, 3 Jan 2025)	Marked cardiomegaly with right ventricular enlargement (RVE configuration) and prominent pulmonary artery segment	Cardiothoracic ratio (CTR) ≤ 0.50; normal pulmonary vascular markings	Pathological RVE configuration with central pulmonary arterial dilation	Radiographic hallmark of chronic left-to-right shunt remodeling into severe pulmonary arterial hypertension
Hematology & Coagulation Readiness (3 Jan 2025)	Hb: 11.3 g/dL; Hct: 36.1%; WBC: 9.33 × 10^3/μL; PLT: 185 × 10^3/μL; Normal hemostasis profile	Hb: 11.0–15.0 g/dL; Hct: 33.0–45.0%; WBC: 4.50–11.00 × 10^3/μL; PLT: 150–450 × 10^3/μL	Hb +2.73% above 11.0 g/dL LLN; PLT +85.00% above 100 × 10^3/μL epidural safety cutoff	No secondary erythrocytosis (gestational hemodilution proposed); platelet count safely supports neuraxial epidural catheterization
Serum Electrolytes & Random Glucose (3 Jan 2025)	Na^+: 136 mmol/L; K^+: 4.04 mmol/L; Cl^-: 105 mmol/L; Random glucose: 75 mg/dL	Na^+: 135–145 mmol/L; K^+: 3.50–5.00 mmol/L; Cl^-: 96–106 mmol/L; Random glucose: 70–140 mg/dL	Na^+ +0.74% above LLN; K^+ within optimal arrhythmogenic safety zone (4.00–4.50 mmol/L)	Normokalemia and normonatremia at baseline

Clinical / Diagnostic Domain & Parameter	Patient Baseline Value (T_0)	Normative Gestational Reference Interval	Relative Deviation (Δ%) / Calculated Index	Pathophysiological & Perioperative Interpretation
Hepatorenal & Oncotic Panel (3 Jan 2025)	Albumin: 3.10 g/dL; AST: 19 U/L; ALT: 9 U/L; Urea: 62.3 mg/dL (BUN 29.07 mg/dL); Creatinine: 0.94 mg/dL	Albumin: 3.50–5.00 g/dL; AST/ALT: 10–35 U/L; Urea: 15.0–40.0 mg/dL; Cr: 0.50–0.95 mg/dL	Albumin Δ% = −11.43% vs LLN; Urea Δ% = +55.75% vs ULN; Urea/Cr = 66.28 (BUN/Cr = 30.93); De Ritis = 2.11	Mild hypoalbuminemia reduces oncotic pressure; elevated Urea/Cr (> 20:1) is in keeping with prerenal cardiorenal congestion (eGFR 90.2 mL/min/1.73 m^2)
Systemic Inflammatory Biomarkers (3 Jan 2025)	C-Reactive Protein (CRP): 0.35 mg/dL; Procalcitonin (PCT): 0.47 ng/mL	CRP: < 0.50 mg/dL; PCT: < 0.50 ng/mL (sepsis exclusion threshold)	CRP −30.00% below 0.50 mg/dL ULN; PCT −6.00% below 0.50 ng/mL cutoff	Argues against sepsis as the cause of hypoxemia; the PCT value is at the cutoff

Notes: *Abbreviations: ASD, atrial septal defect; AST, aspartate aminotransferase; ALT, alanine aminotransferase; BMI, body mass index; BP, blood pressure; BSA, body surface area; BUN, blood urea nitrogen; CRP, C-reactive protein; CRT, capillary refill time; CXR, chest radiograph; EFW, estimated fetal weight; eGFR, estimated glomerular filtration rate; FHR, fetal heart rate; GCS, Glasgow Coma Scale; Hb, hemoglobin; Hct, hematocrit; HR, heart rate; LLN, lower limit of normal; LVEF, left ventricular ejection fraction; MAP, mean arterial pressure; NRBM, non-rebreathing mask; PCT, procalcitonin; PH, pulmonary hypertension; PLT, platelets; PP, pulse pressure; PR, pulmonary regurgitation; RPP, rate-pressure product; RR, respiratory rate; RV, right ventricle; RVE, right ventricular enlargement; S/F, SpO_2/FiO_2 ratio; SI, shock index; SpO_2, peripheral oxygen saturation; SVR, systemic vascular resistance; TM, thyromental distance; TR, tricuspid regurgitation; ULN, upper limit of normal; WBC, white blood cells.*

3.2. Echocardiography, chest radiography, and vasodilator therapy (T_0 to T_1)

Preoperative cardiac and thoracic imaging defined the anatomical and hemodynamic basis of her cardiopulmonary failure (Figure 1). Transthoracic echocardiography on 2 January 2025 showed a large secundum atrial septal defect (ASD) with a balanced bidirectional shunt, severe tricuspid regurgitation (TR) that gave a high echocardiographic probability of severe pulmonary hypertension, moderate pulmonary regurgitation (PR), and reduced right ventricular systolic contractility. Right heart pressure and volume were overloaded, and left ventricular systolic function was mildly reduced. The Biplane Simpson LVEF was 51% (−7.27% below the 55% normal threshold, the cutoff that the CARPREG II score uses for ventricular dysfunction) [29,30], and global wall motion was normokinetic. Left ventricular diastolic function was reduced, however, because of leftward interventricular septal flattening and diastolic ventricular interdependence. Portable anteroposterior chest radiography on 3 January 2025 agreed with these findings. It showed marked cardiomegaly with the classic right ventricular enlargement (RVE) configuration, a prominent convex main pulmonary arterial trunk ('jug-handle' sign), and bilateral central hilar vascular prominence with interstitial congestion.

To stabilize the patient before emergency delivery (T_0 to T_1), a multidisciplinary cardio-obstetric and intensive care protocol was started. Its goals were to lower PVR, decompress RV volume overload, and clear alveolar-interstitial fluid (Table 2). High-concentration oxygen was kept running via NRBM at 15 L/min to attenuate hypoxic pulmonary vasoconstriction in ventilated lung units. Combined oral pulmonary vasodilator therapy comprised sildenafil citrate (PO Revatio) at 3 × 20 mg/day (60 mg/24 h; weight-normalized dose 1.30 mg/kg/day), which inhibits pulmonary vascular PDE-5 and augments NO–cGMP signaling. It was paired with the oral prostacyclin analogue beraprost sodium (PO Dorner) at 3 × 20 µg/day (60 µg/24 h; 1.30 µg/kg/day). Beraprost stimulates vascular smooth muscle IP receptors and raises intracellular cAMP.

Table 2. Perioperative course of cardiopulmonary parameters, fluid balance, invasive monitoring, anesthetic management, and maternal and neonatal outcomes, T_0–T_4.

