

CASE REPORT

Graded Epidural Anesthesia, Norepinephrine–Milrinone Support, and Fluid Restriction in a Parturient with Severe Tricuspid Regurgitation, Pulmonary Hypertension, Lupus, and Patent Foramen Ovale: An Observational Analytic Case Study

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ABSTRACT

Background. Maternal cardiovascular disease carries high morbidity and mortality. Delivery is especially dangerous when severe tricuspid regurgitation, pulmonary hypertension, lupus and an intracardiac right-to-left shunt coexist.

Objective. To describe the anesthetic management, hemodynamic course and early outcome of one parturient with these conditions who needed emergency Cesarean delivery.

Methods. A 30-year-old gravida 3 para 2 woman at 37+1 weeks presented with antepartum hemorrhage. She had systemic lupus erythematosus (SLEDAI 4), severe functional tricuspid regurgitation (effective regurgitant orifice area 0.4 cm², basal right ventricular diameter 8.1 cm), high-probability pulmonary hypertension (estimated systolic pulmonary artery pressure 74.7 mmHg) and a patent foramen ovale with right-to-left shunting. Cesarean delivery used graded epidural anesthesia with 0.5% levobupivacaine, infusions of norepinephrine, milrinone, dobutamine and furosemide, 500 mL of crystalloid and dilute oxytocin. The CARE guideline was followed.

Results. Mean arterial pressure ranged from 64 to 95 mmHg. A 2.56 kg infant was delivered with Apgar scores of 7, 8 and 9. After delivery, PaO₂ fell to 41 mmHg and recovered within 12 minutes with oxygen and vasoactive titration. Vasoactive support stopped by postoperative day 2. On day 3 the patient moved to the ward with lactate 0.9 mmol/L and estimated systolic pulmonary artery pressure 61.7 mmHg.

Conclusion. Graded epidural anesthesia with norepinephrine and milrinone support and restricted fluids was followed by hemodynamic stability and survival of mother and infant. One case cannot establish causation, and follow-up ended on day 3.

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1. Introduction

Cardiovascular disease is a growing cause of indirect maternal morbidity and mortality worldwide. It is an important cause of maternal illness and death.¹ Pregnancy changes normal physiology considerably: circulating intravascular volume expands by 40% to 50%, resting cardiac output rises, systemic vascular resistance falls, and resting heart rate climbs. A structurally normal heart tolerates these changes well. Women with underlying myocardial, valvular, or pulmonary vascular disease often cannot. Validated risk-stratification tools help clinicians anticipate this. They include the modified World Health Organization (mWHO) classification, the Cardiac Disease in Pregnancy (CARPREG II) model, and the ZAHARA risk score, which discriminate well between patients who will decompensate in specialized tertiary centers and those who will not.^{2,3} Serial measurement of N-terminal pro-B-type natriuretic peptide and cardiac troponin adds to risk prediction during gestation.³ Preconception counseling and risk communication remain inconsistent. Many

reproductive-age women are therefore unaware of their cardiac risk, or remain on teratogenic drugs by oversight.⁴

Pulmonary hypertension sits in the highest risk tier of the mWHO classification (mWHO Class IV), and pregnancy has historically been contraindicated in it.⁵ Nationwide epidemiological data show an in-hospital mortality roughly 85-fold higher than in unaffected parturients. More than 40% of deliveries are followed by cardiorespiratory morbidity, and mechanical circulatory support is needed nine times as often.^{6,7} The cause is mechanical. A pressure-overloaded, chronically afterloaded right ventricle cannot absorb the progressive volume expansion of pregnancy, nor the sudden autotransfusion of 300 to 500 mL of blood that follows uterine involution after delivery. Single-center registries report that maternal risk rises steeply with the severity of pulmonary arterial pressure, and adverse maternal-fetal outcomes cluster in the moderate-to-severe subgroups.⁸ Perioperative care is harder still when pulmonary hypertension arises from a connective tissue disorder such as systemic lupus erythematosus, because of chronic immune-mediated vasculopathy, endothelial activation, corticosteroid-induced suppression of the

hypothalamic-pituitary-adrenal axis, and a baseline prothrombotic microvascular state.⁹

Severe functional tricuspid regurgitation adds a second destabilizing problem. It may arise from primary valvular disease or from annular dilatation secondary to chronic right ventricular pressure overload. Either way, the right ventricle becomes volume-overloaded and compliant but contracts poorly. It is highly vulnerable to both hypovolemia and hypervolemia.¹⁰ In a pulmonary hypertension cohort, severe tricuspid regurgitation independently predicted death, with roughly double the mortality of milder grades.¹⁰ A large multicenter registry of transcatheter tricuspid repair found that outcomes depend on the disease stage at presentation, defined by right ventricular function, renal function, and natriuretic peptides.¹¹ Systemic lupus erythematosus is the most common autoimmune disease in women of reproductive age. In a retrospective cohort of 420 lupus pregnancies, higher disease activity scores (SLEDAI) independently predicted low birth weight, premature birth, and emergent Cesarean delivery, while omitting thromboprophylaxis raised the risk of miscarriage. Care must therefore be individualized and multidisciplinary.¹² Cohorts from Egypt and Romania likewise identify disease flares, secondary antiphospholipid syndrome, and markers of lupus nephritis as predictors of adverse pregnancy outcomes.^{13,14}

Choice of anesthetic technique is the central strategic decision in this patient group. General anesthesia exposes the parturient to the sympathetic hypertensive surge of direct laryngoscopy, to pulmonary vasoconstriction from transient hypoxemia or hypercarbia, and to the preload reduction of positive-pressure ventilation. Any of these can precipitate right ventricular ischemia, uncoupling, and fatal arrest. A review of cardiac disease in pregnancy concludes that incremental neuraxial techniques should be preferred over general anesthesia whenever no explicit contraindication exists.¹⁵ Graded epidural anesthesia differs from single-shot spinal blockade. Local anesthetic is given slowly in fractions. Sympathetic blockade therefore develops progressively and can be titrated, baseline systemic vascular resistance is preserved, abrupt hypotension is avoided, and tracheal intubation, positive intrathoracic pressure, and emergence agitation are all eliminated. Management by a multidisciplinary team of obstetric anesthesiologists, cardiologists, maternal-fetal medicine specialists, and intensivists is the recommended model of care in pulmonary vascular disease and Eisenmenger physiology.^{5,16}

The right ventricle is thin-walled and compliant. It evolved to eject into a low-impedance, highly distensible pulmonary vascular bed, and this design is its main weakness. Acute increases in afterload are tolerated very poorly. Once dilatation begins, wall stress and intracavitary diastolic pressure rise and narrow the transmural perfusion gradient of the right coronary artery. Subendocardial ischemia, progressive contractile failure, and worsening functional tricuspid regurgitation follow, each feeding the next in a downward spiral. In a patient with severe tricuspid regurgitation and pulmonary

hypertension, any sudden rise in pulmonary vascular resistance can cause acute right ventricular failure within minutes. Surgical pain, hypoxemia, hypercarbia, metabolic acidosis, and involuntary Valsalva efforts are all possible triggers. A persistent patent foramen ovale adds another danger. When right atrial pressure exceeds left atrial pressure, an intracardiac right-to-left shunt opens, producing severe refractory arterial hypoxemia and a threat of paradoxical systemic or cerebral embolism.

To our knowledge, the combination of severe functional tricuspid regurgitation, high-probability pulmonary hypertension, lupus, and an unmasked patent foramen ovale with dynamic right-to-left shunting has rarely been reported in the anesthesia literature. The management paired two drugs on purpose: milrinone to reduce right ventricular afterload and norepinephrine to preserve systemic vascular resistance and right coronary perfusion pressure. Restricted fluids and continuous invasive hemodynamic monitoring accompanied both drugs. The objective of this case report is to describe the anesthetic management and perioperative course of this patient across eight predefined time points, and to discuss the reasoning behind each element of the plan.

2. Methods

2.1. Report design and reporting guideline

This is a single-patient case report prepared in accordance with the CARE (Consensus-based Clinical Case Reporting) guideline. Inferential hypothesis testing and p-values were not used, because they are not valid for a single subject. Trajectories are described over time, and serial change is expressed as a percentage difference from baseline.

2.2. Setting

The setting was a tertiary referral university academic medical center with high-acuity obstetric surgical suites, Dr Moewardi General Hospital, Surakarta, with multidisciplinary cardio-obstetric rapid-response services, and dedicated adult intensive care facilities.

2.3. Patient information and case selection

The patient was a 30-year-old gravida 3 para 2 woman presenting at term (37+1 weeks of gestation) with acute antepartum vaginal bleeding. She had known systemic lupus erythematosus on long-term low-dose glucocorticoid therapy. Before admission she took sildenafil 60 mg/day and furosemide 40 mg/day; carvedilol, amlodipine, and ivabradine had been stopped in early pregnancy. The case was selected for reporting because severe functional tricuspid regurgitation, pulmonary hypertension, lupus, and a shunting patent foramen ovale rarely occur together in a parturient.

2.4. Case timeline

The clinical timeline, from the antenatal period to ward transfer on postoperative day 3, is set out in relative time in Table 1.

Table 1. Clinical timeline of the case, from the antenatal period to ward transfer, in relative time.

