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Epidural Anesthesia and Staged Inodilator–Vasopressor Support for Emergency Cesarean Section in a Parturient with Severe Tricuspid Regurgitation, Pulmonary Hypertension, and Systemic Lupus Erythematosus: A Case Report

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A B S T R A C T

Introduction: Pregnancy imposes a progressive haemodynamic burden that can be lethal in women with fixed cardiac output states. The coexistence of severe tricuspid regurgitation, pulmonary hypertension and systemic lupus erythematosus (SLE) in a parturient is rare and carries a high risk of right ventricular decompensation, especially when an intracardiac right-to-left shunt is present.

Case Presentation: We describe a 30-year-old gravida 3 para 2 woman at 37+1 weeks of gestation who presented with antepartum vaginal bleeding and known SLE, severe tricuspid regurgitation (effective regurgitant orifice area 0.4 cm^2, vena contracta 0.7 cm), high-probability pulmonary hypertension (estimated systolic pulmonary artery pressure 74.7 mmHg) and a patent foramen ovale with right-to-left shunt. Emergency caesarean section was performed under graded epidural anaesthesia with 0.5% levobupivacaine. Intraoperative haemodynamics were supported with titrated infusions of norepinephrine, milrinone, dobutamine and furosemide under deliberate fluid restriction. A 2.56 kg male neonate was delivered with Apgar scores of 7, 8 and 9. Transient maternal hypotension and tachycardia responded to vasoactive titration; pharmacological support was staged down and discontinued by postoperative day 2, and the patient was transferred to the ward on day 3 with an improved tricuspid regurgitant velocity.

Conclusion: Graded epidural anaesthesia combined with a pathophysiology-driven, staged inodilator–vasopressor strategy and deliberate fluid restriction enabled a favourable maternal and neonatal outcome in a parturient with the tricuspid–pulmonary hypertension–lupus triad. Multidisciplinary planning and meticulous, lesion-specific haemodynamic monitoring were decisive.

1. Introduction

Cardiovascular disease has become a leading cause of maternal morbidity and mortality worldwide, and in many health systems it now outranks haemorrhage and sepsis as a driver of indirect maternal death.[1] The physiological adaptations of pregnancy—a 40–50% rise in blood volume and cardiac output, a fall in systemic vascular resistance, and a resting tachycardia—are well tolerated by the healthy heart but can precipitate decompensation in women with structural or pulmonary vascular disease. Contemporary practice therefore emphasises preconception risk stratification with validated tools such as the modified World Health Organization (mWHO) classification, the Cardiac Disease in Pregnancy (CARPREG II) score and the ZAHARA score; in a recent cohort of 333 pregnancies with cardiac

disease these instruments predicted cardiac events with an area under the curve approaching 0.75 and identified women who should be delivered in a tertiary centre with cardio-obstetric and anaesthetic expertise, while serial natriuretic-peptide testing further refined that risk.[1,2] Despite the existence of these instruments, counselling and risk communication remain inconsistently delivered; in one cohort of 1650 women of reproductive age attending cardiology clinics, preconception counselling was documented in only about one in ten, and a quarter were prescribed a potentially fetotoxic cardiovascular medication.[3]

Pulmonary hypertension (PH) occupies the highest-risk category of the mWHO system. Although pregnancy was historically considered contraindicated, contemporary nationwide analyses confirm that PH still carries a markedly elevated peripartum risk: in one analysis of nearly ten million deliveries, women with PH had an 85-fold higher in-hospital maternal mortality and far more frequent adverse cardiovascular events than those without PH,[4] and a second nationwide study found a composite of maternal cardiopulmonary morbidity and mortality in 41% of PH deliveries, with a nine-fold higher use of mechanical circulatory support.[5] These outcomes reflect the inability of the pressure-overloaded right ventricle to accommodate the acute increases in preload and cardiac output that accompany labour, delivery and the immediate postpartum autotransfusion. A single-centre cohort confirmed that maternal risk rises sharply with the severity of PH, poorer outcomes clustering in the moderate-to-severe group.[6] When PH complicates an autoimmune disease such as SLE, the anaesthetic challenge is compounded by the additional risks of vasculopathy, corticosteroid dependence and a prothrombotic tendency.[7]

Severe tricuspid regurgitation (TR) adds a further dimension of difficulty. Whether the valvular lesion is congenital or functional and secondary to right ventricular and annular dilatation, severe TR produces a volume-loaded, poorly compliant right ventricle that is exquisitely sensitive to both excessive preload and increases in pulmonary vascular resistance (PVR). Its prognostic weight is now well documented: in a cohort of patients with PH, severe TR was an independent predictor of mortality, with deaths roughly doubled compared with milder grades,[8] and large registry data confirm that outcomes in significant TR depend on the stage of right ventricular and end-organ disease at presentation.[9] In parallel, SLE is the autoimmune disease most likely to complicate pregnancy in women of reproductive age; a retrospective cohort of 420 SLE pregnancies found adverse outcomes were frequent and that a higher disease-activity (SLEDAI) score independently predicted low birth weight, preterm birth and caesarean delivery, while omission of thromboprophylaxis raised miscarriage risk.[10] Prospective data similarly identify unplanned pregnancy, disease flares and antiphospholipid syndrome as the dominant predictors of adverse maternal and fetal outcomes, underscoring the need for individualised, multidisciplinary planning.[11,12]

The selection of anaesthetic technique is pivotal in this population. General anaesthesia exposes the patient to the sympathetic surge of laryngoscopy, the pulmonary vasoconstrictive effects of hypoxaemia and hypercarbia, and the preload reduction imposed by positive-pressure ventilation, any of which can be catastrophic in a failing right ventricle. A contemporary review of cardiac disease in pregnancy concludes that, because maternal outcomes do not differ between general and regional techniques, incremental neuraxial anaesthesia should be preferred for caesarean delivery whenever it is not otherwise contraindicated.[13] Graded epidural anaesthesia, by contrast to single-shot spinal blockade, permits a slow, titratable onset of sympathetic blockade that preserves systemic vascular resistance and avoids abrupt cardiovascular collapse, and it obviates airway instrumentation and positive-pressure ventilation. A multidisciplinary model that integrates anaesthesiology, cardiology, obstetrics and intensive care has repeatedly been associated with the most favourable outcomes in complex cardiac parturients.[14]

An appreciation of right ventricular physiology explains why this constellation of disease is so dangerous in pregnancy. The right ventricle is a thin-walled, highly compliant chamber designed to eject against a low-resistance pulmonary circulation; it tolerates acute increases in afterload poorly and, once

it begins to dilate, enters a self-perpetuating spiral in which rising wall tension, falling coronary perfusion and worsening tricuspid regurgitation feed upon one another. Pregnancy loads precisely this vulnerable chamber, first through the gestational expansion of blood volume and cardiac output and then, abruptly, through the autotransfusion of 300 to 500 mL of uterine blood that accompanies delivery and uterine involution. In a woman with severe tricuspid regurgitation and pulmonary hypertension, any acute rise in pulmonary vascular resistance—provoked by pain, hypoxaemia, hypercarbia, acidosis or the Valsalva efforts of labour—can tip a compensated right ventricle into overt failure within minutes. The anaesthetic plan must therefore be constructed explicitly around the protection of right ventricular function rather than around the maintenance of a numerical blood-pressure target alone.[8]

To our knowledge, the simultaneous occurrence of severe tricuspid regurgitation, high-probability pulmonary hypertension and systemic lupus erythematosus, complicated by a patent foramen ovale with a demonstrable right-to-left shunt, has not previously been described in a parturient managed with pure epidural anaesthesia and a staged inodilator–vasopressor strategy. The novelty of this report lies in the deliberate, pathophysiology-driven combination of milrinone-based right ventricular afterload reduction with norepinephrine-based preservation of systemic and coronary perfusion, executed under graded neuraxial blockade and strict fluid restriction, and in the demonstration that this support could be withdrawn in a staged manner within 48 hours as pregnancy-related cardiovascular stress reversed. The aim of this case report is to describe the perioperative haemodynamic management of this rare comorbid triad and to distil transferable principles for the anaesthetic care of similar high-risk cardiac parturients.