Observation Phase & Clinical Milestone	Ventilatory & Oxygenation Status	Cardiovascular & Invasive Hemodynamic Profile	Fluid Balance & Pharmacological Intervention	Obstetric / Neonatal Status & Clinical Trajectory (Δ%)
T_0: Initial High-Risk Triage & Diagnostic Baseline (2–3 Jan 2025)	Spontaneous breathing, RR 20 breaths/min; SpO_2 73% on O_2 NRBM 15 L/min (FiO_2 ~0.90; S/F ratio 81.11); bilateral basal rales (+/+)	BP 104/60 mmHg (MAP 74.67 mmHg, PP 44 mmHg); HR 88 bpm; SI 0.85; RPP 9,152 mmHg·bpm; Echo: Large secundum ASD (balanced shunt), severe TR, moderate PR, reduced RV contractility, LVEF 51%	Initiation of O_2 NRBM 15 L/min; Foley catheterization (baseline urine output 200 mL/4 h = 50.0 mL/h [1,200 mL/24 h]); diagnostic laboratory and CXR completion	G2P0010Ab0010 at 34–36 weeks gestation with exertional dyspnea & presyncope; fundal height 26 cm, EFW 2,015 g, FHR 125 bpm; classified as ASA 5E & mWHO Class IV
T_1: Preoperative Pulmonary Vasodilator & Diuretic Optimization (Pre-OR)	Continuous O_2 NRBM 15 L/min; dyspnea improved at rest with supplemental oxygen and diuretic alveolar decongestion	Pre-induction placement of 18G peripheral IV cannula, invasive Arterial Blood Pressure (ABP) cannula, and Central Venous Catheter (CVC); family counseled regarding Death-on-Table (DOT) risk	Oral Sildenafil 3 × 20 mg/day (60 mg/24 h) + Oral Beraprost 3 × 20 µg/day (60 µg/24 h) + IV Furosemide 3 × 20 mg/day (60 mg/24 h); IV 0.9% NaCl 1,000 mL/24 h + Oral 800 mL/24 h (total input 1,800 mL/24 h); Target BC −500 mL/24 h (target output 2,300 mL/24 h, Δ% = +91.67% vs baseline); fasting from 09:00 WIB bridged with RL 100 mL/h	Acute fetal compromise mandating emergency lower-segment transperitoneal cesarean delivery (SCTP); preoperative coagulation readiness verified (PLT 185 × 10^3/µL)
T_2: Intraoperative Graded Epidural Anesthesia & Emergency SCTP	Spontaneous respiration preserved on high-flow O_2 NRBM 15 L/min; complete avoidance of endotracheal intubation and positive-pressure mechanical ventilation	Graded lumbar epidural block is intended to avoid abrupt sympathectomy; continuous invasive ABP and CVC monitoring guided SVR/PVR management; Milrinone (inodilator) and Phenylephrine (α_1-agonist) prepared on immediate standby	Sole graded Epidural Ropivacaine 0.75% titrated incrementally to T6–T4 surgical anesthesia; strict crystalloid co-loading restriction to prevent RV overdistension	Hemodynamically stable lower-segment transperitoneal cesarean section (SCTP) without intraoperative hypotensive crisis, tachyarrhythmia, or shunt reversal arrest
T_3: Neonatal Extraction & Immediate Perinatal Transition	Maternal spontaneous ventilation and oxygenation maintained; immediate neonatal airway clearance and thermal stabilization	Controlled uterine exteriorization/involution; avoidance of rapid high-dose bolus oxytocin-induced SVR collapse during placental separation	Continuation of epidural surgical analgesia and invasive arterial/central venous pressure surveillance during hysterotomy closure	Live vigorous female neonate delivered (birth weight) Apgar score 7 at 1 min rising to 8 at 5 min; no neonatal death
T_4: Postoperative ICU Cardiopulmonary & Autotransfusion Governance	Continuous ICU respiratory monitoring with high-flow O_2 therapy; resolution of acute cardiogenic pulmonary rales	Continuous invasive ABP and CVC monitoring in ICU across the critical 24–72 h postpartum uterine autotransfusion	Resumption of Sildenafil 3 × 20 mg/day, Beraprost 3 × 20 µg/day, and IV Furosemide 3 × 20 mg/day; enforcement of negative fluid balance	Mother and neonate survived

Observation Phase & Clinical Milestone	Ventilatory & Oxygenation Status	Cardiovascular & Invasive Hemodynamic Profile	Fluid Balance & Pharmacological Intervention	Obstetric / Neonatal Status & Clinical Trajectory (Δ%)
		window; stable maternal hemodynamics	(−500 mL/24 h) and epidural postoperative analgesia	

Notes: Abbreviations: ABP, arterial blood pressure; ASD, atrial septal defect; BC, fluid balance; BP, blood pressure; CVC, central venous catheter; CXR, chest radiograph; DOT, death on table; EFW, estimated fetal weight; FHR, fetal heart rate; HR, heart rate; ICU, intensive care unit; IV, intravenous; LVEF, left ventricular ejection fraction; MAP, mean arterial pressure; mWHO, modified World Health Organization maternal cardiovascular risk classification; NRBM, non-rebreathing mask; OR, operating room; PR, pulmonary regurgitation; PVR, pulmonary vascular resistance; RL, Ringer's lactate; RPP, rate-pressure product; RR, respiratory rate; RV, right ventricle; SCTP, lower-segment transperitoneal cesarean section; S/F, SpO_2/FiO_2 ratio; SI, shock index; SpO_2, peripheral oxygen saturation; SVR, systemic vascular resistance; TR, tricuspid regurgitation.

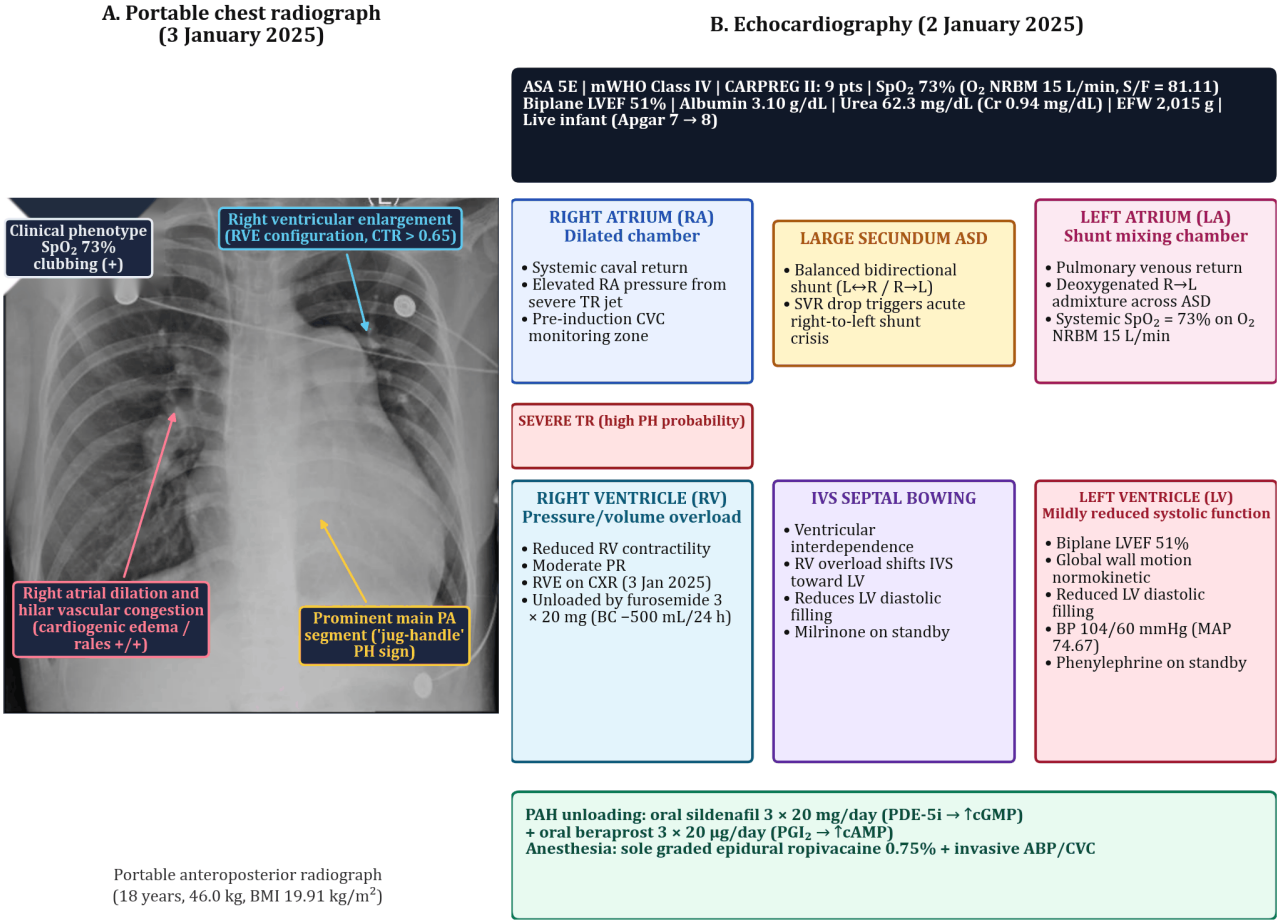
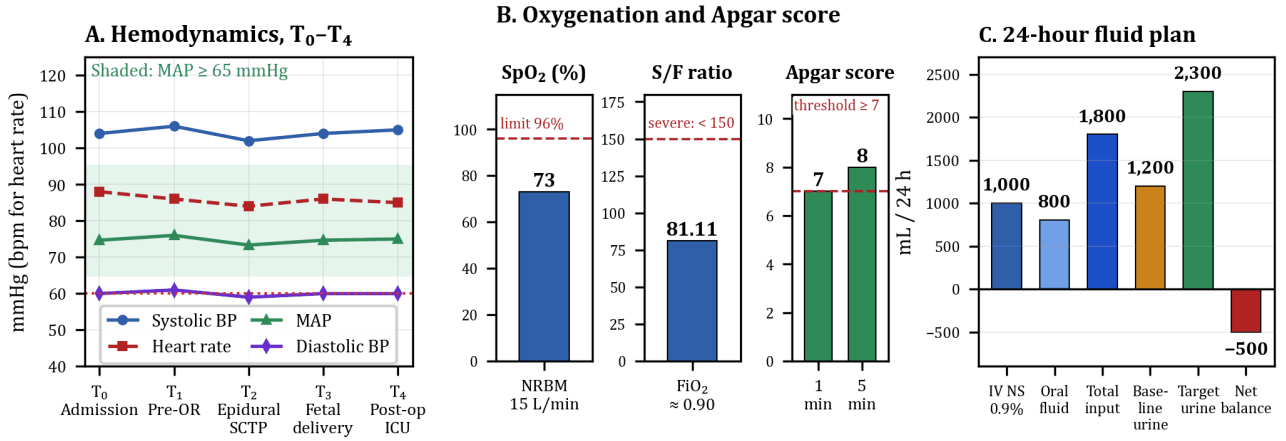


