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Precision Anesthetic Management of the Triple-Pathology Parturient: Graded Epidural Technique for Emergency Cesarean Section in Severe Tricuspid Regurgitation, Pulmonary Hypertension, and Systemic Neurofibromatosis

Purwoko[1], Fitri Hapsari Dewi[1], Helmi Ananta[2*]

[1]Staff of Anesthesiology and Intensive Therapy Department, Dr. Moewardi Regional General Hospital/Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

[2]Resident of Anesthesiology and Intensive Therapy Department, Dr. Moewardi Regional General Hospital/Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

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***Corresponding author:**
Helmi Ananta

E-mail address:
helmot369@gmail.com

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A B S T R A C T

Introduction: Maternal cardiac disease, specifically right-sided valvular lesions exacerbated by pulmonary hypertension, remains a primary driver of maternal mortality. The physiological demands of pregnancy act as a cardiovascular stress test, often leading to decompensation in patients with underlying pathology. This case describes the management of a triple-pathology parturient. **Case presentation:** A 37-year-old female (G2P1A0) at 34 weeks' gestation presented with NYHA Class IV symptoms, including progressive dyspnea and orthopnea. Echocardiography revealed severe tricuspid regurgitation (regurgitant volume 112 mL), right ventricular dilatation, and a high probability of pulmonary hypertension with a mean pulmonary arterial pressure of 50.39 mmHg and a systolic pulmonary arterial pressure of 79.32 mmHg. Systemic neurofibromatosis added concerns regarding neuraxial anatomy and airway management. An emergency Cesarean Section was performed under a graded epidural technique using 0.375 percent Levobupivacaine and 50 mcg Fentanyl, administered in 3 mL increments every 5 minutes. Hemodynamic stability was maintained through strict fluid restriction of 300 mL and titrated vasopressors. **Conclusion:** A carefully titrated graded epidural provides superior stability in the hostile hemodynamics of right heart failure by allowing a slow, compensatory sympathetic blockade. Early multidisciplinary coordination is essential for success in complex cardio-obstetric cases.

1. Introduction

The physiological landscape of pregnancy represents one of the most significant stress tests the human cardiovascular system can undergo.[1] During a normal gestation, blood volume increases by nearly 50%, cardiac output rises, and systemic vascular resistance (SVR) decreases to accommodate the developing fetus.[2] While these adaptations are seamless in healthy parturients, they can prove catastrophic for those with underlying structural heart disease.[3] Globally, cardiac disease complicates approximately 1% to 4% of all pregnancies and remains a primary driver of indirect maternal mortality. Within this cohort, left-sided valvular lesions—such as mitral stenosis—have been extensively documented in obstetric literature.[4] However, the management of severe right-

sided pathology, specifically the combination of severe tricuspid regurgitation (TR) and pulmonary hypertension (PH), presents a unique and formidable clinical challenge. In the context of severe PH, the right ventricle (RV) is forced to function as the weak link of the cardiac circuit.[5] Unlike the robust, thick-walled left ventricle designed to pump against high systemic pressures, the RV is thin-walled and structurally optimized for a low-pressure, high-compliance pulmonary system. When pulmonary vascular resistance (PVR) is chronically or acutely elevated, the RV must generate significantly higher pressures to maintain forward flow. Severe tricuspid regurgitation exacerbates this fragility. As the RV dilates in response to high pulmonary pressures, the tricuspid valve annulus stretches, leading to functional regurgitation. This creates a vicious cycle: the regurgitant volume leads to RV volume overload, further dilation, and reduced contractility. In this hostile hemodynamic environment, the maintenance of coronary perfusion to the RV is paramount.[6] Any sudden drop in SVR—often seen with rapid-onset neuraxial anesthesia—reduces the aortic root pressure required to perfuse the stressed RV, potentially triggering a rapid descent into cardiovascular collapse. The clinical picture is further complicated when cardiac pathology intersects with systemic neurofibromatosis. While often viewed through a dermatological lens, neurofibromatosis introduces critical anesthetic risks.[7] Neurofibromas can manifest within the spinal canal, potentially distorting the neuraxial anatomy. This distortion can lead to erratic or unpredictable spread of local anesthetics, making the achievement of a consistent sensory block difficult. Furthermore, neurofibromas located within the airway pose a severe risk for difficult intubation. In emergency scenarios where general anesthesia (GA) might be considered as a rescue for a failed neuraxial block, the presence of airway neurofibromas combined with the physiological edema of pregnancy creates a high-risk intubation environment. Moreover, the transition to GA itself is hazardous; the positive pressure ventilation required can acutely increase PVR, which may be the final blow to an already struggling RV.[8]