Time point	Clinical event	Investigations and results	Intervention
Antenatal	Known SLE, severe functional TR, pulmonary hypertension, PFO	Echocardiography (Table 3)	Sildenafil 60 mg/day, furosemide 40 mg/day, long-term glucocorticoid; carvedilol, amlodipine, ivabradine stopped in early pregnancy
Day 0, admission (T0)	37+1 weeks; acute antepartum vaginal bleeding	BP 120/82 mmHg, HR 103/min, SpO ₂ 97% (HFNC, FiO ₂ 0.30); Hb 10.4 g/dL; chest radiograph; echocardiography	Emergency Cesarean delivery decided
Day 0, theatre, 0 min	Arrival; left radial arterial line	BP 111/79 mmHg (MAP 92), HR 100/min, SpO ₂ 100%	Epidural at L2-L3; levobupivacaine 0.5% in 3 mL aliquots to 12 mL with fentanyl 50 µg
Day 0, T1	T4 sensory level reached over 20 min	BP 117/69 mmHg (MAP 83), HR 111/min, SpO ₂ 100%	—
Day 0, +15 min (T2)	Incision; surgical dissection	BP 103/60 mmHg (MAP 71), HR 113/min, SpO ₂ 100%	Norepinephrine, milrinone, dobutamine, furosemide infusions; dexamethasone 5 mg; crystalloid 500 mL
Day 0, +45 min (T3)	Delivery of a 2.56 kg infant (Apgar 7, 8, 9); autotransfusion; shunt hypoxemia	BP 101/52 mmHg (MAP 64); pH 7.295, PaO ₂ 41.0 mmHg, PaCO ₂ 38.0 mmHg, BE -11.0 mmol/L, lactate 2.8 mmol/L; TEE: RV distension, right-to-left shunt	FiO ₂ 1.0; norepinephrine and milrinone titration; dilute oxytocin infusion
Within 12 min of T3	Shunt regressed	pH 7.465, PaO ₂ 95.0 mmHg, PaCO ₂ 31.0 mmHg, lactate 2.1 mmol/L	—
Day 0, +105 min (T4)	Closure; transfer to intensive care	BP 112/62 mmHg (MAP 76), HR 109/min, SpO ₂ 100%	Epidural infusion 0.375% levobupivacaine 3 mL/h; 30° head-up; oxygen titrated
Day 1 (T5)	Hemodynamic recovery	BP 101/78 mmHg (MAP 86), HR 113/min, SpO ₂ 99%; leukocytes 6.7×10^3 /µL; urine 1,850 mL	Dobutamine stopped; norepinephrine weaned off; milrinone 0.25 mL/h
Day 2 (T6)	Vasoactive support stopped	BP 118/75 mmHg (mean), HR 90/min (mean), SpO ₂ 98–99% (air); balance -1,100 mL	Milrinone and furosemide stopped
Day 3 (T7)	Transfer to cardiology-obstetric ward	BP 118/76 mmHg (MAP 90), HR 88/min, SpO ₂ 98% (air); TR velocity 3.8 m/s; sPAP 61.7 mmHg; creatinine 0.62 mg/dL; lactate 0.9 mmol/L; no right-to-left shunt seen	Epidural catheter removed
After day 3	Discharge and last follow-up	—	—

Notes: BE, base excess; BP, blood pressure; HFNC, high-flow nasal cannula; HR, heart rate; MAP, mean arterial pressure; PFO, patent foramen ovale; SLE, systemic lupus erythematosus; sPAP, estimated systolic pulmonary artery pressure; TEE, transesophageal echocardiography; TR, tricuspid regurgitation. Times after the start of the epidural are derived from the clock times in the submission.

2.5. Clinical findings and diagnostic assessment

Each diagnosis was ascertained against standardized international clinical criteria. (1) Severe functional tricuspid regurgitation was established by American Society of Echocardiography (ASE) and European Association of Cardiovascular Imaging (EACVI) criteria.^{17,18} It was characterized by an effective regurgitant orifice area (EROA) of 0.4 cm², a vena contracta width of 0.7 cm, gross right ventricular basal diameter (RVD3) of 8.1 cm, and systolic flow reversal in the hepatic veins. (2) Pulmonary hypertension was high-probability under the 2022 ESC/ERS guidelines.¹⁹ It rested on a peak tricuspid regurgitant velocity above 3.4 m/s (recorded at 4.4 m/s post-Valsalva) and an estimated systolic pulmonary artery pressure of 74.7 mmHg (assuming a baseline right atrial pressure of 5 mmHg). Supportive secondary echocardiographic markers were shortened right ventricular outflow tract acceleration time and interventricular septal flattening. (3) Systemic lupus erythematosus was established by the 2019 EULAR/ACR classification criteria. Disease activity was mild [Systemic Lupus Erythematosus Disease Activity Index [SLEDAI] score of 4) on long-term low-dose glucocorticoid therapy. (4) Transesophageal echocardiography confirmed an interatrial patent foramen ovale with right-to-left shunting. Right-to-left microbubble passage and color Doppler flow across the fossa ovalis, unmasked by elevated right atrial pressures, demonstrated it.

For context, the patient's risk was framed against the modified World Health Organization classification (mWHO Class IV), the CARPREG II model,²⁰ and the ZAHARA score,²

which was derived in congenital heart disease and may not fit acquired tricuspid regurgitation. Published registry cohorts of pregnant women with Eisenmenger syndrome, primary Group 1 pulmonary arterial hypertension, and dilated peripartum cardiomyopathy,²¹ the prospective ROPAC (Registry Of Pregnancy And Cardiac disease), and nationwide inpatient databases^{6,7} provided context for intensive care use, mechanical circulatory support, and maternal mortality, together with specialized obstetric anesthesia guidance.²²

2.6. Therapeutic intervention

Standard American Society of Anesthesiologists monitors and an invasive 20-gauge left radial arterial catheter were placed in the obstetric operating theatre. The patient lay in the right lateral decubitus position. At 08:30 an 18-gauge Tuohy needle entered the L2-L3 intervertebral space using the loss-of-resistance to air technique. A multi-orifice epidural catheter was advanced 4 cm into the epidural space and secured. Aspiration was negative for blood and cerebrospinal fluid.

Neuraxial blockade was graded. We gave 0.5% preservative-free levobupivacaine in 3 mL fractional aliquots every five minutes, up to a cumulative volume of 12 mL (60 mg), together with 50 µg of fentanyl. Surgical incision began at 08:45.

A pathophysiology-driven multimodal infusion strategy was used during the 90-minute operation (Table 2). Norepinephrine ran continuously at 5.2 mL/h (0.05 to 0.08 µg/kg/min) to sustain systemic vascular resistance and preserve the aortic root pressure needed for right coronary

arterial perfusion. Milrinone was infused at 1.0 mL/h (0.375 µg/kg/min) with no loading bolus. It inhibits phosphodiesterase-3, which augments right ventricular contractility and dilates the pulmonary vascular bed, lowering right ventricular afterload and functional tricuspid regurgitation. Dobutamine at 2.4 mL/h (2.5

µg/kg/min) added inotropic support for the volume-loaded right ventricle. Low-dose furosemide (0.5 mL/h, 5 mg/h) ran continuously to counter hypervolemic right ventricular distension. Intravenous dexamethasone (5 mg) was given as stress-dose cover for chronic glucocorticoid use and for perioperative anti-inflammatory modulation.

Table 2. Intraoperative pharmacotherapy and the intended hemodynamic rationale.

Pharmacological Agent	Pharmacological Class	Target Dose / Infusion Rate	Target Receptor & Hemodynamic Rationale
Levobupivacaine 0.5% / 0.375%	Long-acting amide local anesthetic	Graded epidural boluses (12 mL total) / 3 mL/h infusion	Selective blockade of voltage-gated Na ⁺ channels; provides segmental T4 surgical anesthesia while preserving systemic vascular resistance and avoiding abrupt sympathectomy
Norepinephrine	Mixed α ₁ > β ₁ -adrenergic agonist	5.2 mL/h (0.05–0.08 µg/kg/min)	Potent peripheral α ₁ vasoconstriction preserving systemic vascular resistance and aortic root pressure, protecting the right coronary artery perfusion gradient
Milrinone	Phosphodiesterase-3 inhibitor (inodilator)	1.0 mL/h (0.375 µg/kg/min)	Increases intracellular cAMP in cardiomyocytes and vascular smooth muscle; augments right ventricular inotropy while inducing selective pulmonary vasodilation, reducing RV afterload and functional TR severity
Dobutamine	Synthetic β ₁ > β ₂ -adrenergic agonist (inotrope)	2.4 mL/h (2.5 µg/kg/min)	Augments RV contractile performance and stroke volume intended to avoid excessive chronotropy at low dose alongside milrinone
Furosemide	Loop diuretic (Na ⁺ -K ⁺ -2Cl ⁻ cotransporter inhibitor)	0.5 mL/h (5 mg/h continuous infusion)	Continuous venodilation and controlled natriuresis; prevents acute hypervolemic volume overload of the dilated, poorly compliant right ventricle
Dexamethasone	Synthetic glucocorticoid	5 mg intravenous bolus	Stress-dose coverage for chronic adrenal suppression in SLE and modulation of systemic inflammatory mediator release during surgical stress
Oxytocin	Synthetic nonapeptide neurohypophysial hormone	Dilute infusion (5 IU in 500 mL 0.9% NaCl at 10 mIU/min)	Maintains adequate uterine tone post-delivery while avoiding the systemic vasodilation, reflex tachycardia, and pulmonary vasoconstriction associated with bolus administration
Crystalloid Solution (0.9% NaCl)	Isotonic crystalloid	500 mL total intraoperative volume	Deliberately restrictive fluid loading balancing ongoing insensible loss and blood loss (200 mL) against the risk of acute RV distension

Notes: cAMP, cyclic adenosine monophosphate; EBL, estimated blood loss; Na⁺-K⁺-2Cl⁻, sodium-potassium-chloride cotransporter; PDE-3, phosphodiesterase type 3; PVR, pulmonary vascular resistance; RCA, right coronary artery; RV, right ventricle; SLE, systemic lupus erythematosus; SVR, systemic vascular resistance; TR, tricuspid regurgitation.