Figure 1. Chest radiograph (3 January 2025) and echocardiographic findings (2 January 2025) at admission (T_0). (A) Portable anteroposterior radiograph showing cardiomegaly with right ventricular enlargement (RVE configuration, cardiothoracic ratio > 0.65), a prominent convex main pulmonary artery segment ('jug-handle' sign), and bilateral hilar vascular congestion with cardiogenic edema. (B) Schematic of the echocardiographic findings: large secundum atrial septal defect (ASD) with balanced bidirectional shunt, severe tricuspid regurgitation (TR), moderate pulmonary regurgitation (PR), reduced right ventricular (RV) contractility, leftward septal (IVS) bowing, and a Biplane left ventricular ejection fraction of 51%.

3.3. Fluid balance, loop diuresis, and laboratory profile (T_1)

At the same time, preload and alveolar congestion were reduced during T_1. Intravenous furosemide at 3 × 20 mg/day (60 mg/24 h; 1.30 mg/kg/day) was combined with a strict 24-hour fluid restriction (Figure 2). Maintenance intravenous fluid was limited to 0.9% normal saline at 1,000 mL/24 h (41.67 mL/h), with an oral allowance of 800 mL/24 h (33.33 mL/h). Total fixed 24-hour fluid input was therefore 1,800 mL/24 h (39.13 mL/kg/24 h). The target negative cumulative fluid balance (Target BC) was −500 mL/24 h. To reach it, furosemide was titrated to raise 24-hour urinary output. Baseline output was 1,200 mL/24 h (200 mL/4 h = 50.0 mL/h). The target diuresis was 2,300 mL/24 h (95.83 mL/h, or 2.08 mL/kg/h). That is a +91.67% increase in renal fluid clearance. Fasting began at 09:00 WIB while the patient awaited transfer to the operating theater. During this window, balanced crystalloid maintenance was bridged with intravenous Ringer's lactate at 100 mL/h under strict central venous preload surveillance.



T_1–T_4 values in panel A are as plotted in the original figure [Author query: AQ6]. MAP = (SBP + 2 × DBP) / 3.

Figure 2. Hemodynamics, oxygenation, Apgar score, and 24-hour fluid plan, T_0–T_4. (A) Systolic and diastolic blood pressure, mean arterial pressure (MAP), and heart rate under invasive arterial (ABP) and central venous (CVC) monitoring and graded 0.75% ropivacaine epidural anesthesia (baseline T_0: 104/60 mmHg, MAP 74.67 mmHg, heart rate 88 beats/min, shock index 0.85). (B) SpO_2 73% on a 15 L/min non-rebreathing mask, SpO_2/FiO_2 (S/F) ratio 81.11, and Apgar scores of 7 (1 minute) and 8 (5 minutes). (C) Intravenous 0.9% NaCl 1,000 mL plus oral intake 800 mL gives 1,800 mL/24 h; urine output was 1,200 mL/24 h at baseline and the target is 2,300 mL/24 h (target balance −500 mL/24 h).

Venous blood tests on 3 January 2025 showed her hematological readiness for neuraxial blockade, Starling oncotic balance, and cardiorenal venous congestion (Figure 3). Hemoglobin was 11.3 g/dL (+2.73% above the 11.0 g/dL third-trimester lower reference limit). Hematocrit was 36.1%. Gestational plasma volume expansion may have masked the secondary erythrocytosis usually seen in non-pregnant cyanotic Eisenmenger patients. The leukocyte count was normal at 9.33 × 10^3/µL (9,330/µL). The platelet count was well preserved at 185 × 10^3/µL (185,000/µL). This is +85.00% above the 100 × 10^3/µL minimum threshold that guards against spinal-epidural hematoma. Clinical coagulation status was normal before surgery. Electrolytes were within range at baseline. Sodium was 136 mmol/L (+0.74% above the 135 mmol/L lower limit) and potassium 4.04 mmol/L (within the 4.00–4.50 mmol/L anti-arrhythmic target zone). Chloride was 105 mmol/L. Random blood glucose was 75 mg/dL. Serum albumin was mildly reduced at 3.10 g/dL (Δ% = −11.43% below the 3.50 g/dL lower reference limit), which lowers plasma colloid oncotic pressure and the threshold for hydrostatic alveolar edema. Transaminases were within normal limits (AST 19 U/L, ALT 9 U/L), but the De Ritis ratio (AST/ALT) of 2.11 may reflect hepatic congestion secondary to severe TR. It is not diagnostic when both enzymes are normal. Serum urea was disproportionately elevated at 62.3 mg/dL (Δ% = +55.75% above the 40.0 mg/dL upper limit; calculated BUN = 29.07 mg/dL). Creatinine was 0.94 mg/dL and (2021 CKD-EPI eGFR = 90.2 mL/min/1.73 m^2, recalculated; the equation is not validated in pregnancy). The Urea-to-Creatinine ratio was 66.28 and the BUN-to-Creatinine ratio 30.93 (> 20:1). These values are in keeping with prerenal cardiorenal venous congestion. Intrinsic tubular injury cannot be excluded from one set of values. C-reactive protein was normal at 0.35 mg/dL (−30.00% below the 0.50 mg/dL upper cutoff) and procalcitonin was 0.47 ng/mL (−6.00% below the 0.50 ng/mL systemic bacterial sepsis threshold). Together they argue against sepsis. The severe hypoxemic respiratory failure (S/F ratio 81.11) is best explained by intracardiac shunting and cardiogenic lung edema.