The convergence of severe TR, PH, and systemic neurofibromatosis leaves the anesthesia provider with a non-existent margin for error. The primary goal is to provide surgical anesthesia while avoiding the rapid sympathectomy associated with standard spinal techniques. A sudden loss of sympathetic tone would cause a precipitous drop in SVR, uncoupling the RV from its coronary supply and leading to acute failure.[9] Consequently, the choice of anesthetic technique must prioritize hemodynamic stability and precision titration. The management of such complex cardio-obstetric cases necessitates a multidisciplinary approach, integrating cardiology diagnostics with meticulous anesthetic planning. In settings where advanced invasive monitoring—such as pulmonary artery catheters—might be unavailable, clinical success relies on the use of titrated techniques that allow for gradual physiological adaptation.[10]

This study aims to delineate the precise anesthetic strategies required to maintain RV preload and systemic vascular resistance (SVR) while simultaneously minimizing pulmonary vascular resistance (PVR) in a triple-pathology parturient. The novelty of this work lies in the successful application of a graded epidural approach as a precision tool to navigate this specific intersection of severe TR, PH, and neurofibromatosis. This case provides a critical blueprint for managing complex multi-organ complications in resource-limited emergency obstetric settings, demonstrating that a carefully titrated block can provide superior stability in the face of hostile hemodynamics.

2. Case Presentation

The patient provided written informed consent for the clinical management described herein and for the subsequent publication of this case report and its associated diagnostic data for academic and research purposes. A 37-year-old female (G2P1A0) at 34 weeks' gestation was referred to our tertiary care center, presenting with a one-week history of rapidly progressing respiratory symptoms, including worsening dyspnea, orthopnea, and a persistent chronic cough. Upon initial assessment, the patient exhibited signs of profound cardiovascular and respiratory

decompensation. She was categorized as New York Heart Association (NYHA) Class IV, indicating an inability to carry out any physical activity without significant discomfort and symptoms of heart failure even at rest. At the time of admission, the patient was in overt respiratory distress, characterized by a respiratory rate of 24 breaths per minute. Her oxygen saturation (SpO_2) was labile, fluctuating between 90% and 96% despite the administration of high-flow oxygen at 10 liters per minute via a non-rebreather mask. This clinical picture suggested a state of critical hypoxemia and impending respiratory failure, necessitating immediate multidisciplinary intervention.

A detailed physical examination further elucidated the severity of the patient's triple-pathology. Cardiovascular auscultation revealed a distinct diastolic murmur at the apex, while pulmonary auscultation confirmed the presence of bilateral basal rales, indicative of significant pulmonary venous congestion and edema. Beyond the cardiopulmonary findings, the patient's physical presentation was marked by the systemic manifestations of neurofibromatosis. Multiple cutaneous neurofibromas were observed distributed across the trunk and neck. This finding was of paramount importance for the anesthetic team, as systemic neurofibromatosis can involve the spinal canal—potentially distorting neuraxial anatomy—and the upper airway, which significantly increases the risk for difficult airway management and erratic local anesthetic spread.

Baseline laboratory investigations were conducted to assess the patient's metabolic and hematological status. Results showed: Hemoglobin: 13.5 g/dL; Leukocytes: 12,700 per microliter, suggesting a mild leukocytosis; Potassium: 2.8 mmol/L The severe hypokalemia (2.8 mmol/L) was a particularly concerning finding, as electrolyte imbalances in the context of a stressed right ventricle (RV) significantly elevate the risk of perioperative arrhythmias and cardiac arrest. Further diagnostic imaging provided evidence of structural heart disease. A chest X-ray revealed a cardiothoracic ratio of 60%, alongside clear radiological signs of pulmonary edema. An electrocardiogram (ECG) was performed, which confirmed right axis deviation and right ventricular hypertrophy, consistent with chronic pressure overload of the right heart secondary to pulmonary hypertension.