Fluid management was restrictive: total intraoperative crystalloid was limited to 500 mL of 0.9% saline. Bolus uterotonics and ergot alkaloids were avoided because of pulmonary hypertension, and uterine hemostasis relied on a dilute, slow oxytocin infusion (5 IU in 500 mL normal saline at 10 mIU/min). After delivery, oxygen was supplemented as needed, and the epidural catheter was kept for postoperative infusion.

2.7. Outcome measures and follow-up

Eight predefined perioperative time points were observed: pre-induction baseline (T0), post-epidural establishment (T1), intraoperative surgical manipulation (T2), immediate post-delivery autotransfusion nadir (T3), post-anesthesia care unit recovery (T4), postoperative day 1 (T5), postoperative day 2 (T6), and postoperative day 3 prior to ward transfer (T7).

Monitoring comprised five-lead electrocardiography, pulse oximetry, and invasive radial arterial catheterization (20-gauge, Arrow International). The catheter was connected to an Edwards Lifesciences TruWave transducer positioned at the phlebostatic axis. This setup captured beat-to-beat arterial waveforms and gave real-time mean arterial pressure (MAP). MAP values in the text are those displayed by the monitor; in the trajectory table MAP was calculated as DBP + [SBP – DBP] / 3. Serial arterial blood gases were drawn into heparinized syringes at prespecified intervals. Analysis followed at once on an automated point-of-care blood gas analyzer (ABL90 FLEX, Radiometer Medical, Copenhagen, Denmark), which determined pH, arterial oxygen tension (PaO₂), arterial carbon dioxide tension (PaCO₂), base excess, and blood lactate concentration. Board-certified cardiac anesthesiologists performed transthoracic (TTE) and

transesophageal echocardiography (TEE) on a Philips EPIQ 7 ultrasound system with S5-1 sector array and X7-2t transesophageal probes. They assessed right ventricular fractional area change, tricuspid annular plane systolic excursion (TAPSE), tricuspid regurgitant jet velocities, and interatrial shunt dynamics. Fluid balance was tallied cumulatively. Sources were a calibrated Foley catheter with an hourly urometer, surgical suction canisters, and gravimetric sponge weighing.

Serial biological change was expressed with percentage delta formulas: Δ% = [(Tn – T0) / T0] × 100%, where T0 represents the pre-induction baseline and Tn the designated follow-up epoch. Hourly urinary output was normalized to body weight (mL/kg/h) to describe renal perfusion. Follow-up ended on postoperative day 3, at ward transfer.

2.8. Ethics and consent

CMHC Research Center Ethics Committee, Indonesia, reviewed the protocol and granted institutional ethical clearance (Protocol Approval Number: 2026/0418). The protocol covered extraction of serial hemodynamic records, arterial blood gas analyses, echocardiographic recordings, and diagnostic imaging. It followed the ethical tenets of the Declaration of Helsinki (2013 Fortaleza revision) and Good Clinical Practice guidelines. The patient gave written informed consent after a full verbal briefing on observational data collection and academic dissemination of anonymized clinical records, laboratory profiles, and diagnostic imaging. All personal identifiers were removed, in line with ICMJE and COPE guidance, and no face is recognizable.

3. Results

3.1. Presentation and baseline findings

On admission the baseline vital signs indicated compensated hemodynamics: blood pressure 120/82 mmHg (MAP 95 mmHg), resting sinus tachycardia with a heart rate of 103 bpm, respiratory rate of 18 breaths/min,

and peripheral oxygen saturation (SpO₂) of 97% on high-flow nasal cannula at an FiO₂ of 0.30. Laboratory results showed mild gestational anemia (hemoglobin 10.4 g/dL), leukocytosis (13.9×10^3 / μ L), serum albumin of 3.3 g/dL, platelet count of 184×10^3 / μ L, and normal baseline coagulation. Table 1 gives the timeline, and Table 3 lists the baseline clinical, laboratory, and echocardiographic findings.

Table 3. Baseline clinical, obstetric, immunological, and echocardiographic characteristics.

Clinical or diagnostic parameter	Observed value	Reference range
Maternal Age and Obstetric Status	30 years, G3P2, 37+1 weeks gestation	Term gestation, emergency presentation
Presenting Obstetric Emergency	Antepartum vaginal bleeding	Indication for emergency Cesarean delivery
Autoimmune Disease Activity	SLEDAI score 4	Mild disease activity
Chronic Pharmacotherapy (Antenatal)	Sildenafil 60 mg/day, Furosemide 40 mg/day	Pre-admission regimen
Baseline Systolic Blood Pressure	120 mmHg	100–120 mmHg
Baseline Diastolic Blood Pressure	82 mmHg	60–80 mmHg
Baseline Mean Arterial Pressure (MAP)	95 mmHg	70–105 mmHg
Baseline Resting Heart Rate	103 bpm	60–100 bpm
Peripheral Oxygen Saturation (SpO ₂)	97% (HFNC, FiO ₂ 0.30)	≥95%
Hemoglobin Concentration	10.4 g/dL	12.0–15.5 g/dL (non-pregnant range)
Total Leukocyte Count (Admission)	13.9×10^3 / μ L	$4.5\text{--}11.0 \times 10^3$ / μ L (non-pregnant range)
Serum Albumin Concentration	3.3 g/dL	3.5–5.0 g/dL (non-pregnant range)
Platelet Count	184×10^3 / μ L	$150\text{--}450 \times 10^3$ / μ L
Tricuspid Regurgitation Severity	Severe (EROA 0.4 cm ² , VC 0.7 cm)	Severe TR: EROA ≥0.40 cm ² , VC ≥0.70 cm
Peak TR Velocity (post-Valsalva)	4.4 m/s (resting 4.0 m/s)	<2.8 m/s; >3.4 m/s indicates high probability of pulmonary hypertension
Estimated Systolic PA Pressure (sPAP)	74.7 mmHg	≤30 mmHg
Pulmonary Vascular Resistance (PVR)	1.56 Wood units	<2.0 Wood units; a value of 2.0 or above indicates a pre-capillary component
Basal RV End-Diastolic Diameter (RVD3)	8.1 cm	<4.1 cm
Interatrial Septal Morphometry	Patent foramen ovale, right-to-left shunt	—
Chest Radiograph Findings	Cardiomegaly, cardiothoracic ratio 60%	—

Notes: EROA, effective regurgitant orifice area; FiO₂, fraction of inspired oxygen; HFNC, high-flow nasal cannula; PA, pulmonary artery; PVR, pulmonary vascular resistance; RV, right ventricle; RVD3, basal right ventricular internal diameter; SLEDAI, Systemic Lupus Erythematosus Disease Activity Index; SpO₂, peripheral capillary oxygen saturation; TR, tricuspid regurgitation; VC, vena contracta width.

3.2. Diagnostic findings

Multimodal echocardiography revealed severe functional tricuspid regurgitation. The effective regurgitant orifice area (EROA) was 0.4 cm² and the vena contracta width was 0.7 cm. The right ventricle was grossly enlarged, with a basal end-diastolic diameter (RVD3) of 8.1 cm, and the septum was flattened. Pulmonary hypertension was high-probability, with an estimated systolic pulmonary artery pressure (sPAP) of 74.7 mmHg and a calculated pulmonary vascular resistance (PVR) of 1.56 Wood units. This PVR is below the 2.0 Wood unit threshold for a pre-capillary component, and right heart catheterization was not performed. Under post-Valsalva strain, the peak tricuspid regurgitant velocity rose from a resting 4.0 m/s to 4.4 m/s. Interatrial color Doppler interrogation confirmed a patent

foramen ovale with intermittent right-to-left shunting. Table 3 lists these findings with their reference ranges.

Because the echocardiogram showed severe right heart enlargement, acute pulmonary congestion had to be excluded before surgical incision. The multidisciplinary team therefore reviewed a preoperative chest radiograph. As shown in Figure 1, it showed marked cardiomegaly with a cardiothoracic ratio of approximately 60%. The lung fields were described as clear, with no cephalization, Kerley B lines, or alveolar infiltrates. The team judged that the resting tachycardia reflected chronic right-sided pressure and volume overload rather than acute left ventricular failure or pulmonary venous congestion, which supported the plan of deliberate fluid restriction.