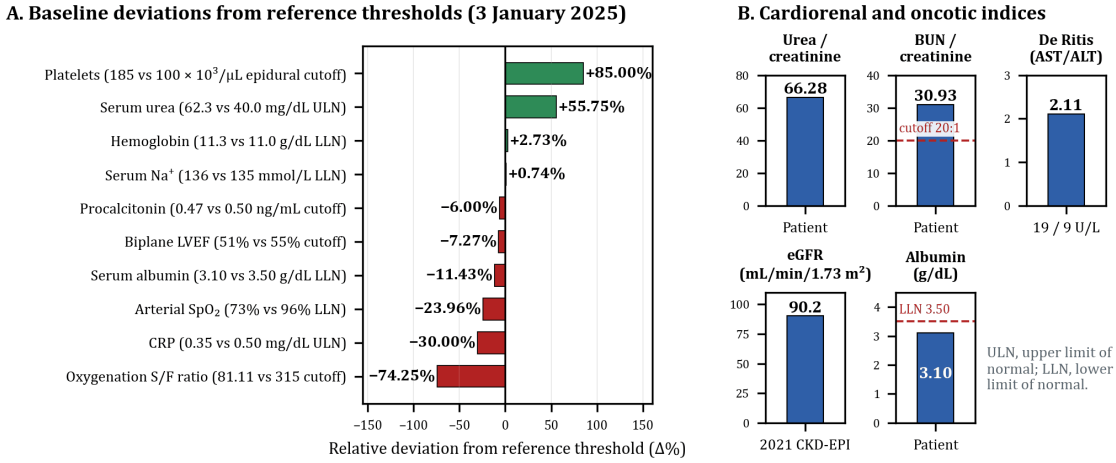


Figure 3. Baseline biomarker profile (3 January 2025). (A) Relative deviations (Δ%) from reference thresholds. (B) Urea/creatinine 66.28, BUN/creatinine 30.93 (cutoff 20:1), De Ritis ratio 2.11 (AST 19 U/L, ALT 9 U/L), 2021 CKD-EPI eGFR 90.2 mL/min/1.73 m^2 (recalculated), and albumin 3.10 g/dL (lower limit 3.50 g/dL).

3.4. Anesthetic management and neonatal outcome (T_2 to T_3)

The patient was assigned ASA Physical Status 5E. The reasons were mWHO Class IV Eisenmenger syndrome, refractory hypoxemia (SpO_2 73%), acute cardiogenic pulmonary edema, reduced RV contractility, and acute fetal compromise requiring emergency surgery. The family was counseled about Death-on-Table (DOT) risk and an 18-gauge peripheral intravenous line was confirmed. Pre-induction invasive radial Arterial Blood Pressure (ABP) cannulation and Central Venous Catheter (CVC) insertion followed, before anesthetic induction (T_2). General endotracheal anesthesia and single-shot spinal anesthesia were rejected to protect the fragile SVR/PVR equilibrium. Sole graded lumbar epidural anesthesia with 0.75% ropivacaine was chosen instead, with the PDE-3 inodilator milrinone and the pure α_1-adrenergic agonist phenylephrine prepared at the bedside (Table 3).

Table 3. Perioperative drugs: weight-normalized doses (46.0 kg), molecular targets, and expected effects on SVR, PVR, RV preload, and contractility.

Therapeutic Agent & Administration Route	Prescribed Regimen & Weight-Normalized Dose (46.0 kg)	Primary Molecular / Receptor Mechanism	Directional Vector on SVR, PVR, RV Preload & Contractility	Specific Protective Role in Gravid Eisenmenger Physiology
Sildenafil Citrate (PO Revatio)	3 × 20 mg/day orally (60 mg/24 h = 1.30 mg/kg/day)	Selective inhibition of phosphodiesterase type 5 (PDE-5), preventing cGMP degradation in pulmonary vascular smooth muscle and amplifying endogenous NO–sGC–cGMP signaling	PVR: ↓↓ (potent reduction); SVR: ↔/↓ (mild); RV Afterload: ↓↓; Shunt (R→L): ↓	Reduces pulmonary arterial resistance and right ventricular pressure overload across the balanced secundum ASD, and published pregnancy data have not shown a clear teratogenic signal[31]
Beraprost Sodium (PO Dorner)	3 × 20 µg/day orally (60 µg/24 h = 1.30 µg/kg/day)	Stable oral prostacyclin (PGI_2) analogue binding vascular smooth muscle and platelet IP receptors → activates adenylate cyclase → elevates intracellular cAMP	PVR: ↓↓ (synergistic with PDE-5i); Platelet Aggregation: ↓↓; Pulmonary Microcirculatory Flow: ↑↑	Provides dual pulmonary vasodilation (cAMP + cGMP synergy with sildenafil) and antithrombotic protection against in situ pulmonary arterial microthrombosis
Furosemide (IV Loop Diuretic)	3 × 20 mg/day IV (60 mg/24 h = 1.30 mg/kg/day)	Inhibition of luminal Na^+-K^+-$2Cl^-$ cotransporter (NKCC2) in the thick ascending limb of the loop of Henle plus early venodilation via prostaglandin release	RV Preload: ↓↓; Alveolar Fluid: ↓↓; Interventricular Septal Bowing: ↓; LV Diastolic Filling: ↑	Drives negative cumulative fluid balance (Target BC −500 mL/24 h), resolves cardiogenic pulmonary edema (bilateral rales), and relieves RV volume overload and septal shift
Ropivacaine 0.75% (Sole Graded Lumbar Epidural)	Incremental titrated epidural dosing via indwelling catheter (0.75% pure S-(−)-enantiomer)	Reversible blockade of voltage-gated sodium channels (Nav1.7/Nav1.8 > cardiac Nav1.5) in spinal nerve roots with slower onset and lower lipophilicity than bupivacaine	SVR: ↔ (preserved via slow segmental titration); Catecholamine Surge: ↓↓; PVR: ↓ (blunts pain-induced pulmonary vasoconstriction); RV Myocardial Depression: 0	Achieves surgical T6–T4 anesthesia while avoiding both spinal-induced precipitous SVR collapse and general anesthesia–induced positive-pressure PVR elevation and myocardial depression
Phenylephrine (IV Standby Vasopressor)	Prepared for immediate titrated IV bolus (25–50 µg) or continuous CVC infusion (0.1–0.5 µg/kg/min)	Selective post-synaptic α_1-adrenergic receptor agonist in systemic resistance arterioles with zero β_1-chronotropic activity	SVR: ↑↑ (restores systemic afterload); HR: ↔/↓ (prevents reflex tachycardia); SVR/PVR Ratio: ↑↑; R→L Shunt: ↓↓	Directly counteracts any epidural-induced systemic venodilation/arteriodilation, maintaining systemic arterial pressure above right ventricular systolic pressure
Milrinone (IV Standby Inodilator)	Prepared for immediate CVC infusion (0.25–0.50 µg/kg/min without hypotensive loading bolus)	Selective inhibition of cardiac and vascular phosphodiesterase type 3 (PDE-3) → increases cardiomyocyte and pulmonary arterial cAMP → activates protein kinase A (PKA)	RV Contractility (Inotropy): ↑↑; RV Lusitropy: ↑↑; PVR: ↓↓; SVR: ↓ (requires co-administration with phenylephrine/vasopressin)	Rescues depressed right ventricular systolic contractility and severe tricuspid regurgitation while simultaneously unloading the pulmonary arterial circuit
Oxygen Therapy (NRBM 15 L/min) & Controlled Crystalloid Protocol	O_2 15 L/min via NRBM (FiO_2 ~0.85–0.90); IV 0.9% NaCl 1,000 mL/24 h + Oral 800 mL/24 h (fasting RL 100 mL/h)	Reversal of hypoxic pulmonary vasoconstriction (HPV) in ventilated alveolar units; controlled isotonic intravascular volume maintenance	PVR: ↓ (attenuates HPV); Alveolar-Arterial O_2 Gradient: ↑; RV Volume Overload: prevented	Maximizes dissolved arterial oxygen delivery to the compromised fetus (EFW 2,015 g, FHR 125 bpm) while preventing fluid-induced RV failure