The most definitive diagnostic data were obtained through comprehensive echocardiography, which revealed a hostile hemodynamic environment. The key findings included: (1) Chamber dilation: Significant dilatation of the right atrium and right ventricle (RV); (2) Tricuspid pathology: Severe tricuspid regurgitation (TR) was identified, with a maximum velocity of 4.01 m/s and a calculated regurgitant volume of 112 mL; (3) Pulmonary hypertension (PH): The echocardiography indicated a high probability of PH, with a mean pulmonary arterial pressure (mPAP) of 50.39 mmHg and a systolic pulmonary arterial pressure (sPAP) of 79.32 mmHg. Interestingly, the left ventricular (LV) ejection fraction was preserved at 85%. This hyperdynamic state of the LV is often observed in severe right-sided failure, as the underfilled LV compensates for the drastically reduced forward flow across the pulmonary circuit. This constellation of findings—severe TR and high-pressure PH—indicated that the patient was in a state of fixed cardiac output, where the RV was struggling against high pulmonary vascular resistance (PVR). In such a delicate state, any sudden decrease in systemic vascular resistance (SVR) would likely lead to a catastrophic drop in coronary perfusion to the already ischemic and failing RV.

Figure 1 provides a longitudinal overview of the serial hemodynamic trends and critical clinical interventions throughout the 55-minute surgical procedure. The data visualizes the successful stabilization of a parturient with triple-pathology using a detailed titrated graded epidural technique. At the Pre-Induction Baseline (T=0), the patient presented with significant cardiovascular strain, indicated by a heart rate of 112 bpm and an oxygen saturation of 92% despite pre-oxygenation. These values reflect the underlying NYHA Class IV status and the compensatory tachycardic response to severe tricuspid regurgitation (TR) and pulmonary hypertension (PH). Following the placement of the epidural catheter at L1-L2, the anesthetic team initiated the induction using 0.375% Levobupivacaine and 50 mcg Fentanyl.

Table 1. Summary of Clinical Findings on Admission

I. PATIENT BACKGROUND & CLINICAL STATUS	
Patient Profile	37-year-old female (G2P1A0) at 34 weeks' gestation.
Functional Capacity	**NYHA Class IV** Severe dyspnea, orthopnea, and chronic cough.
Respiratory Vitals	Respiratory Rate: 24 bpm; SpO2: 90%–96% on 10L/min non-rebreather mask.
II. PHYSICAL EXAMINATION & AIRWAY RISKS	
Cardiopulmonary	Diastolic murmur (apex) and bilateral basal rales indicating pulmonary edema.
Neurofibromatosis	Multiple cutaneous neurofibromas across trunk and neck; risk for neuraxial/airway involvement.
III. LABORATORY, IMAGING, & ECG RESULTS	
Laboratory Values	Hb: 13.5 g/dL; WBC: 12,700/microliter; **K+: 2.8 mmol/L**
Chest Radiography	Cardiothoracic ratio (CTR) of 60%; clear signs of pulmonary venous congestion.
ECG Findings	Right axis deviation (RAD) and right ventricular hypertrophy (RVH).
IV. ECHOCARDIOGRAPHIC PARAMETERS	
Chamber Morphology	Significant right atrial and right ventricular (RV) dilatation.
Right Heart Pressure	**Mean PAP: 50.39 mmHg** Systolic PAP: 79.32 mmHg (High-probability Pulmonary Hypertension).
Tricuspid Valve	**Severe TR** Max velocity: 4.01 m/s; Regurgitant volume: 112 mL.
Left Heart Status	LV ejection fraction (EF) preserved at 85%.

By administering the local anesthetic in 3 mL increments every 5 minutes, the team achieved a controlled sympathectomy. This slow onset is vital; it provides the cardiovascular system with the necessary time to initiate compensatory vasoconstriction in non-blocked areas, thereby maintaining the SVR:PVR ratio. Given the risk of cardiovascular collapse with rapid sympathectomy or increased PVR, a graded epidural

was chosen. An epidural catheter was placed at L1-L2 with the tip at T11-T12. The induction consisted of 12 cc of 0.375 percent Levobupivacaine and 50 mcg Fentanyl, delivered in 3 mL increments every 5 minutes. Sensory block level was assessed after each increment, achieving a T6 level by 20 minutes. By the 3rd Increment (T=15), the heart rate had decreased to 102 bpm while the mean arterial pressure (MAP) stabilized at 75 mmHg. This was supported by the proactive titration of Phenylephrine to maintain systemic vascular resistance (SVR). Maintaining SVR is a critical objective in this case to ensure adequate aortic root pressure, which drives coronary perfusion to the stressed right ventricle (RV). At the point of Surgical Incision (T=25), the patient reached a state of Optimal Stability, with a heart rate of 98 bpm, a MAP of 78 mmHg, and an oxygen saturation of 97%. By avoiding the rapid drop in SVR typically seen with standard spinal anesthesia, the graded technique successfully prevented the RV failure cycle—a catastrophic loop where reduced coronary perfusion leads to RV ischemia, worsening TR, and eventual cardiovascular collapse.