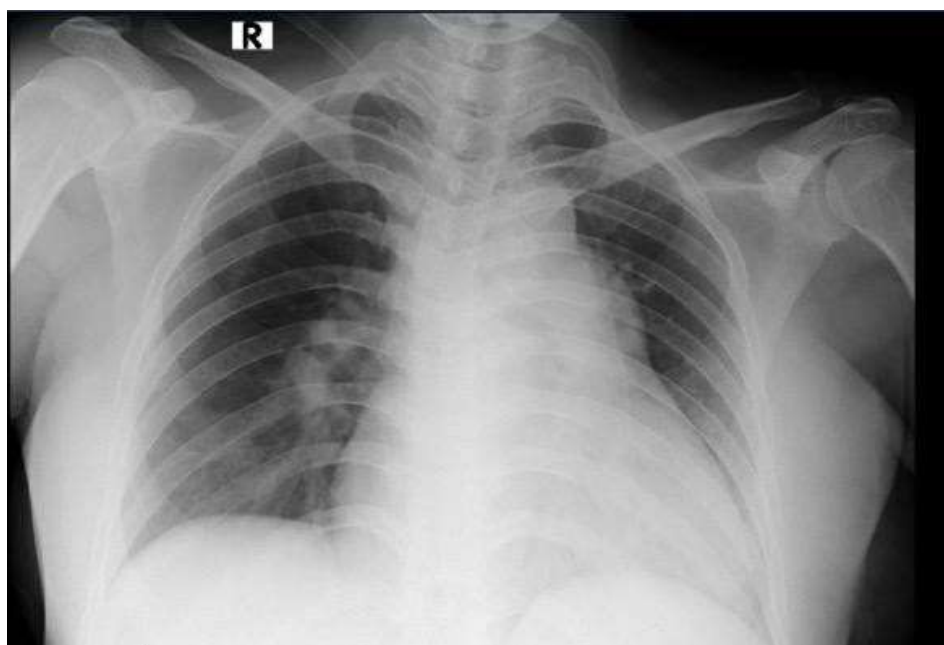


Figure 1. Preoperative chest radiograph showing marked cardiomegaly (cardiothoracic ratio approximately 60%) with lung fields described as clear, consistent with chronic right-sided chamber enlargement rather than acute pulmonary congestion.

3.3. Treatment course and outcome

Blood pressure before induction was 111/79 mmHg (MAP 92 mmHg), heart rate 100 bpm, and SpO₂ 100% on supplemental oxygen. The graded fractional titration produced a slow, progressive bilateral sensory block that

reached the T4 dermatome over 20 minutes. Figure 2 shows the sequential monitor recordings. Blood pressure after the epidural was 117/69 mmHg (MAP 83 mmHg), with a heart rate of 111 bpm and SpO₂ of 100%; no abrupt hypotension occurred.

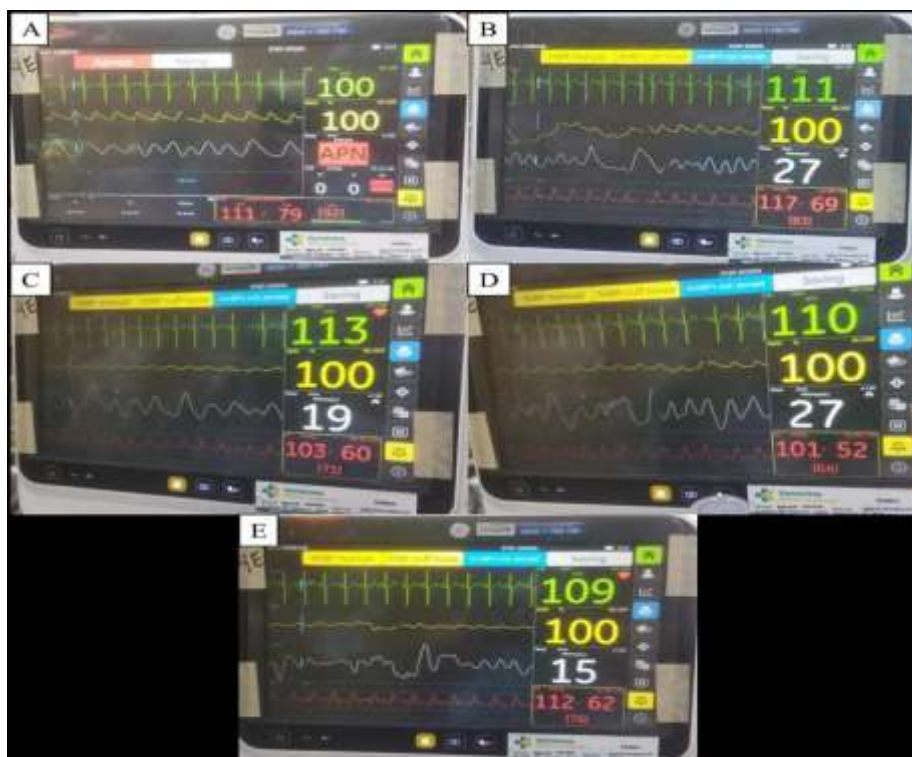


Figure 2. Sequential patient-monitor captures. (A) Pre-induction baseline; (B) post-epidural establishment; (C) intraoperative surgical manipulation; (D) post-delivery nadir; (E) postoperative recovery in the intensive care unit. Each panel displays the electrocardiographic rhythm, heart rate, pulse oximetry, respiratory rate, and arterial blood pressure with mean arterial pressure.

During surgical dissection, blood pressure was 103/60 mmHg (MAP 71 mmHg), with a heart rate of 113 bpm and SpO₂ of 100%. Total intraoperative crystalloid was 500 mL against an estimated surgical blood loss of 200 mL, and the net intraoperative fluid balance was approximately +110 mL.

A male infant weighing 2.56 kg was delivered through a low transverse hysterotomy at 09:15. Adaptation was good. Apgar scores were 7 at 1 minute, 8 at 5 minutes, and 9 at 10 minutes, and umbilical arterial blood gas showed a pH of 7.34 with base excess of −2.4 mmol/L. Routine nursery transfer followed.

After placental delivery and uterine involution, roughly 300 to 500 mL of sequestered blood returned abruptly to the maternal venous circulation. This autotransfusion loaded the right atrium acutely and produced a transient hemodynamic nadir. Blood pressure fell to 101/52 mmHg (nadir MAP 64 mmHg on the monitor), while heart rate climbed to 110–119 bpm.

At the same time, point-of-care arterial blood gas analysis at T3 documented acute metabolic acidemia and hypoxemia. Values were pH 7.295, PaO₂ 41.0 mmHg, PaCO₂ 38.0 mmHg, base excess –11.0 mmol/L, and serum lactate 2.8 mmol/L. Transesophageal echocardiography confirmed acute right ventricular distension and right atrial pressure that had risen above left atrial pressure. It also showed marked right-to-left intracardiac shunting through the patent foramen ovale.

The team responded promptly. They raised the inspired oxygen fraction (FiO₂ 1.0 via non-rebreathing reservoir mask at 10 L/min), kept norepinephrine running to prevent collapse of systemic vascular resistance, and titrated milrinone at 1.0 mL/h. Within 12 minutes the right-to-left shunt regressed on echocardiography and systemic pressure recovered. Follow-up blood gas analysis showed a mild respiratory alkalosis (pH 7.465, PaO₂ 95.0 mmHg, PaCO₂ 31.0 mmHg, base excess –1.2 mmol/L, lactate 2.1 mmol/L).

Closure was uneventful and finished at 10:15. The patient then went to the intensive care unit for specialized invasive monitoring. The immediate post-anesthesia recovery tracing confirmed that hemodynamic stability had returned: blood pressure 112/62 mmHg (MAP 76 mmHg), heart rate 109 bpm, and SpO₂ 100% on supplemental oxygen. The indwelling epidural catheter was switched to a continuous infusion of 0.375% levobupivacaine at 3

mL/h. This gave continuous segmental T10–L1 analgesia (visual analog scale pain score 0–1/10) with no motor blockade, sedation, or respiratory depression. A 30-degree head-up tilt reduced venous return and right ventricular diastolic filling pressures. Oxygen was titrated to keep SpO₂ between 94% and 97%, to avoid both hypoxic pulmonary vasoconstriction and hyperoxic resorption atelectasis.

By postoperative day 1 (T5), hemodynamic recovery was established, although heart rate remained 113 bpm: blood pressure 101/78 mmHg (MAP 86 mmHg), heart rate 113 bpm, and SpO₂ 99% on 3 L/min nasal cannula. Dobutamine was stopped. Norepinephrine was tapered from 3.0 mL/h to full discontinuation by 18:00. Milrinone was reduced to 0.25 mL/h. Total leukocyte count fell by –51.8%, from $13.9 \times 10^3 / \mu\text{L}$ at admission to $6.7 \times 10^3 / \mu\text{L}$, while serum albumin held steady at 3.3 g/dL. Spontaneous diuresis plus infusional furosemide yielded a 24-hour urine output of 1,850 mL against 1,500 mL intake. The cumulative fluid balance was –550 mL.