Notes: *Abbreviations: ASD, atrial septal defect; BC, fluid balance; cAMP, cyclic adenosine monophosphate; cGMP, cyclic guanosine monophosphate; CVC, central venous catheter; EFW, estimated fetal weight; FHR, fetal heart rate; FiO_2, fraction of inspired oxygen; HPV, hypoxic pulmonary vasoconstriction; HR, heart rate; IV, intravenous; LV, left ventricle; NO, nitric oxide; NRBM, non-rebreathing mask; PDE-3, phosphodiesterase type 3; PDE-5, phosphodiesterase type 5; PGI_2, prostacyclin; PKA, protein kinase A; PO, per os (oral); PVR, pulmonary vascular resistance; RL, Ringer's lactate; R→L, right-to-left; RV, right ventricle; sGC, soluble guanylate cyclase; SVR, systemic vascular resistance.*

Incremental epidural titration of 0.75% ropivacaine produced a controlled T6–T4 segmental sensory-motor block for emergency lower-segment transperitoneal cesarean section (SCTP). Spontaneous ventilation continued on 15 L/min NRBM oxygen, and abrupt sympathetic vasomotor collapse did not occur. From incision through hysterotomy and placental delivery (T_2 to T_3), beat-to-beat invasive ABP and CVC monitoring guided the team. Systemic arterial pressure was kept near the baseline of 104/60 mmHg (MAP 74.67 mmHg). Heart rate stayed near 84–88 beats/min. No acute right-to-left shunt crisis or RV ischemic arrest occurred. At T_3 a vigorous live female infant was delivered (birth weight). Apgar scores were 7 at 1 minute and 8 at 5 minutes.

3.5. Risk stratification and postoperative course (T_4)

Table 4 lists the risk scores. The patient fell in the maximal risk tier of the mWHO classification (Class IV), the ASA Physical Status classification (5E), and the CARPREG II index. The CARPREG II score was 9 points. The components were baseline cyanosis (SpO_2 73%) +3, pulmonary hypertension +2, ventricular systolic dysfunction (LVEF 51%, below 55%) +2, no prior cardiac intervention +1, and late pregnancy assessment (after 20 weeks) +1, which sum to 9 [29,30]. The predicted maternal cardiac event rate for a score above 4 is 41% [29,30]. No local anesthetic systemic toxicity, epidural hematoma, or vasodilator-induced systemic collapse was observed. The urea of 62.3 mg/dL was already elevated at baseline (T_0), before the T_1 therapy (Table 2).

Table 4. Maternal risk stratification and observed adverse events.

Analytical Instrument & Criterion Domain	Patient-Specific Clinical & Quantitative Evidence	Assigned Score / Classification	Methodological & Clinical Interpretation
Modified WHO (mWHO) Maternal Cardiovascular Risk Classification	Pulmonary arterial hypertension of any etiology, including Eisenmenger syndrome with large secundum ASD, severe TR, reduced RV contractility, and SpO_2 73%	mWHO Class IV (Extremely High Risk: ≥ 40–100% maternal cardiac event rate; 30–60% mortality)	Pregnancy is formally contraindicated; when continuation occurs into the third trimester, multidisciplinary tertiary delivery with invasive monitoring is advised
ASA Physical Status Classification	Moribund 18-year-old gravida with severe acute-on-chronic cardiopulmonary decompensation (SpO_2 73%, S/F 81.11, cardiogenic lung edema) and acute fetal compromise requiring emergency surgery	ASA Physical Status 5E (Emergency)	Justifies explicit preoperative Death-on-Table (DOT) risk disclosure and immediate ICU bed allocation prior to anesthetic induction
CARPREG II Maternal Cardiac Risk Index	Baseline cyanosis (SpO_2 73%) +3; pulmonary hypertension +2; ventricular systolic dysfunction (LVEF 51%, below 55%) +2; no prior cardiac intervention +1; late pregnancy assessment (after 20 weeks) +1[29,30]	CARPREG II score = 9 points (> 4 points = highest-risk tier; predicted maternal cardiac event rate 41%)[29,30]	Quantitatively explains the onset of acute cardiogenic pulmonary edema and presyncope at 34–36 weeks gestation
Observed adverse events	No local anesthetic systemic toxicity, no epidural hematoma (PLT 185 × 10^3/μL), no refractory hypotension, and no vasodilator-induced maternal collapse were reported	None observed	Reported by the authors; intraoperative and ICU values are needed to document this (AQ5)

Notes: *Abbreviations: ASA, American Society of Anesthesiologists; BC, fluid balance; BUN, blood urea nitrogen; cAMP, cyclic adenosine monophosphate; CARPREG II, Cardiac Disease in Pregnancy II risk index; cGMP, cyclic guanosine monophosphate; Cr, creatinine; CVC, central venous catheter; DOT, death on table; ICU, intensive care unit; LAST, local anesthetic systemic toxicity; LV, left ventricle; MAP, mean arterial pressure; mWHO, modified World Health Organization classification; PLT, platelets; RV, right ventricle; S/F, SpO_2/FiO_2 ratio; SpO_2, peripheral oxygen saturation; TR, tricuspid regurgitation.*

After the cesarean section the patient went directly to the Intensive Care Unit (T_4). There she had continuous invasive ABP and CVC monitoring, high-flow oxygen, and epidural postoperative analgesia. Oral sildenafil (3 × 20 mg/day), oral beraprost (3 × 20 μg/day), and intravenous furosemide (3 × 20 mg/day) continued without interruption. The aim was a net negative fluid balance of −500 mL/24 h across the key 24–72 hour postpartum uterine autotransfusion window. Mother and neonate both survived. There was no postoperative cardiopulmonary arrest, thromboembolism, or need for renal replacement. Table 5 lists published cohorts and series (2021–2025) for comparison.

Table 5. Published cohorts and series of pregnancy in Eisenmenger syndrome and severe pulmonary hypertension (2021–2025), as described in their abstracts, compared with the present report.

Author, Year & Country [Reference]	Study Design & Patient / Cohort Characteristics	Cardiopulmonary & Shunt Severity Profile	Anesthetic Modality, Invasive Monitoring & PAH Therapy	Maternal-Neonatal Outcomes & Key Analytical Convergence / Divergence
Dachlan et al., 2021 (Indonesia)[8]	Retrospective cohort of 18 pregnant women with Eisenmenger syndrome at Dr. Soetomo Hospital, January 2018–December 2019	Atrial septal defect, ventricular septal defect, or patent ductus arteriosus; all had pulmonary hypertension (3 moderate, 15 severe); 94% in NYHA class III–IV	Cesarean delivery in 88.9%; anesthetic technique and PAH therapy not given in the abstract	Seven maternal deaths (7/18 = 38.9%), all from cardiogenic shock; preterm birth 78%, low birth weight 94%, intrauterine growth restriction 55%, severe preeclampsia 33%
Humbert et al., 2022 (Europe)[2]	ESC/ERS guidelines for the diagnosis and treatment of pulmonary hypertension	Group 1 PAH including PAH-CHD and Eisenmenger syndrome	Calcium channel blockers, PDE-5 inhibitors, and inhaled, intravenous or subcutaneous prostacyclin analogues are considered safe in pregnancy; endothelin receptor antagonists, riociguat, and selexipag should be stopped	Treatment in multidisciplinary centers experienced in PH in pregnancy; early termination is advised for intermediate- or high-risk profiles with RV dysfunction; the guideline text gives no recommendation on mode of delivery or type of anesthesia
Zhang et al., 2023 (China)[10]	Multicenter cohort (7 tertiary hospitals, 1993–2019) of 2,220 pregnant women with congenital heart disease, 729 with pulmonary hypertension (398 mild, 331 moderate-to-severe)	Pulmonary hypertension by echocardiography or catheterization; moderate-to-severe defined as an RV–RA gradient > 50 mm Hg	Not summarized in the abstract; multidisciplinary follow-up and antenatal supervision advised	Maternal mortality 0.1% (no PH), 0 (mild PH), and 5.7% (moderate-to-severe PH; 19 women); death or heart failure 7.8% (mild) versus 39.6% (moderate-to-severe)
Sun et al., 2025 (China)[25]	Retrospective study (2010–2020) of 255 pregnant women with shunt-related CHD-associated PH (147 mild-to-moderate, 108 severe)	Severe PH defined as PASP > 70 mm Hg; Eisenmenger syndrome and gestational week among risk factors	Postoperative vasoactive drug therapy for more than 48 h after cesarean section or abortion used as an outcome	Maternal mortality 11.11% in severe PH versus 0.68% in mild-to-moderate PH
Chen et al., 2025 (China)[32]	Retrospective cohort of 70 pregnant women with PH (Zhengzhou, 2012–2022)	Mild versus moderate-to-severe PH by sPAP; WHO functional class	Cesarean section in 87.5% and 86.7% of the two groups; regional anesthesia in 70.0%	Maternal mortality 5.71% (4/70) in severe PH; lower birth weight and more NICU admission in the moderate-to-severe group
Present report (Mudianto and Isngadi, 2026; Malang, Indonesia)	Case report (n = 1); 18-year-old G2P0010Ab0010 at 34–36 weeks; ASA 5E, mWHO IV	Large secundum ASD (balanced shunt), severe TR, moderate PR, reduced RV contractility, LVEF 51%, SpO_2 73% (S/F 81.11), albumin 3.10 g/dL, urea 62.3 mg/dL	Sole graded 0.75% ropivacaine epidural anesthesia; pre-induction ABP and CVC; standby milrinone and phenylephrine; oral sildenafil 3 × 20 mg, oral beraprost 3 × 20 μg, intravenous furosemide 3 × 20 mg (target BC −500 mL/24 h)	Mother and infant survived; live infant (Apgar 7 at 1 min, 8 at 5 min); no intraoperative hemodynamic collapse reported