During infant delivery (T=35), a minor hemodynamic shift was noted, with the heart rate rising to 105 bpm and the MAP adjusting to 74 mmHg. This transition coincided with the administration of an Oxytocin 10 IU infusion and the physiological autotransfusion of blood from the involuting uterus. The success of the intraoperative phase was further cemented by a strict fluid restriction of 300 mL. This prevented RV volume overload and allowed the oxygen saturation to improve to 98% by Skin Closure (T=55), effectively managing the pulmonary edema identified in preoperative investigations. Ultimately, Figure 1 demonstrates that a precision-titrated epidural provides superior stability in the hostile hemodynamics of right heart failure.

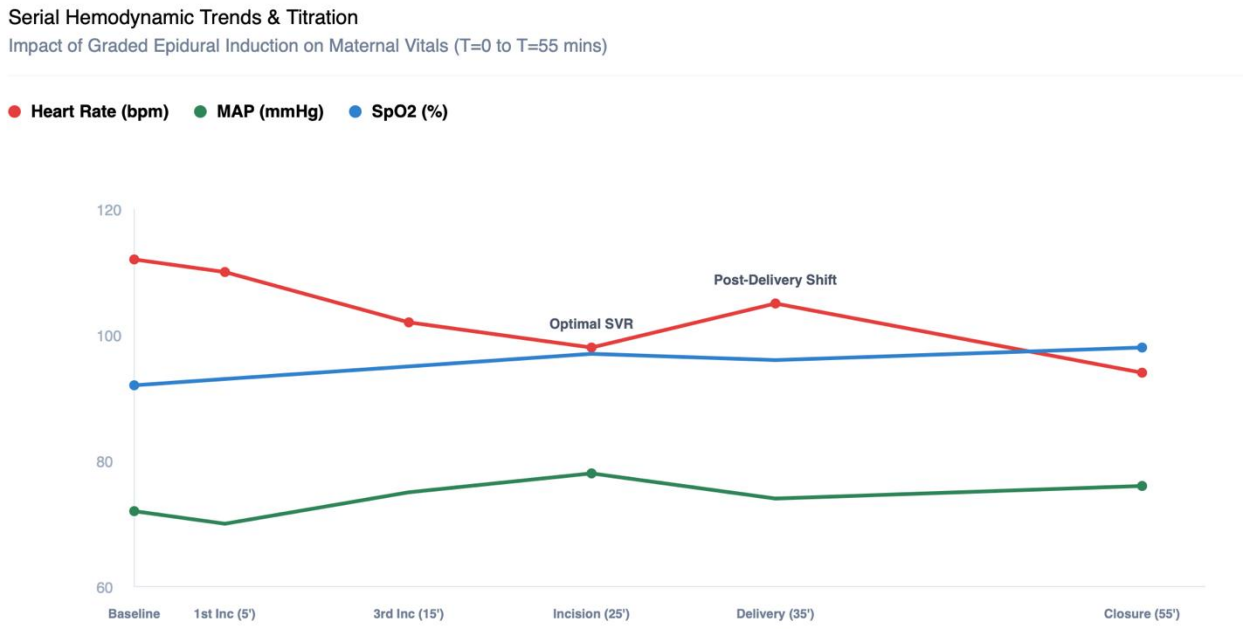

Figure 1. Serial hemodynamic monitoring and titration.

Table 2 provides a comprehensive overview of the clinical trajectory for this high-risk parturient, detailing the diagnostic criteria, interventional strategies, and successful outcomes. The diagnostic framework was defined by the critical intersection of three severe pathologies: tricuspid regurgitation (TR) with a regurgitant volume of 112 mL, pulmonary hypertension (PH) with a mean pulmonary arterial pressure of 50.39

mmHg, and systemic neurofibromatosis. This triple-pathology profile placed the patient in NYHA Class IV, characterized by significant respiratory distress and a fragile right ventricular-pulmonary unit.