2. Case Presentation

A 30-year-old woman, gravida 3 para 2, at 37+1 weeks of gestation presented to the emergency department with antepartum vaginal bleeding. She had an established diagnosis of systemic lupus erythematosus with low disease activity (SLEDAI score 4) and a complex history of right heart disease. Her cardiac medications before admission comprised sildenafil 60 mg/day and furosemide 40 mg/day; carvedilol, amlodipine and ivabradine had been discontinued earlier in pregnancy because of concerns regarding fetal safety. Her lupus had been maintained on chronic low-dose corticosteroid therapy with quiescent disease; dedicated antiphospholipid-antibody testing was not available in the perioperative record, a limitation returned to in the discussion of thromboembolic risk. Given the antepartum haemorrhage at term, an emergency caesarean section was indicated, and a combined cardio-obstetric, anaesthetic and intensive-care team was assembled to plan perioperative management.

Preoperative planning was conducted jointly by anaesthesiology, cardiology, obstetrics and intensive care. The team agreed in advance on the anaesthetic technique, the vasoactive strategy, the conduct of delivery and the postoperative destination, and defined explicit escalation pathways in the event of a pulmonary hypertensive crisis or right ventricular failure, including the availability of inhaled pulmonary vasodilators and a pre-briefed surgical team should mechanical circulatory support become necessary. The operating theatre was prepared with invasive monitoring, prepared vasoactive infusions and resuscitation equipment before the patient entered the room, and the obstetric team was briefed to deliver the uterotonic as a dilute, slow infusion rather than as a bolus. This anticipatory, protocolised approach reflects the consistent message of the cardio-obstetric literature that outcomes in the highest-risk parturients depend less on any single drug than on the coordination of the team around a shared, pathophysiology-driven plan.

Preoperative transthoracic and transoesophageal echocardiography documented severe tricuspid regurgitation with an effective regurgitant orifice area of 0.4 cm^2 and a vena contracta width of 0.7 cm, marked right atrial and right ventricular dilatation (basal right ventricular diameter 8.1 cm), and high-probability pulmonary hypertension with an estimated systolic pulmonary artery pressure of 74.7 mmHg and

a calculated pulmonary vascular resistance of 1.56 Wood units. Serial studies demonstrated a worsening tricuspid regurgitant velocity, reaching 4.4 m/s after a Valsalva manoeuvre, and confirmed a patent foramen ovale with right-to-left shunting that intensified under strain. The designation of high-probability pulmonary hypertension rested on a peak tricuspid regurgitant velocity above 3.4 m/s together with supportive secondary signs—marked right atrial and right ventricular dilatation and septal flattening—in keeping with contemporary echocardiographic criteria, with the corresponding pressure gradient derived later through the simplified Bernoulli relationship. The principal preoperative and echocardiographic findings are summarised in Table 1.

Table 1. Preoperative clinical, laboratory and echocardiographic findings.

Parameter	Value	Reference/interpretation
Age / obstetric status	30 y, G3P2, 37+1 weeks	Term, emergency presentation
Presenting complaint	Antepartum vaginal bleeding	Indication for emergency CS
SLE disease activity	SLEDAI 4	Low/mild activity
Baseline blood pressure	120/82 mmHg	Compensated
Baseline heart rate	**103 bpm**	Sinus tachycardia (normal <100)
SpO_2 (HFNC, FiO_2 0.30)	97%	Adequate oxygenation
Haemoglobin	**10.4 g/dL**	Mild anaemia
Leukocytes (admission)	**13.9 × 10^3/µL**	Leukocytosis
Albumin	**3.3 g/dL**	Hypoalbuminaemia
Tricuspid regurgitation	**Severe (EROA 0.4 cm^2, VC 0.7 cm)**	Volume-loaded right ventricle
TR velocity (post-Valsalva)	**4.4 m/s**	High-probability PH
Systolic PA pressure	**74.7 mmHg**	High-probability pulmonary hypertension
Pulmonary vascular resistance	1.56 Wood units	Pre-capillary component
Right ventricular diameter (RVD3)	**8.1 cm**	Severe RV dilatation
Interatrial septum	**Patent foramen ovale, right-to-left shunt**	Risk of paradoxical embolism / hypoxaemia
Chest radiograph	**Cardiomegaly, CTR 60%**	No acute pulmonary oedema

Notes: CS, caesarean section; CTR, cardiothoracic ratio; EROA, effective regurgitant orifice area; FiO_2, fraction of inspired oxygen; HFNC, high-flow nasal cannula; PA, pulmonary artery; PH, pulmonary hypertension; RV, right ventricular; SLE, systemic lupus erythematosus; SLEDAI, SLE Disease Activity Index; SpO_2, peripheral oxygen saturation; TR, tricuspid regurgitation; VC, vena contracta. Red values denote results outside the reference range.

On arrival in the operating theatre the patient was haemodynamically compensated, with a blood pressure of 120/82 mmHg, a heart rate of 103 bpm and an oxygen saturation of 97% on high-flow nasal cannula at an inspired oxygen fraction of 0.30. Standard monitoring was supplemented by invasive arterial pressure measurement. An epidural catheter was sited at 08:30 and anaesthesia was established with 0.5% levobupivacaine administered in graded increments, achieving a surgical sensory level without immediate cardiovascular compromise. Instantaneous monitor captures obtained at each critical perioperative stage are shown in Figure 1; because these represent point-in-time readings, they differ slightly from the charted baseline values. As shown in Figure 1A, the pre-induction tracing confirmed sinus

rhythm with full oxygen saturation, while Figure 1B documented the post-epidural state.