On postoperative day 2 (T6), milrinone was also stopped, so no vasoactive infusion remained. Spontaneous diuresis exceeded 1.0 mL/kg/h (>1,250 mL/day), so the furosemide infusion could end. Vital signs were self-sustaining: blood pressure 110–125/70–80 mmHg (mean 118/75 mmHg, MAP 89 mmHg), heart rate 85–95 bpm (mean 90 bpm), and SpO₂ 98% to 99% on ambient air. Cumulative balance reached –1,100 mL. Figure 3 plots the arterial blood pressure profiles across the eight epochs. Figure 4 shows the concurrent vasoactive infusion rates, estimated pulmonary artery pressure, arterial oxygenation, and fluid balance. Table 4 gives the hemodynamic, blood gas, and laboratory values across the perioperative stages.

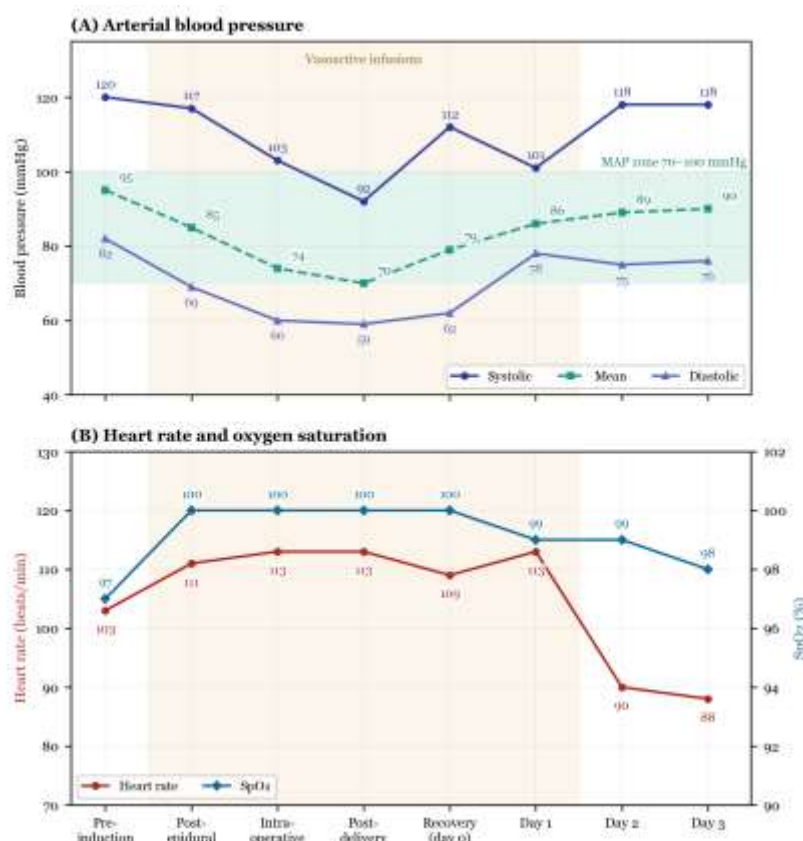


Figure 3. Perioperative hemodynamic trajectories reconstructed from the anesthetic record. (A) Systolic, mean, and diastolic arterial pressure (mmHg), with the shaded 70–100 mmHg MAP zone and the period of vasoactive infusion. (B) Heart rate and SpO₂.

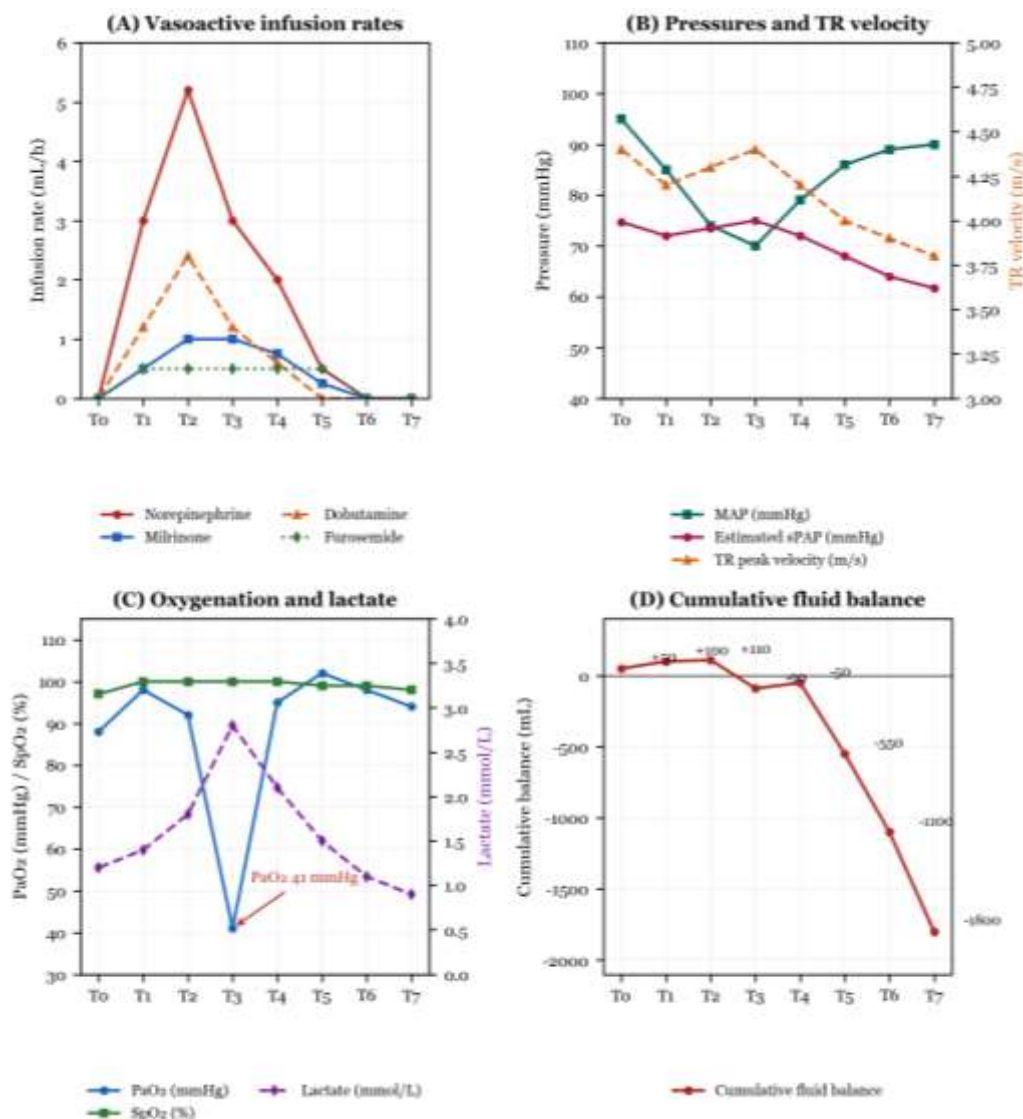


Figure 4. Perioperative trajectories across eight time points. (A) Vasoactive infusion rates. (B) Mean arterial pressure, estimated systolic pulmonary artery pressure, and tricuspid regurgitant peak velocity. (C) PaO₂, SpO₂, and serum lactate. (D) Cumulative fluid balance.

Table 4. Hemodynamic, blood gas, and biochemical values across perioperative stages, with change from baseline.

Parameter	Pre-induction (T0)	Intra-operative (T2)	Post-delivery nadir (T3)	Recovery (T4)	Day 1 (T5)	Day 3 (T7)	Change, T7 vs T0
Systolic Blood Pressure (mmHg)	120	103	92	112	101	118	-1.7%
Diastolic Blood Pressure (mmHg)	82	60	59	62	78	76	-7.3%
Mean Arterial Pressure (mmHg)	95	74	70	79	86	90	-5.3% (nadir -26.3% at T3)
Heart Rate (bpm)	103	113	113	109	113	88	-14.6%
SpO ₂ on Supplemental Oxygen (%)	97	100	100	100	99	98 (Ambient air)	+1 point (HFNC at T0, room air at T7)
Arterial pH	7.410	7.380	7.295	7.465	7.420	7.430	+0.02
Arterial PaO ₂ (mmHg)	88.0	92.0	41.0	95.0	102.0	94.0	+6.8%
Arterial PaCO ₂ (mmHg)	34.0	36.0	38.0	31.0	35.0	36.0	+5.9%
Base Excess (mmol/L)	-1.8	-3.2	-11.0	-1.2	+0.4	+1.2	+3.0 mmol/L
Serum Lactate (mmol/L)	1.2	1.8	2.8	2.1	1.5	0.9	-25.0%
Peak TR Velocity (m/s)	4.4	4.3	4.4	4.2	4.0	3.8	-13.6% (resting baseline 4.0 m/s: -5.0%)
Estimated sPAP (mmHg)	74.7	73.5	75.0	72.0	68.0	61.7	-17.4%

Parameter	Pre-induction (T0)	Intra-operative (T2)	Post-delivery nadir (T3)	Recovery (T4)	Day 1 (T5)	Day 3 (T7)	Change, T7 vs T0
Total Leukocyte Count ($\times 10^3 / \mu\text{L}$)	13.9	—	—	—	6.7	6.4	–54.0%
Serum Albumin (g/dL)	3.3	—	—	—	3.3	3.3	0.0%
Cumulative Fluid Balance (mL)	+50	+110	–90	–50	–550	–1800	—
Vasoactive Support Status	None	NE+MIL+DOB+FUR	NE+MIL (Titrated)	NE+MIL (Weaning)	NE tapered off, DOB stopped	None	Stopped by day 2

Notes: Percentage change is $[(Tn - T0) / T0] \times 100\%$; absolute change is given for pH, SpO_2 , and base excess. MAP is calculated as $\text{DBP} + [\text{SBP} - \text{DBP}] / 3$. An em dash denotes a value not measured at that time. DOB, dobutamine; FUR, furosemide; MIL, milrinone; NE, norepinephrine; PaO_2 , arterial oxygen tension; PaCO_2 , arterial carbon dioxide tension; sPAP, estimated systolic pulmonary artery pressure; SpO_2 , pulse oximetric oxygen saturation; TR, tricuspid regurgitation.