The therapeutic intervention centered on a precision-based graded epidural technique. This involved the incremental administration of 0.375 percent Levobupivacaine and 50 mcg of Fentanyl in 3 mL doses every five minutes. Such meticulous titration was designed to facilitate a slow, compensatory sympathetic blockade, thereby preventing the catastrophic drop in systemic vascular resistance (SVR) that frequently precipitates right-heart failure in PH patients. To further safeguard the right ventricle, intraoperative care included strict fluid restriction—totaling only 300 mL—and the use of titrated phenylephrine to maintain coronary perfusion pressures.

Postoperative follow-up was conducted in the Intensive Care Unit, focusing on mitigating the risks of autotransfusion-induced volume overload through early diuresis and vigilant fluid balance during the first 24 hours. The outcome phase confirms the efficacy of this multidisciplinary approach: maternal hemodynamic stability was preserved throughout the 55-minute procedure. Neonatal results were equally favorable, with the delivery of a 2300-gram infant achieving APGAR scores of 6, 7, and 8. This systematic pathway underscores that technical precision and early coordination are indispensable when navigating the hostile hemodynamics of right-heart failure in complex obstetric cases.

TABLE 2. CLINICAL DIAGNOSIS, TREATMENT, AND OUTCOME PATHWAY

STAGE 1: CLINICAL DIAGNOSIS & PREOPERATIVE STATUS	
Cardiovascular Diagnosis	Severe TR & PH Severe Tricuspid Regurgitation (112 mL volume); High-probability PH (Mean PAP 50.39 mmHg; sPAP 79.32 mmHg); RV Dilatation with preserved LV EF (85%).
Systemic Complications	Neurofibromatosis Cutaneous neurofibromas (neck/trunk); Potential spinal canal involvement and difficult airway risks.
STAGE 2: INTRAOPERATIVE TREATMENT STRATEGY	
Anesthetic Technique	Graded Epidural L1-L2 placement (tip T11-T12); Incremental 3 mL dosing of 0.375% Levobupivacaine and 50 mcg Fentanyl every 5 minutes (Total: 12 cc).
Hemodynamic Support	Targeted Vasopressors Phenylephrine titration for SVR maintenance; Oxytocin 10 IU infusion post-delivery; Strict fluid restriction (300 mL crystalloid).
STAGE 3: POSTOPERATIVE FOLLOW-UP	
ICU Management	Intensive Monitoring for 24 hours postpartum; Monitoring for autotransfusion-induced RV volume overload; Early diuresis protocol.
STAGE 4: SURGICAL & MATERNAL-NEONATAL OUTCOMES	
Maternal Outcome	Hemodynamic Stability Successful management of 55-minute procedure; Blood loss limited to 300 mL; Stable discharge from ICU.
Neonatal Outcome	Healthy Delivery 2300-gram male infant; APGAR scores: 6 (1 min), 7 (5 min), 8 (10 min).

3. Discussion

The central anesthetic challenge in the management of the triple-pathology parturient—marked by severe tricuspid regurgitation (TR), pulmonary hypertension (PH), and systemic neurofibromatosis—revolves around the precarious fixed cardiac output state. In a healthy physiological environment, the right ventricle (RV) is structurally characterized as a thin-walled, highly compliant, low-pressure pump. It is evolutionarily designed to move the entire stroke volume into a high-compliance, low-resistance pulmonary circuit with minimal energy expenditure.[11] However, the introduction of severe PH fundamentally alters this dynamic. The RV is forced to undergo significant structural and functional adaptations, such as hypertrophy and dilatation, to generate the high pressures required to overcome increased pulmonary vascular resistance (PVR). In this specific case, the mean pulmonary arterial pressure of 50.39 mmHg and a systolic pulmonary arterial pressure of 79.32 mmHg represent a massive afterload against which the RV must struggle.

When the RV reaches the upper limits of its compensatory capacity, it becomes a weak link in the cardiovascular chain, possessing no reserve to meet further physiological demands. This state of fixed cardiac output means the heart cannot increase flow in response to the stress of pregnancy or surgery.[12] Consequently, the RV becomes exquisitely sensitive to even minor changes in afterload and coronary perfusion pressure. The primary driver of maternal mortality in such cases is the sudden decompensation of this right-heart unit.