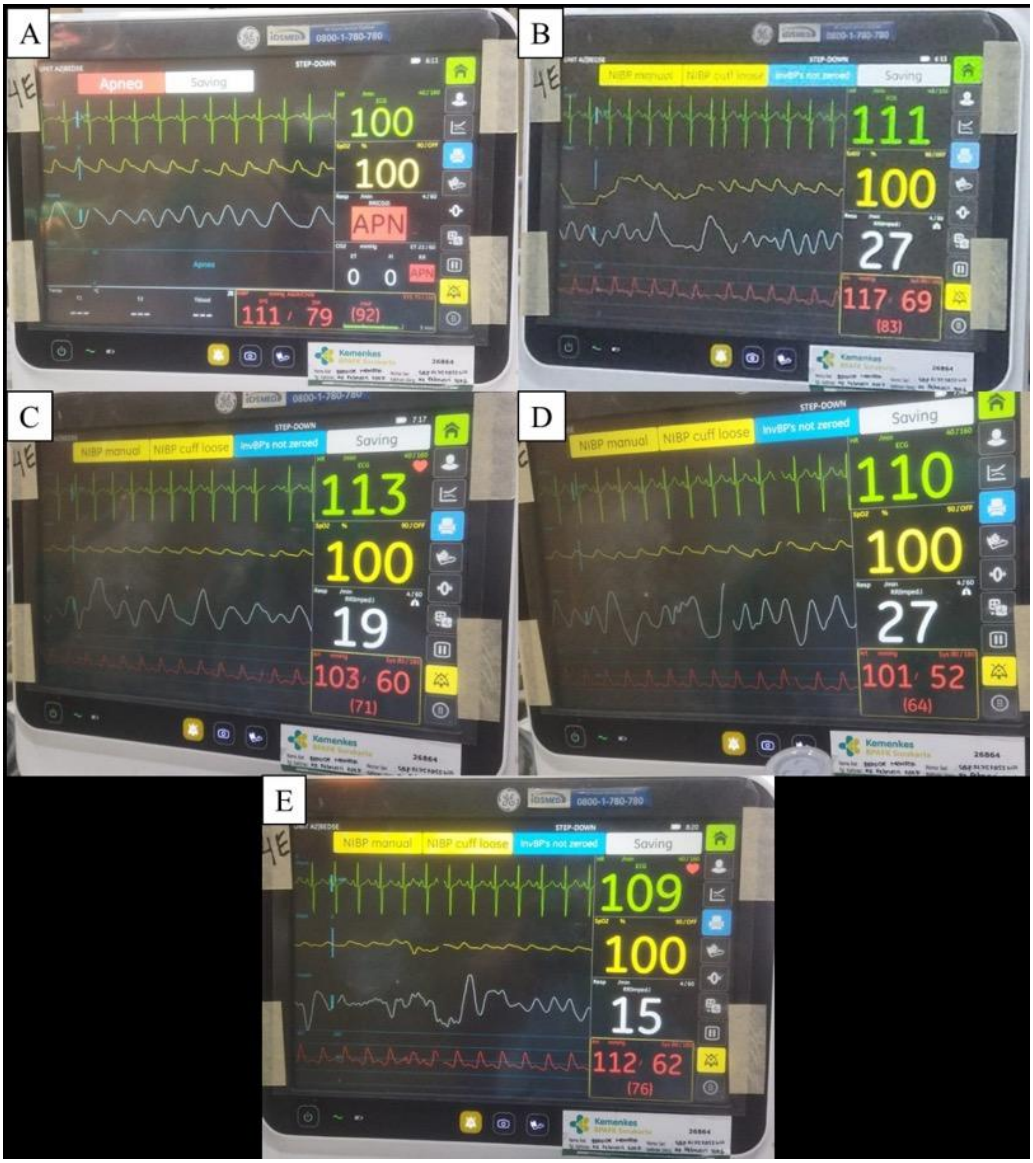

Figure 1. Sequential intraoperative and perioperative patient-monitor captures. (A) Pre-induction; (B) post-epidural; (C) during operation; (D) post-delivery; (E) postoperative recovery. Each panel displays the heart rate, peripheral oxygen saturation, respiratory rate and non-invasive blood pressure (with mean arterial pressure in parentheses) recorded at that stage.

The procedure lasted 90 minutes and required continuous vasoactive support throughout. A norepinephrine infusion (5.2 mL/h) was used to preserve systemic vascular resistance and coronary perfusion, milrinone (1 mL/h) was selected as an inodilator to augment right ventricular contractility while reducing pulmonary vascular resistance, dobutamine (2.4 mL/h) provided additional inotropic support, and low-dose furosemide (0.5 mL/h) was infused to prevent volume overload of the dilated right ventricle. The composition and rationale of this regimen are detailed in Table 2. Intraoperative blood pressure fluctuated between systolic values of 60 and 110 mmHg and diastolic values of 40 to 80 mmHg, with heart rates of 80 to 120 bpm; oxygen saturation was maintained at 97–100% via a non-rebreather mask at 10 L/min. Fluid administration was deliberately restrictive, totalling 500 mL of crystalloid against an estimated blood loss of 200 mL; the recorded net fluid balance, after accounting for urine output and insensible losses, was modestly positive at approximately 110 mL.

Table 2. Intraoperative vasoactive and adjunctive pharmacological regimen.			
Agent	**Pharmacological class**	**Infusion rate**	**Haemodynamic rationale**
Norepinephrine	α/β-adrenergic agonist	5.2 mL/h	Maintain SVR and coronary perfusion pressure without disproportionate rise in PVR
Milrinone	Phosphodiesterase-3 inhibitor (inodilator)	1 mL/h	Augment RV contractility and lower PVR; reduce functional TR severity
Dobutamine	β_1-adrenergic agonist (inotrope)	2.4 mL/h	Additional inotropic support for the volume-loaded right ventricle
Furosemide	Loop diuretic	0.5 mL/h	Prevent volume overload of the dilated, poorly compliant RV
Levobupivacaine 0.5% / 0.375%	Amide local anaesthetic	Graded epidural / 3 mL/h	Titratable sympathetic blockade and postoperative analgesia
Dexamethasone	Corticosteroid	5 mg intraoperative	Stress-dose coverage and modulation of systemic inflammation in SLE

Notes: PVR, pulmonary vascular resistance; RV, right ventricular; SVR, systemic vascular resistance; TR, tricuspid regurgitation.

Diagnostic imaging informed every step of perioperative planning. Chest radiography confirmed cardiomegaly with a cardiothoracic ratio of 60% but no acute pulmonary congestion (Figure 2), which reassured the team that the dyspnoea reflected chronic right heart disease rather than acute pulmonary oedema and supported a strategy of fluid restriction. Transthoracic echocardiography detailed the severity of the tricuspid lesion, while transoesophageal interrogation characterised the patent foramen ovale and demonstrated that the right-to-left shunt worsened under strain, directly informing decisions about positional management and the avoidance of manoeuvres that acutely elevate pulmonary vascular resistance.

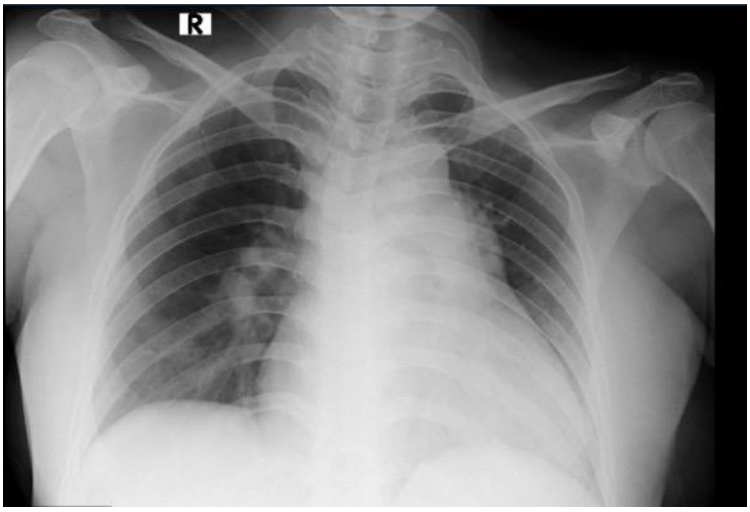

Figure 2. Preoperative posteroanterior chest radiograph demonstrating cardiomegaly with a cardiothoracic ratio of approximately 60% and clear lung fields, consistent with chronic right heart enlargement rather than acute pulmonary oedema.

A male neonate weighing 2.56 kg was delivered at 09:15 with Apgar scores of 7, 8 and 9 at one, five and ten minutes respectively, and was transferred to the routine nursery in stable condition. In keeping with the unit protocol for pulmonary hypertension, the uterotonic was administered as a dilute, slowly titrated oxytocin infusion rather than as a bolus, and ergometrine was avoided. Immediately after delivery

the mother showed a transient stabilisation (blood pressure 92/59 mmHg, heart rate 107–119 bpm) that required continued norepinephrine (3 mL/h) and milrinone (1 mL/h). Arterial blood gas analysis at this point revealed a metabolic acidosis (pH 7.295, base excess −11 mmol/L) with hypoxaemia (PaO_2 41 mmHg) reflecting transient right-to-left shunting; this corrected to a respiratory alkalosis (pH 7.465) following augmented oxygenation. The complete perioperative haemodynamic and oxygenation trajectory across all stages is presented graphically in Figure 3, which illustrates the controlled fall in mean arterial pressure to a nadir of approximately 70 mmHg during delivery (Figure 3A) and its progressive recovery, together with the corresponding heart-rate and saturation trends (Figure 3B).