On postoperative day 3 (T7), the patient moved safely from the intensive care unit to the cardiology-obstetric inpatient ward. Vital signs were stable on examination: blood pressure 118/76 mmHg (MAP 90 mmHg, $\Delta\%$ –5.3% vs baseline T0), heart rate 88 bpm ($\Delta\%$ –14.6% vs baseline T0), respiratory rate 16 breaths/min, and SpO_2 98% on room air. Normal coagulation was verified, and the epidural catheter came out intact.

Follow-up transthoracic echocardiography still showed right heart dilatation, with improved function. Peak tricuspid regurgitant velocity was 3.8 m/s, compared with 4.4 m/s at baseline ($\Delta\%$ –13.6%). By the simplified Bernoulli equation this is a 19.6 mmHg fall in the estimated right ventricular-to-right atrial systolic gradient; estimated systolic pulmonary artery pressure fell by 13.0 mmHg, to 61.7 mmHg ($\Delta\%$ –17.4%). No interatrial right-to-left shunt was seen on follow-up color Doppler. Cumulative fluid balance reached –1,800 mL, with serum creatinine 0.62 mg/dL and lactate 0.9 mmol/L.

4. Discussion

4.1. Principal lesson of the case

In this parturient with severe functional tricuspid regurgitation, high-probability pulmonary hypertension, lupus, and an unmasked patent foramen ovale, graded epidural anesthesia combined with norepinephrine, milrinone, restricted fluids, and a dilute oxytocin infusion was followed by hemodynamic stability, a transient shunt hypoxemia that resolved within 12 minutes, and survival of mother and infant.

Neuraxial blockade is the preferred modality for routine Cesarean delivery. Its use in parturients with complex right heart volume overload, fixed pulmonary vascular resistance, and dynamic intracardiac shunts is still debated, and the literature on it is sparse. The favorable course followed four elements applied together: (1) pairing milrinone-mediated pulmonary afterload reduction with norepinephrine-mediated systemic vascular resistance support, (2) restriction of fluid loading to prevent right ventricular distension, (3) avoidance of bolus uterotonics and ergot alkaloids, and (4) staged de-escalation of vasoactive support within 48 hours after delivery. Because they were applied together, the contribution of each cannot be separated.

4.2. Comparison with the published literature

The main hemodynamic goal in a parturient with severe tricuspid regurgitation and pulmonary hypertension is ventriculo-arterial coupling at its optimum. That coupling hinges on a delicate ratio between systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR). We combined norepinephrine, a mixed α_1/β_1 -adrenergic agonist, with milrinone, a selective phosphodiesterase-3

(PDE-3) inhibitor. The aim was to address both sides of this equation at once. Norepinephrine raises systemic arteriolar tone and thereby maintains systemic vascular resistance and aortic root pressure. Milrinone raises intracellular cyclic adenosine monophosphate (cAMP) in pulmonary vascular smooth muscle and in right ventricular cardiomyocytes. The result is lower pulmonary vascular resistance (afterload reduction) and stronger right ventricular inotropic contractility.²³

This balanced approach differs sharply from the routine obstetric reliance on phenylephrine to correct neuraxial-induced hypotension. Phenylephrine is a pure α_1 -adrenergic agonist. It reliably raises systemic blood pressure, but at the price of dose-dependent reflex bradycardia.^{24,25} A right ventricle already loaded with pressure and volume is likely to tolerate a fall in heart rate and cardiac output poorly.

Coronary perfusion of the hypertrophied, dilated right ventricle is a central element of this hemodynamic strategy. A randomized trial in obstetric spinal anesthesia found that intravenous boluses of norepinephrine gave higher maternal cardiac output and less bradycardia than phenylephrine.²⁵ A meta-analysis of vasopressors for Cesarean birth, stratified by maternal risk, likewise favored norepinephrine over phenylephrine in its subgroup analysis.²⁶

Randomized trials in obstetric spinal anesthesia increasingly favor norepinephrine over phenylephrine. In preeclamptic pregnancies, where maternal cardiovascular reserve is limited, a systematic review reports that norepinephrine is associated with higher cardiac output and lower uteroplacental resistance, and with less bradycardia, than phenylephrine.²⁷ A double-blind trial in 100 twin pregnancies, which place a high gestational demand on cardiac output, found no significant difference in the change in heart rate between norepinephrine and phenylephrine infusions; the area under the curve of systolic blood pressure was lower with norepinephrine.²⁸ In healthy women, a titrated prophylactic norepinephrine infusion was followed by less hypotension than norepinephrine boluses given only when pressure fell (17% versus 66%).²⁹ These trials were done in healthy or preeclamptic women with spinal hypotension, not in pulmonary hypertension or right heart failure, so they supply a general rationale only. Avoiding reflex bradycardia and preserving cardiac output were the priorities in our patient.

Milrinone was placed at the center of right ventricular pharmacological support because of the cellular pharmacology of the inodilator class. Houston et al., in a review of right ventricular failure, noted that pulmonary vasodilators and inotropes are used to unload the right ventricle while augmenting contractile performance.³⁰ Yoshida et al. found, in patients with pulmonary arterial

hypertension, that a larger tricuspid regurgitant volume went with higher right atrial pressure, larger right ventricular volumes, and poorer event-free survival.³¹ This suggests, without proving, that reducing right ventricular afterload and volume load could lessen regurgitation and improve forward flow. Ünlü et al. reported that right ventricular strain related to pulmonary artery pressure predicted outcome in pulmonary arterial hypertension better than established parameters.³²

We co-administered milrinone at 1.0 mL/h with low-dose dobutamine (2.4 mL/h, 2.5 µg/kg/min). This gave extra inotropic support; heart rate stayed at 109–113 bpm, above the baseline of 100–103 bpm, so a chronotropic effect cannot be excluded. Milrinone was intended to decompress the pulmonary vasculature.^{23,30}

Fluid management was a second departure from standard obstetric practice. Total intraoperative crystalloid was limited to 500 mL. This followed a basic cardiovascular principle: a dilated, volume-overloaded, poorly compliant right ventricle tolerates fluid loading very badly.³³ In severe functional tricuspid regurgitation, the right ventricle already works on the descending limb of the Frank-Starling curve. Excess volume overdistends the chamber immediately, enlarges the tricuspid annulus further, worsens systolic regurgitation, and pushes the interventricular septum leftward. Through ventricular interdependence, left ventricular filling and systemic cardiac output then suffer.³⁴

The approach contrasts with the preloading or coload strategies routinely used to prevent post-spinal hypotension in healthy parturients or in patients with fixed left-sided stenotic lesions. Fluid regimens in cardio-obstetrics must match the lesion. Preoperative chest radiography showed cardiomegaly without pulmonary venous congestion (Figure 1). This supported our decision to keep the patient in a state of controlled fluid restriction. Kim et al., in recent echocardiographic investigations, stressed that subtle volume and pressure shifts in late gestation carry major prognostic weight. Third-trimester pericardial effusion, for example, can be an early sign of heart failure and preeclampsia in high-risk pregnancies.³⁵

Which uterotonic is chosen, and how it is given, bears directly on safety in parturients with pulmonary vascular disease. A standard rapid intravenous bolus of oxytocin causes abrupt peripheral vasodilation, hypotension, and reflex tachycardia, and may transiently raise pulmonary arterial pressure.³⁶ Randomized trials in Cesarean delivery show this cost directly. In preeclamptic parturients, bolus oxytocin caused significantly larger blood pressure drops and more tachycardia than carbetocin,³⁶ and in high-risk Cesarean sections carbetocin gave better hemodynamic stability, with higher mean arterial pressures and lower heart rates than oxytocin.³⁷

For these reasons, bolus dosing must be avoided entirely in pulmonary hypertension. Oxytocin should run as a dilute, slowly titrated infusion (10 mIU/min) under continuous arterial pressure monitoring.¹ Ergot alkaloids (ergometrine, methylergonovine) and prostaglandin F_{2α} (carboprost) are best avoided, because they constrict the pulmonary vasculature and bronchi.¹ In our patient, a slow, dilute oxytocin infusion secured uterine hemostasis, and no pulmonary hypertensive crisis or systemic collapse followed.