A precipitous decrease in systemic vascular resistance (SVR) is the most feared trigger for cardiovascular collapse in these patients.[13] This drop in resistance is a common side effect of rapid-onset neuraxial anesthesia, such as a standard spinal block, which causes a sudden, widespread sympathectomy. When SVR falls, there is a corresponding drop in aortic root pressure. In the setting of severe PH and RV hypertrophy, the aortic root pressure is the primary driver of coronary perfusion to the stressed RV myocardium. If this pressure falls below a critical threshold, the RV—already suffering from high metabolic demand—is plunged into a state of acute ischemia.

This ischemia initiates a lethal, self-perpetuating hemodynamic cascade. As myocardial oxygen delivery fails, RV contractility is severely impaired. Reduced contractility leads to further RV dilatation, which in turn stretches the tricuspid valve annulus, acutely worsening functional TR.[14] The massive regurgitant volume (measured at 112 mL in this patient) further reduces forward cardiac output into the pulmonary artery. This leads to a further drop in systemic blood pressure, worse coronary perfusion, and more profound ischemia. Without precise intervention, this downward spiral inevitably culminates in rapid and irreversible cardiovascular collapse.

The presence of systemic neurofibromatosis adds a third layer of risk, as neurofibromas can manifest within the spinal canal or the airway. Anatomical distortions in the spinal column can complicate the placement of neuraxial needles and lead to erratic, unpredictable spread of local anesthetics. Meanwhile, airway neurofibromas pose a risk for difficult intubation in emergency scenarios. This makes the avoidance of general anesthesia (GA) even more critical. GA is particularly hazardous for the PH patient because positive pressure ventilation and laryngoscopy can acutely increase PVR, which would be catastrophic for an RV already functioning at its limit.[15]

To navigate this hostile environment, a graded epidural technique using 0.375 percent Levobupivacaine and 50 mcg Fentanyl in 3 mL increments was chosen. This precision approach allows for a controlled, slow-onset sympathectomy. By delivering the anesthetic incrementally every 5 minutes, the clinician provides the cardiovascular system with the necessary time to initiate compensatory vasoconstriction in non-blocked areas. This preserves the SVR to PVR ratio, ensuring that aortic root pressure remains sufficient to drive coronary perfusion to the RV throughout the surgical procedure. Coupled with strict fluid restriction of 300 mL to prevent RV volume overload, this strategy allows the clinician to provide surgical anesthesia while shielding the patient from the lethal cycle of right-heart failure.

This temporal window is critical because it gives the patient's cardiovascular system sufficient time to initiate compensatory vasoconstriction in the non-blocked sympathetic segments (Figure 2). By preserving the SVR to PVR ratio, the graded epidural ensures that systemic pressures remain high enough to maintain forward flow across the tricuspid valve and sustain the pressure gradient necessary for RV coronary perfusion. In this patient, the T6 sensory level achieved by 20 minutes provided adequate surgical conditions while maintaining the delicate right-heart balance.[16]

The clinical management of the triple-pathology parturient is defined by a precarious intersection of cardiopulmonary fragility and anatomical unpredictability. While the hemodynamic challenges of severe tricuspid regurgitation (TR) and pulmonary hypertension (PH) are central to the case, the presence of systemic neurofibromatosis introduces a critical third layer of complexity that fundamentally reshapes the anesthetic risk profile. Neurofibromatosis type 1 (NF1) is far more than a dermatological condition; in the context of obstetric anesthesia, it represents a multifaceted threat to both neuraxial access and airway security.

The systemic nature of neurofibromatosis means that benign nerve sheath tumors—neurofibromas—can manifest anywhere in the peripheral or central nervous system. Within the spinal canal, these tumors can lead to significant distortions of the neuraxial anatomy. For the anesthesiologist, this creates a blind procedural environment where standard anatomical landmarks may no longer correspond to the expected internal structures.

These distortions present dual risks during the placement of an epidural catheter: (1) Direct neural injury: The presence of unrecognized intraspinal neurofibromas increases the risk of traumatic needle-to-nerve contact or catheter-induced compression, which can lead to permanent neurological deficits; (2) Erratic block spread: Neurofibromas can act as physical barriers within the epidural or subarachnoid spaces, leading to an unpredictable or patchy spread of local anesthetics. In a patient where hemodynamic stability depends on a precise, gradual sympathectomy, an erratic or failed block is not merely a technical inconvenience—it is a trigger for emergency intervention.[17]

Consequently, the placement of the epidural catheter at the L1-L2 interspace (with the tip positioned at T11-T12) was performed with extreme caution. The choice of a graded approach served a secondary purpose: it allowed the clinical team to assess the quality of the block incrementally, ensuring that any anatomical barriers to the spread of Levobupivacaine were identified before the surgical incision.