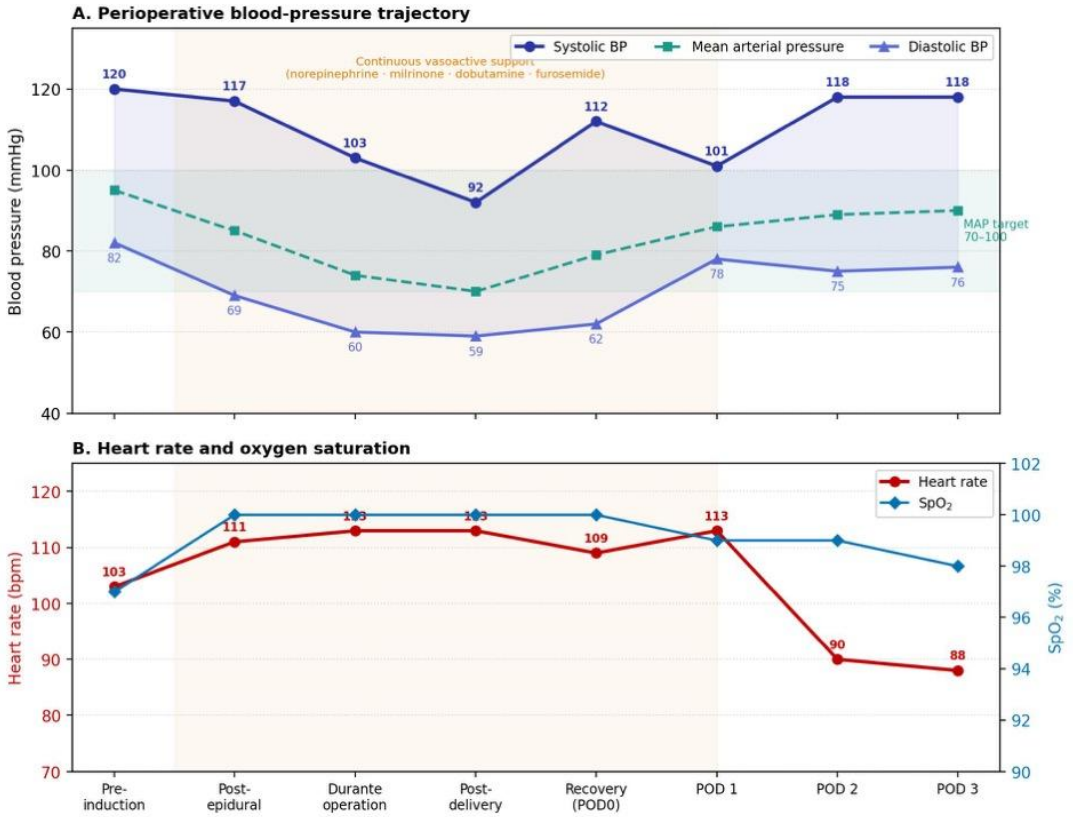

Figure 3. Perioperative haemodynamic trajectory reconstructed from the anaesthetic record. (A) Systolic, mean and diastolic blood pressure across the eight perioperative stages, with the mean arterial pressure target band (70–100 mmHg) shaded and the window of continuous vasoactive support indicated. (B) Heart rate and peripheral oxygen saturation over the same period. POD, postoperative day.

Postoperative care emphasised right-heart-protective strategies. The patient was nursed in a 30-degree head-up position to limit venous return, oxygen was titrated meticulously (target SpO_2 94–97%) to avoid hypoxic pulmonary vasoconstriction, and electrolytes and lactate were monitored every six hours to guide fluid and electrolyte management. Epidural analgesia with 0.375% levobupivacaine at 3 mL/h provided effective pain control without respiratory depression, and early mobilisation was encouraged to mitigate the thromboembolic risk conferred by both SLE and recent surgery. On the first postoperative day the vasoactive infusions were weaned: norepinephrine was tapered from 3 mL/h to discontinuation and dobutamine was stopped, while the blood pressure improved to 101/78 mmHg and oxygenation normalised (SpO_2 99% on 3 L/min nasal cannula). Laboratory trends were encouraging, with resolving leukocytosis (13.9 declining to 6.7 × 10^3/μL), although hypoalbuminaemia persisted at 3.3 g/dL and warranted continued nutritional support.

By the second postoperative day the cardiovascular parameters were self-sustaining, with blood pressure maintained at 110–125/70–80 mmHg and heart rate at 85–95 bpm. Adequate diuresis (greater than 0.5 mL/kg/h) permitted discontinuation of the furosemide infusion, reflecting improved right ventricular function and an optimised fluid balance. The patient was transferred to the ward on the third postoperative day with stable vital signs (blood pressure 118/76 mmHg, heart rate 88 bpm, SpO_2 98% on room air). Although echocardiography continued to show right heart dilatation, the tricuspid regurgitant velocity had improved to 3.8 m/s, suggesting partial reversal of the pregnancy-induced haemodynamic stress. The evolution of the principal haemodynamic and laboratory variables is summarised in Table 3.

Table 3. Perioperative haemodynamic and laboratory trends.

Parameter	Pre-induction	Intraoperative	POD 1	POD 2	POD 3
Systolic BP (mmHg)	120	60–110	101	110–125	118
Diastolic BP (mmHg)	82	40–80	78	70–80	76
Heart rate (bpm)	**103**	**80–120**	**113**	85–95	88
SpO_2 (%)	97	97–100	99	98–99	98
Leukocytes (×10³/μL)	**13.9**	—	**6.7**	Normal	Normal
Albumin (g/dL)	**3.3**	—	**3.3**	Low	Low
TR velocity (m/s)	**4.4**	—	—	—	**3.8**
Vasoactive support	Nil	NE+MIL+DOB+ FUR	NE taper, DOB off	FUR off	Nil

Notes: BP, blood pressure; DOB, dobutamine; FUR, furosemide; MIL, milrinone; NE, norepinephrine; POD, postoperative day; SpO_2, peripheral oxygen saturation; TR, tricuspid regurgitation. Em dash (—) denotes not measured at that time point. Red values denote results outside the reference range.

3. Discussion

This case report describes the successful use of graded epidural anaesthesia in a parturient with the rare triad of severe tricuspid regurgitation, high-probability pulmonary hypertension and systemic lupus erythematosus, further complicated by a patent foramen ovale with a right-to-left shunt. Although neuraxial anaesthesia is well established for obstetric surgery, its application in a patient with concurrent right heart dysfunction, pulmonary vascular disease and autoimmune disease has rarely been documented. The management deviated from several conventional approaches—most notably in the deliberate combination of an inodilator with a vasopressor, in the strict restriction of fluids, and in the staged, time-limited withdrawal of support—and these departures merit detailed examination.

Balancing pulmonary and systemic vascular resistance

The central haemodynamic objective in this patient was to support the right ventricle while preserving systemic and coronary perfusion, a balance that hinges on the ratio between systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR). The combination of norepinephrine, a mixed α/β-adrenergic agonist, with milrinone, a phosphodiesterase-3 inhibitor, was chosen precisely because it addresses both limbs of this equation: norepinephrine maintains SVR and coronary perfusion pressure with comparatively little effect on the pulmonary circulation, while milrinone augments right ventricular contractility and reduces right ventricular afterload through pulmonary vasodilatation. This strategy stands in deliberate contrast to the widespread reliance on phenylephrine for the correction of post-neuraxial hypotension during caesarean section. Phenylephrine, a pure α-agonist, raises systemic pressure reliably but does so at the cost of reflex bradycardia and a potential rise in pulmonary vascular tone, a combination that is poorly tolerated by a pressure- and volume-loaded right ventricle.[15]

The preservation of coronary perfusion deserves particular emphasis. The right coronary artery supplies the right ventricle during both systole and

diastole, and its perfusion pressure depends directly on the aortic root pressure exceeding right ventricular pressure. As the right ventricle dilates and its intracavitary pressure rises, the gradient driving its own blood supply narrows, so that any fall in systemic arterial pressure can provoke right ventricular ischaemia, a further loss of contractility and a downward spiral toward failure. By maintaining systemic vascular resistance and aortic root pressure, norepinephrine protects this fragile coronary gradient at the same time as milrinone reduces the afterload the ventricle must overcome. The two agents are therefore not merely additive but complementary, each compensating for the principal hazard of the other, and it is this physiological synergy—rather than the properties of either drug in isolation—that underpinned the stability achieved in this patient.[16]