Notes: *Abbreviations: ABP, arterial blood pressure; ASA, American Society of Anesthesiologists; ASD, atrial septal defect; BC, fluid balance; CHD, congenital heart disease; CVC, central venous catheter; EFW, estimated fetal weight; ESC/ERS, European Society of Cardiology / European Respiratory Society; GA, general anesthesia; ICU, intensive care unit; IV, intravenous; LVEF, left ventricular ejection fraction; mWHO, modified World Health Organization classification; NYHA, New York Heart Association; PAH, pulmonary arterial hypertension; PDA, patent ductus arteriosus; PDE-5i, phosphodiesterase-5 inhibitor; PH, pulmonary hypertension; PO, oral; PR, pulmonary regurgitation; RV, right ventricle; S/F, SpO_2/FiO_2 ratio; SpO_2, peripheral oxygen saturation; TR, tricuspid regurgitation; VSD, ventricular septal defect.*

4. Discussion

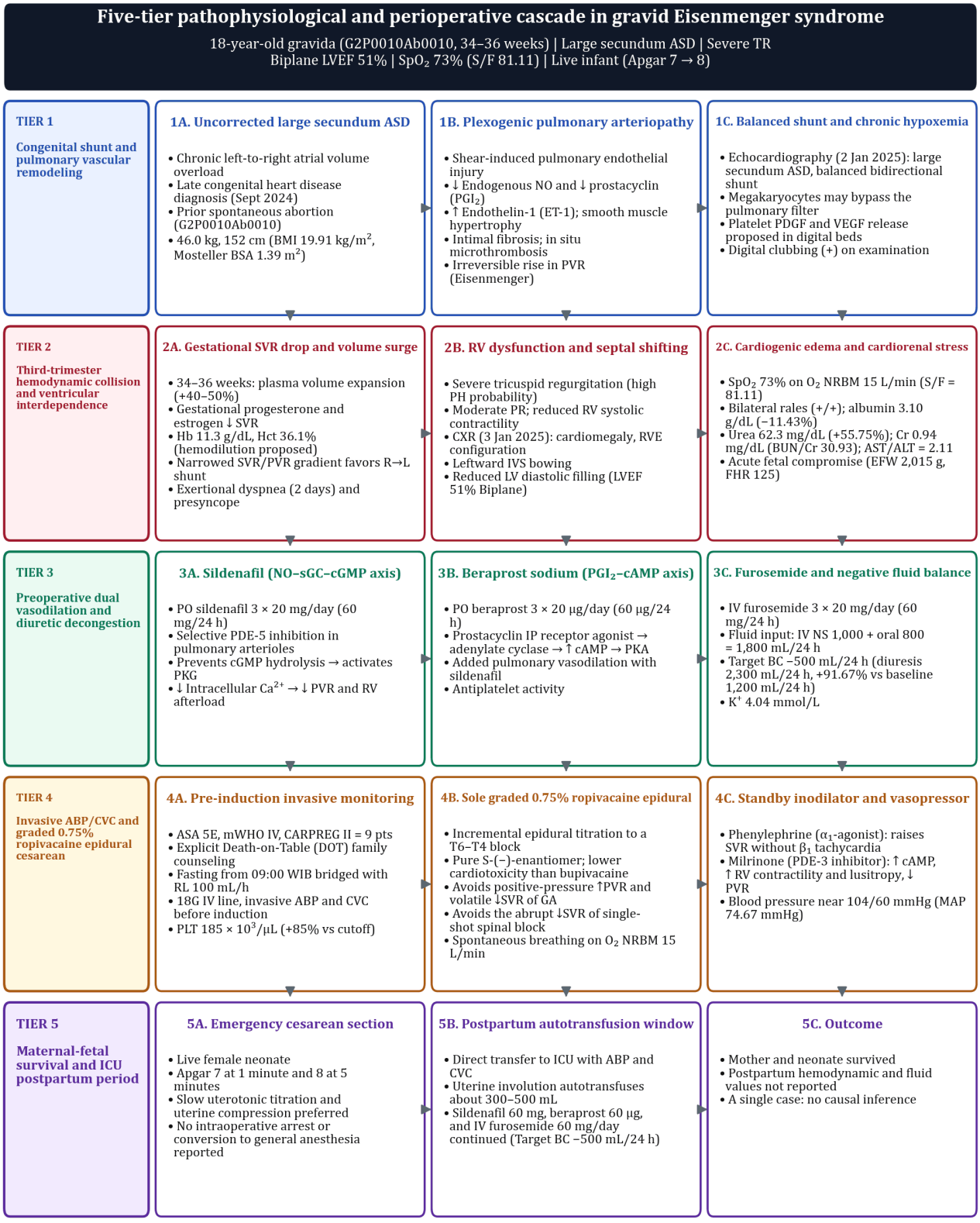
4.1. Pathophysiological cascade

The perioperative course of an 18-year-old ASA 5E gravida with decompensated secundum ASD–induced Eisenmenger syndrome offers a useful model for cardio-obstetric intensive care. She also had severe tricuspid regurgitation, reduced RV contractility, cardiogenic pulmonary edema (SpO_2 73%, S/F ratio 81.11), and acute fetal compromise. The five-tier pathophysiological and anesthetic cascade is shown in Figure 4. Eisenmenger syndrome in pregnancy has long been seen as an almost insurmountable predicament. Maternal mortality has stayed between 30% and 60% for several decades despite advances in obstetric care [5,7]. Even in contemporary tertiary referral cohorts from Indonesia and elsewhere in Asia it remains high. Seven of 18 women died in the series by Dachlan et al. (38.9%), and the deaths clustered in the immediate peripartum and early postpartum windows from refractory right heart failure and cardiogenic shock [8,33]. To understand why this patient survived an emergency third-trimester cesarean delivery without hemodynamic collapse, the physiological interactions can be taken apart across five connected tiers [34].