Graded epidural anesthesia was chosen over single-shot spinal or general anesthesia, and this choice was the technical basis of the plan. Single-shot subarachnoid

anesthesia produces rapid, deep sympathetic blockade. In patients with fixed cardiac output who cannot raise stroke volume, the resulting systemic vasodilation can precipitate profound hypotension.³⁸ Suzuki et al. described, in a small retrospective series without a control group, sequential combined spinal and epidural anesthesia with diluted bupivacaine for Cesarean delivery that gave sufficient analgesia.³⁹

Levobupivacaine, the pure S(–)-enantiomer of bupivacaine, was the primary local anesthetic. It carries a lower risk of myocardial and central nervous system toxicity than racemic bupivacaine, a safeguard when cardiovascular reserve is limited.⁴⁰ A meta-analysis by Li et al. in labor analgesia found levobupivacaine and ropivacaine equally effective for maternal and fetal outcomes, so institutional familiarity and drug availability can guide the choice.⁴¹ In a systematic review of regional anesthesia after cardiac surgery, Atalia et al. reported less pain and lower opioid consumption than with opioid therapy.⁴²

After surgery, the epidural catheter stayed in place with a low-dose infusion of 0.375% levobupivacaine at 3 mL/h. It gave continuous segmental analgesia without opioid-related respiratory depression, and it blunted postoperative sympathetic stress. Early physical mobilization became possible.

Chang reviewed the hemodynamic challenges of pregnancy in pulmonary hypertension and noted that labor, delivery, and the early postpartum period are particularly hazardous because abrupt shifts in preload and filling pressures can precipitate right ventricular failure, hypoxemia, or circulatory collapse.⁴³ Emergency presentation and inadequate risk stratification are thought to worsen maternal outcome. Dominoni et al. likewise stressed expert multidisciplinary care for pregnant women with pulmonary hypertension.⁵ Triggers that acutely raise pulmonary vascular resistance must be avoided.

Parturients with systemic lupus erythematosus who take chronic glucocorticoids need perioperative stress-dose cover; guidance on adrenal insufficiency recommends stress doses of hydrocortisone.⁴⁴ Intravenous dexamethasone also has analgesic and opioid-sparing effects after Cesarean delivery.⁴⁵ Dexamethasone 5 mg was used here for both purposes.

Formal risk stratification shows the size of the challenge (Table 5). Under the modified WHO classification, severe pulmonary hypertension places a parturient in mWHO Class IV, and pregnancy is conventionally contraindicated; maternal cardiac event rates of 40% to 100% have been estimated in pregnant women with pulmonary arterial hypertension.⁵ Validated risk models, including CARPREG II and ZAHARA, place such patients at high risk for peripartum cardiovascular events.^{2,20}

Registry analyses from the ROPAC study and contemporary nationwide inpatient databases agree with this. Pregnant women with pulmonary hypertension have high rates of heart failure and intensive care admission. Emergency Cesarean delivery is frequent, and mechanical circulatory support is used nine times more often than in unaffected deliveries.^{6,7} Both mother and infant survived in our case, although the mother had a transient shunt hypoxemia. Pharmacological support was de-escalated by postoperative day 2, and the patient moved to the ward by day 3. This course is compatible with a pathophysiology-driven approach in high-risk cardio-obstetric patients, but

one case cannot show that the approach lowers risk. Table 5 sets the index case beside published comparator groups.

Table 5. Index case compared with published comparator groups of high-risk cardio-obstetric patients undergoing Cesarean section.

Dimension	Index case (present report)	Eisenmenger syndrome or primary PAH	Peripartum or dilated cardiomyopathy
Underlying pathophysiology	Severe functional TR, high-probability pulmonary hypertension, SLE with mild activity, PFO with right-to-left shunt	Fixed pre-capillary pulmonary vascular remodeling with irreversible shunt reversal	Primary left ventricular systolic failure with reduced ejection fraction (<30%)
Modified WHO (mWHO) risk class	Class IV (pregnancy historically contraindicated)	Class IV (high maternal mortality; pregnancy strongly advised against)	Class III–IV (high risk of acute peripartum cardiac failure)
CARPREG II and ZAHARA risk scores	High risk (CARPREG II ≥ 3 ; ZAHARA >3.5) †	High risk (CARPREG II ≥ 4 ; ZAHARA >5.0)	Intermediate to high risk (CARPREG II 2–3) †
Primary anesthetic technique	Graded epidural anesthesia (0.5% levobupivacaine, incremental)	Single-shot spinal, combined spinal-epidural, or general anesthesia	General anesthesia or low-dose combined spinal-epidural
Vasoactive support	Milrinone, norepinephrine, and dobutamine	Phenylephrine or inhaled prostacyclins (iloprost, nitric oxide)	Dobutamine or levosimendan, with or without vasopressors
Intraoperative fluid management	Restrictive (500 mL crystalloid; net balance +110 mL; EBL 200 mL)	Restrictive to cautiously euvolemic	Cautiously restrictive with invasive filling pressure guidance
Uterotonic protocol	Dilute oxytocin infusion (10 mIU/min); bolus and ergometrine avoided	Slow dilute oxytocin infusion; prostaglandin F $_{2\alpha}$ and ergometrine contraindicated	Titrated oxytocin infusion; rapid bolus avoided
Maternal hemodynamic range	SBP 60–110 mmHg, DBP 40–80 mmHg, HR 80–120 bpm, MAP 70–95 mmHg	Severe lability; frequent sudden hypotensive crises	Hypotension common; low cardiac index and pulmonary congestion
Postpartum ICU and mechanical support	Pharmacological support weaned by day 2; ward on day 3; no ECMO or mechanical circulatory support	Prolonged ICU stay; frequent ECMO or mechanical circulatory support (nine-fold higher use ⁷)	High incidence of postpartum decompensation and mechanical ventilation
Maternal and neonatal outcome	Mother alive on day 3; 2.56 kg neonate, Apgar 7, 8, 9 at 1, 5, 10 min	Maternal mortality 30–50%; high rates of fetal growth restriction and prematurity †	Maternal mortality 5–15%; frequent emergency preterm delivery †

Notes: CARPREG, Cardiac Disease in Pregnancy Study; ECMO, extracorporeal membrane oxygenation; EBL, estimated blood loss; HR, heart rate; mWHO, modified World Health Organization; PAH, pulmonary arterial hypertension; PFO, patent foramen ovale; SBP, systolic blood pressure; SLE, systemic lupus erythematosus; TR, tricuspid regurgitation; ZAHARA, Zwangerschap bij Aangeboren Hartafwijkingen.

4.3. Pathophysiological or mechanistic interpretation

The left ventricle is perfused mainly in diastole. Under physiological low-pressure conditions, the right coronary artery supplies the right ventricular myocardium in both systole and diastole. Severe pulmonary hypertension with gross chamber enlargement (basal RV diameter 8.1 cm in our patient) changes this. Elevated right ventricular intracavitary systolic and diastolic pressures compress the intramural microvasculature and narrow the net coronary perfusion pressure gradient (aortic root pressure minus right ventricular end-diastolic pressure). Under such conditions, a modest fall in systemic arterial pressure is enough. Right ventricular subendocardial ischemia follows quickly, then a sudden loss of contractile force, worsening tricuspid regurgitation, and rapid progression toward irreversible cardiogenic shock.

In patients with left heart failure and pulmonary hypertension, Hamud et al. found that right coronary artery diastolic perfusion pressure was a strong independent marker of mortality.⁴⁶ If systemic arterial pressure falls or right ventricular end-diastolic pressure rises, the right coronary perfusion gradient shrinks and right ventricular ischemia may accelerate. A review of right ventricular pressure overload by Dayer et al. describes how chronic afterload elevation leads to maladaptive hypertrophy, dilatation, and impaired ventriculo-arterial coupling.³³ Our protocol held aortic root pressure up with a titrated norepinephrine infusion and lowered right ventricular afterload with milrinone, which was intended to protect the right coronary artery perfusion gradient.

The patient's systemic lupus erythematosus brought several pathophysiological risks. Her clinical disease activity was mild (SLEDAI score 4). Even so, pregnancy in lupus carries an elevated baseline risk of microvascular inflammation, preeclampsia, and thrombosis mediated by

secondary antiphospholipid antibodies.^{12–14} Because of chronic corticosteroid therapy, intraoperative stress-dose glucocorticoid cover (intravenous dexamethasone 5 mg) was given to prevent acute relative adrenal insufficiency under surgical stress.⁴⁴

Three factors converged: pregnancy-induced hypercoagulability, lupus-associated endothelial activation, and postoperative venous stasis. Together they created a very high risk of deep venous thrombosis. With an unmasked patent foramen ovale and right-to-left intracardiac shunting, a venous thrombus could cross into the arterial side. Paradoxical systemic or cerebral arterial embolism, ending in acute stroke, was the feared outcome. Management had to balance thromboprophylaxis against surgical hemostasis. We used early physical mobilization, pneumatic compression devices, and close clinical surveillance.

The patent foramen ovale with dynamic right-to-left shunting caused the most serious intraoperative emergency in this case. Normally, left atrial pressure exceeds right atrial pressure, and the valve of the fossa ovalis stays functionally closed. In our patient, severe tricuspid regurgitation and pulmonary hypertension had produced chronic right atrial hypertension. After delivery, the sudden autotransfusion of 300 to 500 mL of uterine blood into the central circulation worsened it acutely.