A growing body of randomised evidence supports norepinephrine as the more physiologically favourable vasopressor in obstetric anaesthesia. Head-to-head trials have shown that norepinephrine is at least as effective as phenylephrine in preventing and correcting maternal hypotension while producing significantly less bradycardia, thereby better preserving maternal cardiac output.[15] Comparative studies of bolus administration have reached similar conclusions, demonstrating preserved heart rate and stable neonatal Apgar scores with norepinephrine.[17] A systematic review and meta-analysis of vasopressors used to treat, rather than merely to prevent, intraoperative hypotension in high-risk caesarean deliveries found a modest but consistent advantage for norepinephrine over phenylephrine, and for phenylephrine over ephedrine, in restoring blood pressure.[18] In preeclamptic women—another group in whom cardiac output is precarious—a systematic review found norepinephrine better preserved cardiac output and uteroplacental flow with less bradycardia than the alternatives.[19] The same advantage is amplified in twin pregnancy, where the gestational rise in cardiac output is greatest and a double-blind trial showed norepinephrine better maintained systolic pressure than phenylephrine without the attendant reflex bradycardia.[20] These data provide a coherent rationale for the vasopressor choice made in the present case, in which the maintenance of cardiac output and the avoidance of reflex bradycardia were paramount. It must be acknowledged, however, that these randomised data derive from low-, intermediate- and preeclamptic-risk parturients; no randomised vasopressor trial has been conducted specifically in women with severe pulmonary hypertension and a right-to-left shunt, so their application here is a mechanistic extrapolation rather than a trial-proven choice.

Selection of the inodilator

The decision to centre right ventricular support on milrinone reflects the particular pharmacology of the inodilator class. Experimental and clinical work comparing dobutamine, milrinone and levosimendan in pulmonary hypertension with right ventricular failure has shown that all three agents can improve right ventricular ejection fraction and ventriculo-arterial coupling, but they differ in their effects on pulmonary vascular resistance and heart rate. Dobutamine tends to increase heart rate and pulmonary artery pressure, whereas the phosphodiesterase inhibitors and calcium sensitisers more selectively unload the right ventricle. In a randomised trial in patients with pulmonary hypertension undergoing mitral valve replacement, both levosimendan and milrinone reduced pulmonary artery pressure, although milrinone was more frequently associated with systemic hypotension requiring norepinephrine—precisely the interaction anticipated and pre-empted in our patient by the concurrent norepinephrine infusion.[16] The same trial reaffirmed milrinone's value as an inodilator that relieves both right ventricular afterload and contractile failure, provided its systemic vasodilatory effect is matched by a vasopressor.[16] In the present case, the addition of low-dose dobutamine provided supplementary inotropy without committing the patient to the tachycardia that a higher dobutamine dose would have produced, and the milrinone-norepinephrine pairing delivered the desired reduction in right ventricular afterload while protecting systemic pressure.

Fluid management and uterotonic strategy

Fluid management represented a second deliberate departure from routine obstetric practice. Restricting

crystalloid administration to 500 mL aligned with the established principle that a dilated, poorly compliant right ventricle tolerates volume loading badly, and that even modest fluid excess can precipitate right ventricular failure and functional worsening of tricuspid regurgitation. This approach contrasts with the modest preloading sometimes advocated for fixed left-sided obstructive lesions, and the divergence underscores the need for lesion-specific, rather than generic, fluid strategies when several pathologies coexist. The chest radiograph, which showed cardiomegaly without pulmonary congestion, reinforced the decision to keep the patient on the dry side of euvolaemia. The clinical relevance of subtle volume and pressure shifts in high-risk pregnancy is increasingly recognised; echocardiographic findings such as a new pericardial effusion in the third trimester have been proposed as markers of impending preeclampsia and peripartum heart failure, lending support to a strategy of vigilant volume surveillance in this population.[21]

The choice and manner of uterotonic administration is a further critical consideration in PH. Bolus oxytocin causes systemic vasodilatation, reflex tachycardia and a transient rise in pulmonary artery pressure. Randomised trials in caesarean delivery confirm this haemodynamic penalty directly: compared with the longer-acting analogue carbetocin, oxytocin produced significantly greater falls in blood pressure and rises in heart rate in preeclamptic parturients,[22] and in high-risk caesarean sections carbetocin maintained a higher mean arterial pressure and lower heart rate than oxytocin.[23] For this reason, uterotonics in patients with pulmonary hypertension should be given as a dilute, slow infusion rather than a bolus, with continuous haemodynamic monitoring, and ergometrine, which raises pulmonary vascular resistance, is avoided. These randomised comparisons were, it should be noted, performed in preeclamptic and general high-risk populations rather than in pulmonary hypertension specifically, so they inform principle rather than provide direct evidence in this lesion. The restrained approach to uterotonic and fluid therapy in our patient is consistent with the strategies reported in prospective cohorts of pulmonary arterial hypertension in pregnancy, in which avoidance of oxytocin boluses formed part of a deliberate protocol.[14]

Neuraxial technique and choice of local anaesthetic

The selection of graded epidural anaesthesia, in preference to single-shot spinal or combined spinal-epidural techniques, was driven by the need for haemodynamic stability. A single-shot spinal produces a rapid, profound sympathetic blockade that can precipitate catastrophic hypotension in a patient unable to compensate by increasing cardiac output, whereas an epidural catheter allows the block to be established slowly and titrated to effect, preserving systemic vascular resistance.[13] The avoidance of general anaesthesia also eliminated the risks of laryngoscopy-induced sympathetic surges, hypercarbia and the preload reduction of positive-pressure ventilation, each of which is hazardous in right ventricular failure. The literature on cardiac parturients consistently favours such modifiable, graded neuraxial techniques, and low-dose, diluted local anaesthetic regimens have been shown to provide adequate surgical conditions while limiting the incidence and severity of maternal hypotension.[24]

It is worth making explicit why general anaesthesia is so specifically hazardous in this physiology, since the decision to avoid it was central to the plan. Direct laryngoscopy and tracheal intubation provoke an intense sympathetic response that acutely raises both systemic and pulmonary arterial pressures and can precipitate a pulmonary hypertensive crisis. Positive-pressure ventilation raises intrathoracic pressure and reduces venous return, diminishing the preload of a right ventricle that is preload-dependent for its output, while any period of hypoventilation, hypoxaemia or hypercarbia on induction or emergence elevates pulmonary vascular resistance precisely when the ventricle can least afford it. Volatile and intravenous induction agents depress myocardial contractility and vasomotor tone, and emergence carries the additional risks of coughing, straining and the Valsalva effect, each of which can acutely worsen a right-to-left shunt. Graded epidural anaesthesia sidesteps this entire sequence of threats, which is why it, rather than

general anaesthesia, is repeatedly favoured for fixed-output states in the cardio-obstetric literature.[13]

Levobupivacaine, the S-enantiomer of bupivacaine, was an appropriate agent for this purpose because of its favourable cardiotoxicity profile relative to racemic bupivacaine, an important consideration in a patient with limited cardiovascular reserve. Comparative meta-analytic data indicate that levobupivacaine and ropivacaine offer broadly equivalent maternal and fetal outcomes when used for neuraxial obstetric anaesthesia and analgesia, with no major differences in the risk of hypotension or in neonatal condition, allowing the choice of agent to be guided by availability and familiarity.[25] Nevertheless, neuraxial techniques demand careful dosing and vigilant neurological monitoring; observational data show that low-dose, diluted local-anaesthetic regimens deliver adequate surgical conditions while attenuating the incidence and severity of maternal hypotension, an attribute of particular value when cardiovascular reserve is limited.[24] In the present case, the use of an indwelling epidural catheter for both intraoperative anaesthesia and postoperative analgesia provided continuity of sympathetic control during the vulnerable early postpartum period.