In Tiers 1 and 2 of the cascade, an uncorrected large secundum ASD exposes the right atrium, right ventricle, and pulmonary arterial tree to chronic volume overload throughout childhood and adolescence. Plexogenic pulmonary arteriopathy develops gradually until PVR approximates SVR and a balanced bidirectional shunt results [1,3]. Pre-tricuspid shunts such as secundum ASD expose the pulmonary vasculature to low-pressure volume overload. Post-tricuspid shunts, such as ventricular septal defects or patent ductus arteriosus, cause high-pressure shear stress instead. True Eisenmenger physiology in isolated secundum ASD therefore usually appears later in adulthood, or when genetic predisposition and pregnancy-induced vascular remodeling are added [35]. In this 18-year-old patient, clinical and echocardiographic findings pointed to established bidirectional or right-to-left shunting and severe pulmonary hypertension. Marked digital clubbing (+) was present. It is thought to result from systemic venous megakaryocytes that bypass the pulmonary capillary filter and lodge in peripheral digital capillaries, where they may release platelet-derived growth factor (PDGF) and vascular endothelial growth factor (VEGF) [36]. SpO_2 fell to 73%, even on 15 L/min NRBM oxygen. Chest radiography showed a prominent main pulmonary artery trunk and RVE configuration. Echocardiography showed severe TR. In the third trimester, gestational plasma volume expansion (+40–50%) coincided with hormonally mediated SVR reduction and mild hypoalbuminemia (3.10 g/dL, Δ% −11.43%). Three consequences followed. First, the systemic-to-pulmonary resistance ratio (SVR/PVR) fell. Right-to-left venous admixture across the large secundum ASD increased. Second, RV dilatation and raised RV systolic and diastolic pressures bowed the interventricular septum leftward into the left ventricle (ventricular interdependence). This impaired LV diastolic filling and caused exertional presyncope despite a Biplane LVEF of 51%. Third, high right-sided filling pressures together with low plasma oncotic pressure favored acute cardiogenic pulmonary edema (bilateral rales +/+) and severe hypoxemic respiratory failure (S/F ratio 81.11 < 150) [14,16,18].

4.2. Oral vasodilation and diuresis

In Tier 3, preoperative stabilization depended on two-pathway oral pulmonary vasodilation combined with loop diuretic preload unloading. Inhaled nitric oxide and intravenous epoprostenol or treprostinil infusions are often unavailable in resource-limited or emergency LMIC settings. There, oral sildenafil citrate (3 × 20 mg/day = 60 mg/24 h) plus oral beraprost sodium (3 × 20 µg/day = 60 µg/24 h) acts on two distinct cyclic nucleotide pathways in pulmonary arterial smooth muscle cells [37–39]. Sildenafil selectively inhibits PDE-5. This prevents hydrolysis of the cGMP generated by endothelial nitric oxide–soluble guanylate cyclase (NO–sGC) signaling. The raised cGMP activates protein kinase G (PKG), which phosphorylates large-conductance calcium-activated potassium (BKCa) channels, hyperpolarizes the smooth muscle membrane, inhibits L-type voltage-gated calcium channels, and lowers cytosolic Ca^{2+}. The result is pulmonary vasodilation, and published pregnancy data on PDE-5 inhibitors have not shown a clear teratogenic signal [2,31,40]. Beraprost sodium is a chemically stable oral prostacyclin (PGI_2) analogue. Its target is different. It binds prostacyclin IP receptors on vascular smooth muscle and platelets, which are coupled to Gs proteins, and stimulates adenylate cyclase to raise intracellular cAMP and activate protein kinase A (PKA) [41]. PKA activation can add to the effect of PKG by dephosphorylating myosin light chains and lowering PVR further. Beraprost also has antiplatelet activity, which may protect the remodeled pulmonary microvasculature from in situ microthrombosis in the hypercoagulable third trimester [41]. Clinical studies in adults with ASD-related PAH and in left heart failure with PAH have examined combined sildenafil and beraprost [37,38].



Abbreviations: ABP, arterial blood pressure; BC, fluid balance; cAMP, cyclic AMP; cGMP, cyclic GMP; CVC, central venous catheter; DOT, death on table; EFW, estimated fetal weight; FHR, fetal heart rate; IVS, interventricular septum; PKA/PKG, protein kinase A/G.

Figure 4. Five-tier cascade in gravid Eisenmenger syndrome: congenital shunt and pulmonary vascular remodeling (Tier 1), third-trimester hemodynamic collision and ventricular interdependence (Tier 2), preoperative vasodilation and diuretic decongestion (Tier 3), invasive monitoring and graded epidural anesthesia (Tier 4), and maternal-fetal outcome and the postpartum period (Tier 5). The schematic combines literature mechanisms with the findings of this case; the mechanisms were not measured in this patient.

Pulmonary vasodilation alone, in a patient with reduced RV contractility and cardiogenic pulmonary edema, would have been insufficient and possibly hazardous. Simultaneous RV preload decompression was needed. Intravenous furosemide (3 × 20 mg/day = 60 mg/24 h) and strict fluid restriction provided it. Total input was 1,800

mL/24 h and the target cumulative fluid balance −500 mL/24 h, which required 2,300 mL/24 h diuresis (Δ% +91.67% above the 1,200 mL/24 h baseline) [42]. A dilated, failing right ventricle works on the flat or descending limb of the Frank-Starling curve. Excess intravascular volume then aggravates tricuspid annular dilatation, worsens severe TR, raises RV free-wall tension and myocardial oxygen consumption, and exaggerates leftward septal shifting into the left ventricle [16,43]. A controlled negative fluid balance of −500 mL/24 h is intended to unload the distended right ventricle, allow the septum to move back toward the midline, improve left ventricular filling, and clear alveolar-interstitial fluid. In this patient dyspnea improved at rest and the rales had resolved by ICU admission (Table 2); no repeat echocardiogram is reported. Cardiorenal values add a diagnostic point. The elevated serum urea (62.3 mg/dL, Δ% +55.75%; BUN 29.07 mg/dL), with a creatinine of 0.94 mg/dL (CKD-EPI eGFR 90.2 mL/min/1.73 m^2) and a BUN/Cr ratio of 30.93 (> 20:1), is in keeping with venous congestion–related cardiorenal physiology. Elevated central venous pressure is transmitted to the renal efferent venules and increases proximal tubular urea reabsorption [17]. Normokalemia (K^+ 4.04 mmol/L) and low inflammatory markers (CRP 0.35 mg/dL, procalcitonin 0.47 ng/mL) argue against sepsis.

4.3. Epidural anesthesia and invasive monitoring

In Tier 4, the choice of sole graded lumbar epidural anesthesia with 0.75% ropivacaine probably contributed to intraoperative stability during emergency lower-segment transperitoneal cesarean section (SCTP). Pre-induction radial ABP and CVC lines and standby milrinone and phenylephrine supported it [21,25,44,45]. In parturients with Eisenmenger syndrome, the anesthetic technique directly governs the SVR/PVR ratio and right ventricular coronary perfusion. General endotracheal anesthesia is generally avoided in Eisenmenger cesarean delivery. Four hemodynamic mechanisms explain this. First, intravenous induction agents (such as propofol or thiopental) and volatile halogenated anesthetics depress RV myocardial contractility and reduce SVR, which accelerates right-to-left shunting across the large secundum ASD. Second, laryngoscopy and tracheal intubation provoke sympathetic catecholamine surges that constrict the reactive pulmonary vasculature and raise PVR. Third, positive-pressure ventilation raises mean intrathoracic and alveolar pressures, compresses alveolar capillaries, increases PVR (Zone 1/2 West lung mechanics), and impedes systemic venous return to the right atrium. Fourth, general anesthesia promotes basal atelectasis. Atelectasis worsens intrapulmonary shunting and is associated with postoperative pneumonia, and apneic desaturation develops faster in a patient whose baseline SpO_2 is already 73% [23,24].