Perhaps the most significant concern regarding neurofibromatosis is the potential for airway involvement. Neurofibromas can infiltrate the tongue, pharynx, or larynx, transforming a standard obstetric airway—already narrowed by the physiological edema of pregnancy—into a difficult airway scenario.[18] In an emergency Cesarean Section, the anesthesia team must always be prepared for the possibility of a failed neuraxial block, which would necessitate a rapid conversion to general anesthesia (GA).

In the context of this patient's severe PH (mean pulmonary arterial pressure of 50.39 mmHg), GA is fraught with life-threatening danger. The transition to GA involves two major physiological stressors that are catastrophic for a failing right heart: (1) Laryngoscopy and Intubation: The sympathetic surge associated with airway manipulation can acutely spike systemic and pulmonary pressures; (2) Positive pressure ventilation (PPV): The initiation of PPV increases intrathoracic pressure, which directly reduces venous return (preload) and increases pulmonary vascular resistance (PVR).

For a right ventricle (RV) already struggling to overcome a fixed, high-pressure pulmonary circuit, an acute increase in PVR can trigger the terminal RV failure cycle. As PVR rises, the RV is unable to maintain forward flow, leading to acute RV ischemia, worsening tricuspid regurgitation, and immediate cardiovascular collapse. Therefore, the successful application of the graded epidural was not just a preference; it was a life-saving maneuver designed specifically to avoid the death trap of emergency general anesthesia in a PH patient with potential airway neurofibromas.[19]

The intersection of these conditions created an environment where the margin for error was non-

existent. The success of this case hinged on recognizing that neurofibromatosis is a multisystem disorder that complicates every facet of the ABCD (Airway, Breathing, Circulation, and Drugs) of anesthesia.[20] By utilizing a meticulously titrated, graded epidural, the team navigated the anatomical risks of NF1 while shielding the fragile RV from the hemodynamic shocks of both rapid sympathectomy and the hazards of general anesthesia.

The clinical responsibility did not end with the successful delivery of the 2300-gram infant. The first 24 hours postpartum represent a high-risk period for parturients with right-heart disease. Following delivery, the involution of the uterus results in a significant autotransfusion of blood back into the maternal systemic circulation. While this is a normal physiological process, it can cause a sudden and severe RV volume overload in a patient whose right heart is already dilated and failing.

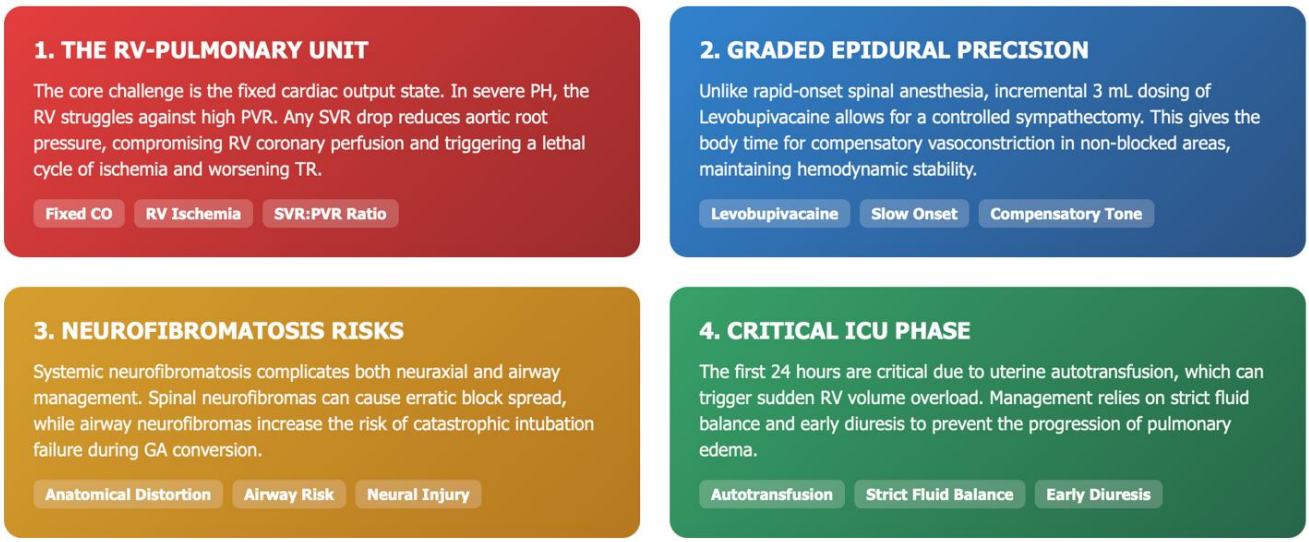

Figure 2. Conceptual framework of this study.