Systemic lupus erythematosus and the prothrombotic milieu

The patient's systemic lupus erythematosus introduced several layers of complexity. Although her disease activity was low (SLEDAI 4) and overt lupus nephritis was absent, SLE in pregnancy is associated with an elevated risk of flare, pre-eclampsia, thrombosis and adverse fetal outcomes, and these risks are highest in women with active disease or renal involvement.[10] Chronic corticosteroid exposure mandated perioperative stress-dose coverage, provided here as intraoperative dexamethasone, both to prevent relative adrenal insufficiency during surgical stress and to modulate systemic inflammation. The combination of SLE-related hypercoagulability and the venous stasis of recent surgery created a substantial thromboembolic risk, which was particularly dangerous given the right-to-left shunt across the patent foramen ovale and the consequent potential for paradoxical embolism. Early mobilisation and meticulous attention to haemostasis were therefore prioritised. Cohort data in SLE pregnancy reinforce this stance, showing that omission of thromboprophylaxis—particularly low-molecular-weight heparin—is associated with a significantly higher risk of fetal loss, while antiphospholipid syndrome independently predicts adverse fetal outcome, so that prophylaxis must be weighed individually against the bleeding risk.[11]

The thromboembolic dimension of this case warranted explicit attention. Systemic lupus erythematosus, particularly when accompanied by antiphospholipid antibodies, confers a prothrombotic state that compounds the physiological hypercoagulability of pregnancy and the venous stasis of surgery and bed rest. In a patient with a right-to-left interatrial shunt, a venous thrombus carries the additional and grave possibility of paradoxical systemic or cerebral embolism. Management therefore balanced the competing risks of thrombosis and haemorrhage—the latter immediately relevant given the antepartum bleeding that prompted delivery—through early mobilisation, mechanical thromboprophylaxis and careful, individualised decisions about the timing and intensity of pharmacological anticoagulation in conjunction with the haematology and obstetric teams. The principle that anticoagulation must be tailored to the individual confluence of bleeding and clotting risks, rather than applied by protocol, is central to the safe care of the pregnant woman with lupus and structural heart disease.[10]

The patent foramen ovale and right-to-left shunting

The quantitative echocardiographic indices recorded in this patient deserve interpretation, because they anchored the assessment of severity. An effective regurgitant orifice area of 0.4 cm^2 and a vena contracta width of 0.7 cm both lie well within the range that defines severe tricuspid regurgitation, and a basal right ventricular diameter of 8.1 cm denotes gross chamber dilatation. The resting study, assuming a right atrial pressure of approximately 5 mmHg, yielded the estimated systolic pulmonary artery pressure of 74.7 mmHg that placed this patient in the high-

probability pulmonary hypertension category, while the rise in tricuspid regurgitant velocity to 4.4 m/s on Valsalva—corresponding through the simplified Bernoulli relationship to a right-ventricular-to-right-atrial gradient of roughly 77 mmHg—demonstrated the dynamic, provocable nature of her pulmonary hypertension. The improvement of that velocity to 3.8 m/s by the third postoperative day, equating to a fall of roughly 13 mmHg in the estimated gradient, provided objective evidence that the haemodynamic burden of pregnancy was already reversing. Such serial echocardiographic surveillance is increasingly advocated in high-risk pregnancy, where evolving structural findings carry prognostic weight.[21]

The presence of a patent foramen ovale with demonstrable right-to-left shunting was a defining feature of this case and shaped several management decisions. As right atrial pressure rises with worsening tricuspid regurgitation and pulmonary hypertension, shunting across the foramen ovale increases, producing systemic hypoxaemia—as reflected in the transient post-delivery arterial oxygen tension of 41 mmHg—and exposing the patient to paradoxical embolism. Any manoeuvre that acutely raises pulmonary vascular resistance, including hypoxaemia, hypercarbia, acidosis, pain and a Valsalva effect, can intensify the shunt, which is why the strategy combined scrupulous oxygenation, avoidance of hypercarbia, effective neuraxial analgesia and milrinone-mediated reduction of pulmonary vascular tone. Transoesophageal echocardiography was invaluable in confirming that the shunt remained stable during the procedure, providing real-time reassurance that the low-dose epidural technique was not aggravating intracardiac shunting and supporting the safety of the chosen approach.[13]

Risk stratification and comparative outcomes

Formal risk stratification frames the magnitude of the challenge this patient represented. Severe pulmonary hypertension places a parturient in the highest mWHO category, in which pregnancy is conventionally considered contraindicated and maternal mortality is high; comparative cohort studies of the mWHO, CARPREG II and ZAHARA instruments confirm their value in predicting cardiac events and in directing such women to tertiary, multidisciplinary care.[1] Earlier engagement through preconception counselling, which remains underutilised, might have allowed disease optimisation and informed decision-making before conception.[3] Risk is, moreover, graded and lesion-specific rather than uniform: both biomarker trajectories during pregnancy and the structural stage of the right heart at presentation independently inform prognosis, underscoring that anaesthetic strategy cannot be generic but must follow the underlying pathophysiology.[2,9] Table 4 places the present case alongside other complex cardiac parturients, highlighting the distinctive features of its management and outcome.

Table 4. Comparative management and outcomes in complex cardiac parturients undergoing caesarean section.

Parameter	Present case	Eisenmenger / group 1 PH	Dilated cardiomyopathy
Dominant lesion	Severe TR + high-probability PH + PFO	Fixed PH with shunt reversal	LV systolic dysfunction
Anaesthetic technique	Graded epidural	Combined spinal-epidural / epidural	General or combined spinal-epidural
Principal vasoactive	Norepinephrine + milrinone + dobutamine	Phenylephrine/iloprost	Inotrope ± vasopressor
Fluid strategy	Restrictive (500 mL)	Restrictive	Variable
Systolic BP range (mmHg)	60–110	80–120	90–130
Maternal outcome	Favourable; support withdrawn by POD 2	High mortality (30–50%)	Risk of postpartum heart failure
Postpartum complication	None	Pulmonary hypertensive crisis	Heart failure

Notes: BP, blood pressure; LV, left ventricular; PFO, patent foramen ovale; PH, pulmonary hypertension; POD, postoperative day; TR, tricuspid regurgitation. Comparative columns summarise patterns reported in the cited literature and are illustrative rather than derived from a matched cohort.

The comparison in Table 4 illustrates several points. Unlike many reported Eisenmenger and group 1 pulmonary hypertension cases, in which combined spinal-epidural anaesthesia and pure α-agonist vasopressors predominate, the present patient achieved haemodynamic stability with epidural anaesthesia alone and a balanced inodilator–vasopressor regimen, avoiding both the cerebrospinal-fluid shift of a dural puncture and the pulmonary vasoconstriction of unopposed α-stimulation. The absence of any postpartum cardiac complication contrasts favourably with the heart-failure rates reported among parturients with dilated cardiomyopathy and with the high mortality of untreated severe pulmonary hypertension, and it lends practical support to the multidisciplinary, monitoring-intensive model advocated throughout the cardio-obstetric literature.[4,14]

The timing and mode of delivery were themselves therapeutic decisions. In a parturient with fixed-output cardiac disease, the goal is to avoid both the prolonged haemodynamic stress of labour and the sudden swings of an unplanned emergency, and a planned caesarean section under graded neuraxial anaesthesia at or near term often represents the most controlled option. In this patient the antepartum haemorrhage at 37+1 weeks converted an anticipated plan into an emergency, yet the prior multidisciplinary preparation meant that the team could proceed without improvisation. Reaching term also benefited the neonate, sparing it the morbidity of prematurity, and allowed delivery before any further deterioration in maternal right ventricular function. The case thus illustrates the broader lesson that, in high-risk cardiac pregnancy, contingency planning for emergency delivery should be embedded in the care pathway from the moment of booking, since the margin for reactive decision-making at the time of crisis is narrow.[5]