Single-shot subarachnoid (spinal) anesthesia avoids airway manipulation and positive-pressure ventilation. It also produces a dense thoracolumbar sympathectomy within 2 to 5 minutes of intrathecal injection. That block cannot be titrated afterward. Consider a patient with a baseline blood pressure of 104/60 mmHg (MAP 74.67 mmHg) and balanced Eisenmenger shunt physiology. A rapid spinal-induced fall in SVR may precipitate arterial hypotension, right-to-left shunt reversal, severe arterial hypoxemia, and right ventricular subendocardial ischemia. The hypertrophied, pressure-overloaded right ventricle depends on aortic diastolic pressure, here 60 mmHg, to perfuse the right coronary artery throughout the cardiac cycle [46,47]. Published series have nevertheless used low-dose spinal techniques with success in severe pulmonary hypertension, so the choice remains one of balance [46]. With sole graded lumbar epidural anesthesia, 0.75% ropivacaine was titrated incrementally through an epidural catheter. Segmental sympathetic blockade advanced gradually to the T6–T4 dermatome over 15 to 25 minutes. Compensatory baroreceptor vasoconstriction in the unblocked upper-thoracic and cervical segments could therefore preserve SVR and keep MAP near 74.67 mmHg [44,48]. The 0.75% ropivacaine used is a pure S-(−)-enantiomer amide local anesthetic. Compared with racemic bupivacaine it has lower lipid solubility and a lower cardiotoxic potential, and it gave dense surgical anesthesia for cesarean delivery while limiting the risk of local anesthetic-induced RV myocardial depression or refractory arrhythmias [49].

Pre-induction invasive radial ABP and CVC monitoring, together with standby phenylephrine and milrinone, adds to the safety of graded epidural anesthesia [50,51]. Beat-to-beat arterial pressure allows subclinical SVR trends to be seen before pulse oximetry or oscillometric cuffs register decompensation. CVC pressure tracking helps to avoid both hypovolemic preload starvation and crystalloid-induced RV overdistension. Phenylephrine and milrinone were available as a two-part rescue. Phenylephrine, a selective α_1-adrenergic receptor agonist, raises systemic arteriolar resistance (SVR) without β_1-mediated sinus tachycardia, which would shorten diastolic RV filling time and increase myocardial oxygen demand. Milrinone, a selective PDE-3 inhibitor with inotropic and vasodilator effects, raises intracellular cAMP independently of β-adrenergic receptors. It augments depressed RV systolic contractility, improves RV diastolic relaxation, and dilates the pulmonary arterial bed [52,53]. Uterotonic management during fetal extraction (T_3) was also important. Rapid intravenous bolus oxytocin causes profound systemic vasodilation (↓ SVR), reflex tachycardia, and pulmonary vasoconstriction (↑ PVR), and it can trigger circulatory collapse in PAH parturients. Slow titrated uterotonic administration and mechanical uterine compression were therefore preferred. A vigorous female neonate (Apgar 7 at 1 minute and 8 at 5 minutes) was delivered without maternal hypotension [27,54].

4.4. Postpartum period, strengths, and limitations

In Tier 5, the protocol addressed the most dangerous period in the natural history of gravid Eisenmenger syndrome, the first 24 to 72 hours postpartum [14,55]. Registries and case series show that most maternal deaths in Eisenmenger syndrome and severe PAH-CHD occur not at surgical incision but within the first days to two weeks after delivery [34,56,57]. After placental separation and uterine involution, decompression of the inferior vena cava and contraction of the gravid uterus autotransfuse about 300 to 500 mL of uteroplacental blood into the maternal central venous circulation [58,59]. Extravascular interstitial fluid accumulated during gestation also moves into the intravascular space [14]. In a patient with a large secundum ASD, severe TR, and reduced RV systolic contractility, this abrupt preload surge is hazardous. It can overdistend the right ventricle, worsen tricuspid regurgitation, and shift the interventricular septum into the left ventricle. Cardiogenic shock or thromboembolic arrest may follow [7,14]. The patient went directly to the ICU with invasive ABP and CVC lines in situ. Oral sildenafil (60 mg/day), oral beraprost (60 μg/day), and intravenous furosemide (60 mg/day) continued without interruption, targeting a net negative cumulative fluid balance of −500 mL/24 h. Mother and neonate both survived[60,61].

The main strengths of this report are its six-system (B1–B6) physiological profiling and its quantitative description of anthropometric, oxygenation (S/F ratio 81.11), cardiorenal (BUN/Cr 30.93, CKD-EPI eGFR 90.2 mL/min/1.73

m^2), and fluid-balance (−500 mL/24 h) findings. The report concerns one severely ill adolescent parturient (n = 1). Its observations describe physiological and hemodynamic relationships in this patient and do not provide population-level risk estimates. Pulmonary artery (Swan-Ganz) catheterization was avoided because of the hazard of catheter-induced malignant ventricular arrhythmias, tricuspid valve entanglement in severe TR, and pulmonary arterial rupture in Eisenmenger syndrome. Continuous radial arterial pressure, central venous pressure, transthoracic echocardiography, non-invasive S/F oxygenation indexing, and cardiorenal biomarkers guided care instead[7,45]. This report describes a physiology-guided approach that may inform care in similar centers. A single case cannot establish that it is replicable. The main limitations are the missing intraoperative and postoperative record (AQ5), the single set of laboratory values, and the absence of invasive pulmonary pressure measurements.

5. Conclusion

In this adolescent gravida with decompensated secundum ASD–induced Eisenmenger syndrome (ASA 5E, mWHO Class IV, CARPREG II score 9), emergency cesarean delivery was completed with survival of mother and neonate under a physiology-guided perioperative protocol. Her disease was severe. She had severe tricuspid regurgitation, reduced right ventricular systolic contractility, and cardiogenic pulmonary edema (SpO_2 73%, S/F ratio 81.11). Mild hypoalbuminemia (3.10 g/dL) and acute fetal compromise added to the risk. Two oral vasodilators unloaded the pulmonary circulation before surgery: sildenafil (3 × 20 mg/day), which targets the NO–sGC–cGMP pathway, and beraprost sodium (3 × 20 µg/day), which targets the prostacyclin IP receptor–cAMP pathway. Intravenous furosemide (3 × 20 mg/day) enforced a target negative fluid balance of −500 mL/24 h. Dyspnea improved at rest and, by ICU admission, the rales had resolved (Table 2). Intraoperatively, sole graded lumbar epidural anesthesia with 0.75% ropivacaine was given under real-time invasive radial arterial and central venous pressure monitoring, with milrinone and phenylephrine on standby. The technique was chosen to avoid both the positive-pressure PVR surge of general anesthesia and the precipitous SVR collapse of single-shot spinal block. A vigorous neonate was delivered (Apgar 7 to 8). No fatal postpartum decompensation occurred in the intensive care unit. These observations come from one patient. They do not establish that the approach is effective or safe in general.

Declarations

Use of Generative Artificial Intelligence. The authors did not use any generative artificial-intelligence tools to write, analyze, or prepare this manuscript. The authors take full responsibility for its content.

Author Contributions. F.T.M.: Conceptualization, Methodology, Investigation, Data curation, Formal analysis, Writing – original draft. I.: Supervision, Validation, Writing – review & editing. Both authors read and approved the final manuscript.

Funding. None. This research received no external funding.

Conflict of Interest. The authors declare no conflicts of interest.

Ethical Approval. This case report was approved by the CMHC Research Center Ethics Committee, Indonesia (protocol approval number 2025/0894) and followed the Declaration of Helsinki. Written informed consent, including disclosure of the risk of death on the operating table, was obtained from the patient and her legal guardian before clinical intervention.

Patient Consent. Written informed consent was obtained from the patient and her legal guardian for the anonymized publication of clinical, laboratory, echocardiographic, and radiographic data and images, in line with ICMJE and COPE privacy standards.

Data Availability. All clinical, hemodynamic, echocardiographic, radiographic, and laboratory data analyzed in this case report are presented in the article, its tables, and its figures.

Acknowledgments. The authors thank the cardio-obstetric, maternal-fetal medicine, cardiology, nursing, and intensive care teams of the Department of Anesthesiology and Intensive Therapy, Faculty of Medicine, Universitas Brawijaya / Dr. Saiful Anwar General Hospital, Malang, East Java, Indonesia, for their collaboration in the care of this patient.

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