In the Intensive Care Unit (ICU), management focused on preventing this overload. This was achieved through meticulous fluid balance and the early initiation of diuresis. These interventions were essential to prevent the worsening of the pulmonary edema identified on the preoperative chest X-ray and to ensure the patient remained hemodynamically stable during the critical transition to the postpartum state.[19,20]

While the outcome for both the mother and the neonate was successful, this case highlights significant technical limitations often encountered in emergency obstetric settings. The management was conducted without the benefit of invasive hemodynamic monitoring, such as a pulmonary artery catheter, which could have provided real-time data on PVR and cardiac output.

Future clinical practice and research, particularly in resource-limited environments, should emphasize the integration of point-of-care ultrasound (POCUS). POCUS and other non-invasive cardiac output monitoring tools can provide vital, real-time guidance for fluid administration and vasopressor titration. As we move toward more precision anesthetic management, these technologies will be essential for managing multi-organ complications in high-risk cardio-obstetric patients.

4. Conclusion

The management of a parturient presenting with the synergistic complexities of severe tricuspid regurgitation (TR), high-probability pulmonary hypertension (PH), and systemic neurofibromatosis

represents one of the most formidable challenges in obstetric anesthesia. This triple-pathology creates a precarious physiological state where the margins for hemodynamic error are non-existent. The central anesthetic priority in such cases is the preservation of the right ventricular (RV)-pulmonary unit, which functions at the edge of compensatory failure. As demonstrated in this case, the graded epidural technique emerges not merely as an alternative, but as the preferred anesthetic choice for ensuring maternal and neonatal survival during emergency cesarean section.

The efficacy of the graded epidural lies in its ability to facilitate a controlled, slow-onset sympathectomy. By administering 0.375% Levobupivacaine in meticulous 3 mL increments, clinicians can bypass the catastrophic cardiovascular collapse often precipitated by the rapid sympathectomy of standard spinal anesthesia. This precision approach allows the cardiovascular system time to initiate compensatory vasoconstriction in non-blocked areas, thereby maintaining the critical SVR to PVR ratio. Such stability is paramount for sustaining aortic root pressure, which drives essential coronary perfusion to the already stressed and hypertrophied RV. Furthermore, the avoidance of general anesthesia (GA) is critical; the positive pressure ventilation and laryngoscopy associated with GA can acutely spike pulmonary vascular resistance, potentially triggering a terminal cycle of RV ischemia and worsening TR.

Success in these high-stakes cardio-obstetric scenarios hinges on a robust, early multidisciplinary approach. The integration of precise cardiological diagnostics—such as echocardiographic quantification of regurgitant volumes and mean pulmonary arterial pressures—with meticulous anesthetic titration and strict fluid management (limited to 300 mL in this case) provides a lifeline for the failing right heart. This case serves as a clinical blueprint, demonstrating that even in resource-limited emergency settings, precision management can be achieved through disciplined pharmacological titration and vigilant hemodynamic monitoring.

Looking forward, the management of complex triple-pathology cases should move toward a more technology-integrated, bedside approach. Clinicians should prioritize the mastery of point-of-care ultrasound (POCUS) as a non-invasive tool for real-time assessment of RV function, volume status, and pulmonary congestion. This allows for dynamic adjustments to fluid and vasopressor therapy without the risks associated with invasive pulmonary artery catheterization. The adoption of non-invasive cardiac output monitoring (NICOM) should be explored to provide continuous data on systemic vascular resistance and stroke volume, further refining the precision of graded induction. Institutions should develop high-fidelity simulation programs focused on right-heart crises in obstetrics, ensuring that multidisciplinary teams can execute graded epidural techniques and emergency rescue maneuvers with seamless coordination. In resource-limited settings, the focus must remain on the development of simplified yet rigorous titration protocols for long-acting local anesthetics, ensuring that high-level hemodynamic stability is accessible even in the absence of advanced invasive technology.

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