Monitoring and the staged-withdrawal paradigm

A distinctive and instructive feature of this case was the speed and orderliness of the recovery. The requirement for vasoactive support fell progressively from the intraoperative period through the first postoperative day and was fully withdrawn by the second, a trajectory captured in Figure 3 and Table 3. This pattern reflects the rapid reversal of the cardiovascular stresses unique to pregnancy—the relief of aortocaval compression, the diuresis of the extracellular fluid accumulated during gestation, and the fall in cardiac output and heart rate toward pre-pregnancy values—once delivery has occurred. The improvement in tricuspid regurgitant velocity from 4.4 to 3.8 m/s over three days provides objective echocardiographic evidence of this reversal. The case therefore demonstrates that, in an optimised critical-care environment, even a parturient with severe right heart disease can be expected to improve quickly after delivery, and that vasoactive support should be actively de-escalated rather than continued by default. The peripartum period nonetheless remains the time of greatest danger, and the literature on cardiogenic shock and mechanical circulatory support in pregnancy is a reminder that escalation pathways, including extracorporeal support, must be planned in advance even when—as here—they prove unnecessary; nationwide data show mechanical circulatory support is used nine times more often in PH deliveries than in unaffected ones.[5]

Continuous invasive monitoring underpinned every one of these decisions. Invasive arterial pressure measurement allowed beat-to-beat titration of the vasoactive infusions, serial arterial blood gases tracked the shunt fraction and acid-base status, and transoesophageal echocardiography provided real-time assessment of right ventricular function and shunt dynamics. The value of pre-delivery echocardiography and invasive monitoring in this population is well documented, and cohort data in obstetric pulmonary hypertension reinforce that management in high-acuity, multidisciplinary centres with such monitoring is associated with better outcomes.[7] Where available, preoperative right heart catheterisation can further refine the characterisation of pulmonary vascular resistance and guide the precise selection and dosing of vasoactive agents, and it would be a reasonable addition to the work-up of comparable future patients.

Postoperative intensive care and analgesia

The postoperative course was managed in a critical-care environment with the same physiological

priorities that had governed the intraoperative period. Nursing the patient in a 30-degree head-up position reduced venous return to the overloaded right ventricle, while careful oxygen titration to a saturation of 94–97%—deliberately lower than the 97–100% maintained intra-operatively, because once the acute shunt stress of delivery had passed excessive inspired oxygen offered no benefit—avoided both the hypoxic pulmonary vasoconstriction that accompanies under-oxygenation and the absorption atelectasis and potential hyperoxic vasoconstriction associated with excessive inspired oxygen. Six-hourly measurement of electrolytes and lactate allowed early detection of hypoperfusion and guided a conservative approach to fluid and electrolyte replacement. Effective analgesia is not merely a comfort measure in this setting but a haemodynamic intervention: uncontrolled pain elevates sympathetic tone and pulmonary vascular resistance and can precipitate decompensation. The indwelling epidural catheter, used postoperatively with 0.375% levobupivacaine at 3 mL/h, delivered continuous segmental analgesia without the respiratory depression or sympatholytic unpredictability of systemic opioids, and it preserved the ability to mobilise the patient early, an important consideration given the combined thromboembolic risk of systemic lupus erythematosus and recent surgery.[24]

The staged weaning of vasoactive support was conducted against continuous haemodynamic feedback rather than to a fixed schedule. Norepinephrine was reduced only once the mean arterial pressure and urine output confirmed adequate perfusion, dobutamine was discontinued as the heart rate and contractility allowed, and the furosemide infusion was stopped when spontaneous diuresis exceeded 0.5 mL/kg/h, signalling that the right ventricle was handling its volume load without pharmacological assistance. This feedback-driven de-escalation minimised the risk of rebound hypotension or fluid overload during the transition off support and exemplifies the principle that, in the recovering cardiac parturient, the discontinuation of therapy demands as much vigilance as its initiation.[16]

Neonatal considerations and a practical framework

The neonatal outcome in this case was reassuring, with a birthweight of 2.56 kg and Apgar scores of 7, 8 and 9, reflecting both the preservation of maternal cardiac output and uteroplacental perfusion throughout the procedure and the avoidance of agents likely to depress the neonate. The choice of vasopressor is relevant to the fetus as well as the mother: norepinephrine has been shown to preserve uteroplacental blood flow and neonatal acid-base status at least as well as phenylephrine, and considerably better than ephedrine, which crosses the placenta and can produce fetal acidaemia.[19] The graded epidural technique avoided the neonatal exposure to induction agents and the risk of difficult maternal airway management inherent in general anaesthesia, and it allowed the mother to remain awake and to bond with her infant in the immediate postpartum period.

Drawing these threads together yields a practical framework for the anaesthetic management of the parturient with severe right heart disease. The principles are, first, to choose a graded, titratable neuraxial technique in preference to single-shot spinal or general anaesthesia wherever the clinical situation permits; second, to support the circulation by explicitly balancing pulmonary against systemic vascular resistance, typically by pairing an inodilator such as milrinone with a vasopressor such as norepinephrine, rather than by reaching reflexively for a pure α-agonist; third, to restrict fluids and to administer uterotonics only as a dilute, slow infusion; fourth, to monitor invasively and echocardiographically so that every intervention is guided by real-time physiology; and fifth, to plan the team, the destination and the escalation pathways in advance.[25,26] The favourable course of the present patient, in whom support could be withdrawn within forty-eight hours, suggests that adherence to these principles can, as in this patient, convert a situation of formidable predicted risk into a manageable one, although the contemporary cohorts cited still report substantial residual maternal mortality and this favourable single-case experience should not be over-generalised.[1,13]

Limitations

Several limitations of this report should be acknowledged. As a single case, it cannot establish the superiority of any particular technique, and the favourable outcome may not be reproducible in patients with higher disease activity, decompensated right ventricular failure or active lupus nephritis. The pulmonary hypertension was characterised by echocardiography and designated high-probability rather than confirmed by right heart catheterisation, so the absolute pulmonary pressures and resistance should be interpreted with appropriate caution. There remains a theoretical risk that epidural volume extension could alter intracardiac shunting through the patent foramen ovale, although continuous transoesophageal echocardiography confirmed stable shunt dynamics throughout and supported the safety of the low-dose levobupivacaine technique. Antiphospholipid-antibody testing was not available in the perioperative record, which constrains the interpretation of thrombotic risk and of the paradoxical-embolism hazard posed by the right-to-left shunt. Finally, subclinical lupus inflammation may have influenced the haemodynamic course in ways that cannot be fully quantified. Despite these caveats, the case offers a coherent, mechanistically grounded account of how a rare and dangerous combination of pathologies can be managed to a favourable conclusion.

4. Conclusion

This case demonstrates that graded epidural anaesthesia, coupled with a pathophysiology-driven haemodynamic strategy, can be a viable and safe approach for a high-risk parturient with the uncommon combination of severe tricuspid regurgitation, high-probability pulmonary hypertension and systemic lupus erythematosus complicated by a patent foramen ovale. The deliberate pairing of milrinone-based right ventricular afterload reduction with norepinephrine-based preservation of systemic perfusion, executed under strict fluid restriction and continuous invasive and echocardiographic monitoring, achieved haemodynamic stability through delivery and allowed the support to be withdrawn in a staged manner within forty-eight hours as the cardiovascular stresses of pregnancy resolved. Both mother and neonate had favourable outcomes. The key transferable lessons are the primacy of balancing pulmonary against systemic vascular resistance rather than simply chasing a blood-pressure target, the importance of lesion-specific fluid and uterotonic strategies, and the need for anticipatory multidisciplinary planning with predefined escalation pathways. Future work should aim to standardise vasoactive and neuraxial protocols for parturients in whom cardiac and autoimmune disease overlap, and to extend outcome tracking beyond the immediate peripartum period.