Echocardiography indicated that right atrial pressure had risen above left atrial pressure. This forced open the patent foramen ovale and sent a massive right-to-left shunt of deoxygenated blood directly into the systemic circulation. The evidence was a post-delivery arterial PaO $_2$ nadir of 41.0 mmHg and metabolic acidemia (base excess –11.0 mmol/L, lactate 2.8 mmol/L). The cardio-obstetric team responded promptly. They raised FiO $_2$ to 1.0, kept systemic vascular resistance up with norepinephrine, and unloaded

the pulmonary vascular bed with milrinone. The intracardiac shunt regressed within 12 minutes, and arterial oxygenation recovered. No neurological deficit or end-organ injury was noted during the three days of follow-up.

Figure 5 is a hypothetical multi-tier framework that brings together the pathophysiological cascade and the therapeutic mechanisms behind this case. Tier 1 shows the baseline autoimmune vasculopathy that may accompany systemic lupus erythematosus, with endothelial activation and impaired prostacyclin synthesis, and Tier 2 covers the

pulmonary vascular remodeling and high-probability pulmonary hypertension that may follow (sPAP 74.7 mmHg, PVR 1.56 Wood units). Tier 3 captures right ventricular chamber dilatation (RVD3 8.1 cm) and severe functional tricuspid regurgitation (EROA 0.4 cm², VC 0.7 cm). It also shows the resulting interatrial right-to-left shunt across the patent foramen ovale. Tier 4 lists the acute stress triggers: gestational hypervolemia, autotransfusion, and surgical sympathetic surges. Tier 5, finally, shows the combined strategy: graded epidural blockade, milrinone and norepinephrine support, restrictive fluid management, and dilute oxytocin infusion.

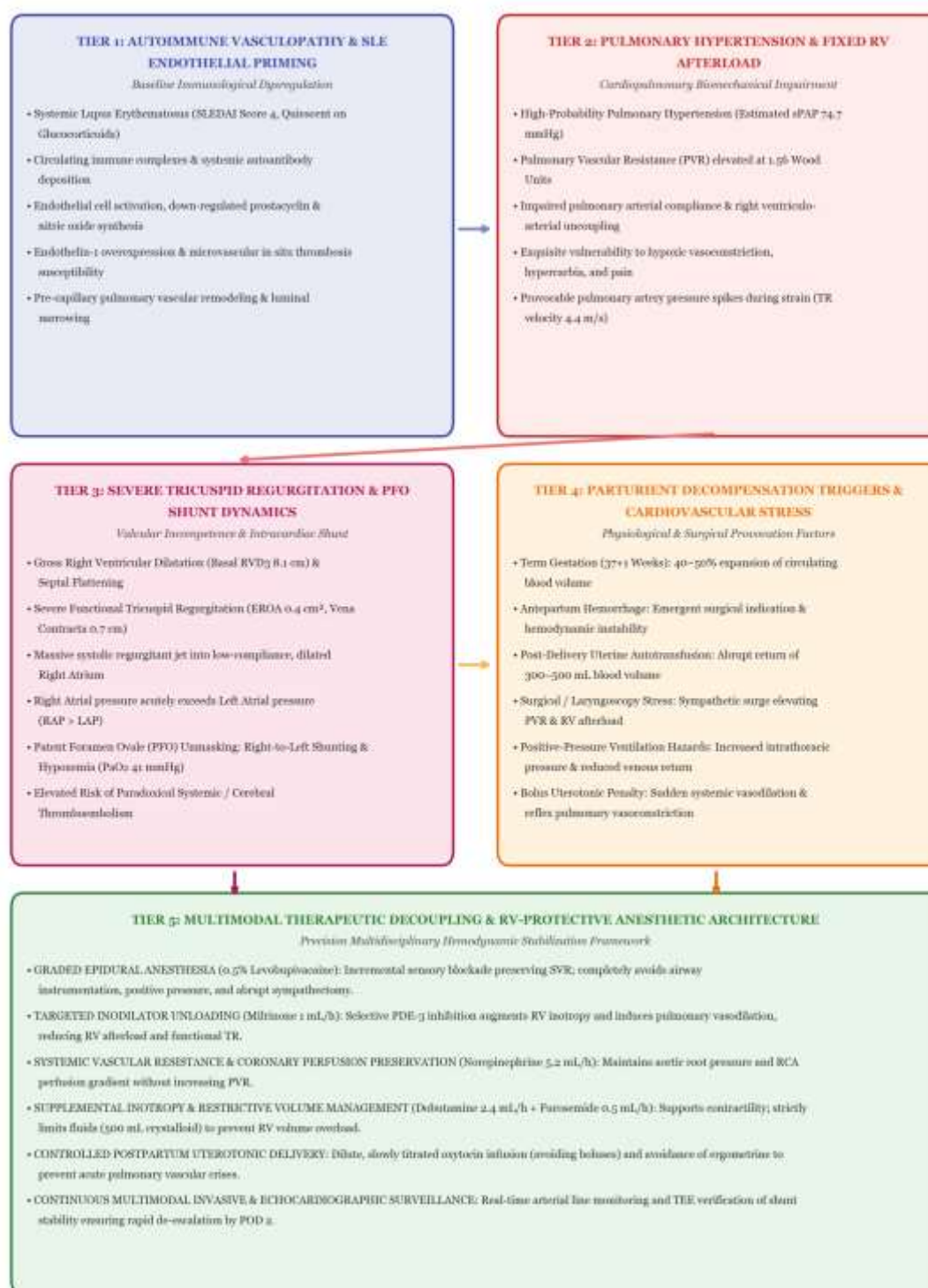


Figure 5. Hypothetical multi-tier schematic of the pathophysiological cascade and the therapeutic strategy in this patient.

4.4. Implications for practice

These observations suggest a framework for parturients with severe right-sided heart disease, whose transferability is uncertain. The framework has five parts. First, choose a graded, titratable neuraxial technique over single-shot spinal or general anesthesia whenever contraindications are absent. Second, manage hemodynamics by explicitly balancing pulmonary against systemic vascular resistance, using combined inodilator and vasopressor support (milrinone plus norepinephrine) instead of pure α_1 -agonists. Third, restrict fluid loading and give uterotonics only as slow, dilute infusions. Fourth, set up continuous invasive arterial and echocardiographic surveillance to guide beat-to-beat titration. Fifth, form a pre-briefed multidisciplinary cardio-obstetric team with escalation pathways agreed in advance.

4.5. Strengths

The record is unusually complete for a case of this complexity. It includes diagnostic criteria for each lesion, equipment and doses, serial hemodynamic, blood gas, and laboratory values across eight time points, echocardiographic follow-up, and an open account of the post-delivery shunt hypoxemia and its treatment.

4.6. Limitations

This is a single patient (n=1), so causal inferences cannot be drawn and the findings cannot be generalized to all parturients with pulmonary hypertension, especially those with active lupus nephritis, severe left ventricular dysfunction, or acute uncompensated right heart failure. Several interventions were given together, and no comparator exists. Pulmonary hypertension was defined echocardiographically as high-probability and was not confirmed by preoperative right heart catheterization, so direct thermodilution measurement of pulmonary vascular resistance and cardiac index was not possible. Formal antiphospholipid antibody titers were absent from the perioperative record, which limits stratification of thrombotic risk. Follow-up ended on postoperative day 3, and some values differ between text, tables, and figures.

4.7. Recommendations for future work

A multicenter registry of cardio-obstetric anesthesia, with prospective recording of hemodynamic targets, vasoactive drug doses, fluid balance, and shunt events in women with pulmonary hypertension and right heart failure, would show whether this combination improves outcome compared with other regimens.

5. Conclusion

This case report describes graded epidural anesthesia with norepinephrine and milrinone support, restricted fluids, and a dilute oxytocin infusion in a parturient with severe tricuspid regurgitation, high-probability pulmonary hypertension, lupus, and a patent foramen ovale with dynamic right-to-left shunting. Mother and infant survived. A transient post-delivery shunt hypoxemia was recognized and treated, and vasoactive support was weaned within 48 hours. The transferable lessons are to balance pulmonary against systemic vascular resistance instead of pursuing arbitrary blood pressure targets, to restrict fluids and uterotonic boluses, and to plan proactively as a multidisciplinary team. A single case cannot show that this approach caused the favorable course.

6. Declarations

Ethical approval and consent to participate. This case report was approved by the CMHC Research Center Ethics Committee, Indonesia, under reference number 2026/0418.

Consent for publication. Written informed consent for publication of this case and the accompanying images was obtained from the patient. All personal identifiers were removed and no facial features are displayed.

Availability of data and materials. The de-identified clinical datasets, serial arterial blood gas profiles, and echocardiographic recordings supporting the conclusions of this report are available from the corresponding author on reasonable request.

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

Author contribution statement. F.H.D. conceptualized the report, directed the intraoperative anesthetic and hemodynamic management, curated the clinical data, and wrote the original draft (Conceptualization; Investigation; Data curation; Writing – original draft). P.H.S.M. took part in the critical care management, performed the serial echocardiographic examinations, and critically revised the methods (Investigation; Formal analysis; Writing – review & editing). A.B. contributed to perioperative cardiovascular optimization, coordinated multidisciplinary cardio-obstetric planning, and refined the discussion (Project administration; Writing – review & editing). All authors approved the final version.

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

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