Declarations

Ethics approval and consent to participate

All procedures were performed in accordance with institutional and national ethical standards and with the principles of the Declaration of Helsinki.

Consent for publication

Written informed consent was obtained from the patient for the publication of this case report and the accompanying images. Identifying details have been minimised and no facial or identifying features are shown.

Availability of data and materials

The clinical data supporting the findings of this case report are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

All authors contributed to the perioperative care of the patient, the conception of the report, the drafting and critical revision of the manuscript, and approved the final version.

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5. References

1. Poungsuntorn P, Suwanrath C, Chainarong N, et al. Prediction of cardiac events in pregnant women with cardiac diseases using modified WHO, CARPREG II, and ZAHARA risk assessments in southern Thailand. Int J Gynaecol Obstet. 2025;171(1):281-287. doi:10.1002/ijgo.70196
2. Chang SA, Khakh P, Janzen M, et al. Trending cardiac biomarkers during pregnancy in women with cardiovascular disease. Circ Heart Fail. 2022;15(8):e009018. doi:10.1161/CIRCHEARTFAILURE.121.009018
3. Osmanska J, Jackson AM, Simpson J, et al. Preconception counselling in women of reproductive age attending cardiology clinics in Scotland. Heart. 2024;110(13):908-915. doi:10.1136/heartjnl-2023-323666
4. Maligireddy A, Jabri A, Zghouzi M, et al. Maternal and fetal outcomes in pulmonary hypertension during pregnancy: a contemporary nationwide analysis. Am J Cardiol. 2024;221:113-119. doi:10.1016/j.amjcard.2024.04.016
5. Potnuru PP, Jefferies H, Lei R, et al. Maternal pulmonary hypertension and cardiopulmonary outcomes during delivery hospitalization in the United States: a nationwide study from 2016-2020. Pregnancy Hypertens. 2024;38:101170. doi:10.1016/j.preghy.2024.101170
6. Chen G, Zhang Z, Zhao X, et al. Maternal and neonatal outcomes in pregnancies complicated with pulmonary hypertension: a retrospective cohort study. Eur J Med Res. 2025;30(1):717. doi:10.1186/s40001-025-02987-5
7. McNeil AL, Al-Shibli NK, Fuller ME, et al. Pregnancy-related outcomes in obstetric patients with pulmonary hypertension: a single-center retrospective cohort study. Int J Obstet Anesth. 2024;57:103964. doi:10.1016/j.ijoa.2023.103964
8. Albulushi A, Al-Saidi K, Al-Farhan H. Impact of tricuspid regurgitation severity on mortality in pulmonary hypertension patients: a comprehensive analysis. Curr Probl Cardiol. 2024;49(4):102405. doi:10.1016/j.cpcardiol.2024.102405
9. Schlotter F, Stolz L, Kresoja KP, et al. Tricuspid regurgitation disease stages and treatment outcomes after transcatheter tricuspid valve repair. JACC Cardiovasc Interv. 2025;18(3):339-348. doi:10.1016/j.jcin.2024.10.034
10. Xu Y, Fang Y, Xu H, et al. Predictors of adverse pregnancy outcomes in systemic lupus erythematosus: a retrospective cohort study. Clin Rheumatol. 2025;45(2):1279-1289. doi:10.1007/s10067-025-07882-2
11. Mokbel A, Attia DH, Zayed HS, et al. Pregnancy outcomes among Egyptian women with systemic lupus erythematosus: a prospective cohort study. Lupus. 2023;32(4):521-530. doi:10.1177/09612033231159468
12. Vicoveanu P, Vasilache IA, Nemescu D, et al. Predictors associated with adverse pregnancy outcomes in a cohort of women with systemic lupus erythematosus from Romania - an observational study (stage 2). J Clin Med. 2022;11(7):1964. doi:10.3390/jcm11071964
13. Rex S, Devroe S. Cardiac disease in pregnancy. Best Pract Res Clin Anaesthesiol. 2022;36(1):191-208. doi:10.1016/j.bpa.2022.02.005
14. Jha N, Divya MB, Jha AK. Management and outcomes of pulmonary artery hypertension and Eisenmenger syndrome during pregnancy: a prospective observational cohort study. BJOG. 2023;130(10):1258-1268. doi:10.1111/1471-0528.17474
15. Banihashem N, Minagar M, Shirafkan H, et al. Effect of norepinephrine and phenylephrine on maternal hemodynamic changes after spinal anesthesia in cesarean section: a randomized clinical trial. Caspian J Intern Med. 2026;17(1):67-73. doi:10.22088/cjim.17.1.67
16. Virmani S, Garg S, Malik I, et al. Intravenous levosimendan versus milrinone: coronary sinus lactate and PA catheter derived parameters in patients with pulmonary hypertension undergoing elective mitral valve replacement. Ann Card Anaesth. 2025;28(3):298-304.

doi:10.4103/aca.aca_4_25
17. Ansari DJ, Chaurasiya MK, Singh M, et al. Intravenous bolus phenylephrine and intravenous bolus norepinephrine for treatment of maternal hypotension in spinal anesthesia during cesarean section: a prospective, randomized, comparative study. Cureus. 2026;18(2):e103343. doi:10.7759/cureus.103343
18. Babul A, Ashraf S, Desai J, et al. Intraoperative hypotension after neuraxial anesthesia: a systematic review and meta-analysis of randomized controlled trials of vasopressors for cesarean birth stratified by maternal risk. Cureus. 2025;17(10):e94900. doi:10.7759/cureus.94900
19. Gunawan MFB, Soetomo CT, Richard R, et al. Dissecting vasopressor efficacy in the management of maternal hypotension in preeclamptic cesarean delivery: a systematic review of randomized controlled trials. Matern Fetal Med. 2025;7(4):234-243. doi:10.1097/FM9.0000000000000314
20. Chen Z, Zhou J, Wan L, et al. Norepinephrine versus phenylephrine infusion for preventing postspinal hypotension during cesarean section for twin pregnancy: a double-blinded randomized controlled clinical trial. BMC Anesthesiol. 2022;22(1):17. doi:10.1186/s12871-022-01562-3
21. Kim H, Bae JG, Lee HJ, et al. The implication of pericardial effusion in the third trimester for preeclampsia and heart failure in high-risk pregnant women. J Echocardiogr. 2025;23(3):180-187. doi:10.1007/s12574-024-00679-3
22. Bahr MH, Alkhatip A, Ahmed AG, et al. Hemodynamic effects of oxytocin and carbetocin during elective cesarean section in preeclamptic patients under spinal anesthesia: a randomized double-blind controlled study. Anesth Pain Med. 2023;13(1):e128782. doi:10.5812/aapm-128782
23. Oza V, Rawal K, Parmar V, et al. Comparison of the effectiveness, safety, and hemodynamic profile of prophylactic administration of carbetocin vs. oxytocin for the prevention of postpartum hemorrhage in high-risk cesarean sections: a randomized, double-blind clinical trial. Cureus. 2025;17(8):e89968. doi:10.7759/cureus.89968
24. Suzuki M, Sato C, Nishii H, et al. The effects of the use of diluted bupivacaine in sequential combined spinal and epidural anesthesia for cesarean delivery on maternal hypotension and motor block after surgery: a retrospective observational study. J Nippon Med Sch. 2022;89(5):533-539. doi:10.1272/jnms.JNMS.2022_89-514
25. Li Z, Zhou X, Wang H. The impact of epidural ropivacaine versus levobupivacaine for labor analgesia on maternal and fetal outcomes: a meta-analysis. BMC Anesthesiol. 2024;24(1):378. doi:10.1186/s12871-024-02767-4
26. Atalia A, Cresma AC, Athallah AF, et al. Regional anesthesia vs opioid therapy for postoperative pain management in cardiac surgery: A systematic review. Solo J Anesth Pain Critical Care. 2025;5(1):43. doi: 10.20961/soja.v5i1.91746