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Perioperative Management in a Cesarean Section Patient with Rheumatic Heart Disease and Pulmonary Hypertension

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ABSTRACT

Background: Pregnancy complicated by rheumatic heart disease (RHD) and pulmonary hypertension (PH) is a high-risk condition with maternal mortality reaching 20–50%. The physiological burden of pregnancy can precipitate cardiovascular decompensation, making perioperative management of cesarean section extremely challenging. Objective: This case report aims to describe the perioperative challenges and the multidisciplinary anesthetic strategy implemented in a high-risk parturient with RHD and PH, emphasizing the rationale for choosing general anesthesia over regional techniques. Methods: A 25-year-old woman (G3P2A0) at 32–33 weeks gestation presented in labor with signs of fetal distress. She had a history of RHD with moderate-to-severe mitral stenosis, moderate tricuspid regurgitation, PH, and atrial fibrillation with rapid ventricular response. Due to significant coagulopathy (INR 2.3), regional anesthesia was contraindicated. The patient underwent general anesthesia with gradual induction, invasive hemodynamic monitoring (arterial and central venous lines), and lung-protective ventilation for an emergency cesarean section. Findings: The procedure was completed successfully with the delivery of a live infant with good Apgar scores. Intraoperatively, the patient remained hemodynamically stable with support from inotropes and vasopressors. Postoperatively, she was managed in the intensive care unit (ICU) for four days before being transferred to the general ward and discharged without major complications. Implications: This case underscores that in specific high-risk scenarios where regional anesthesia is contraindicated, a carefully conducted general anesthesia with invasive monitoring can be a safe and effective alternative.

KEYWORDS Anesthesia; Pulmonary; Hypertension; Heart; Pregnancy; Cesarean Section

INTRODUCTION

Pregnancy complicated by rheumatic heart disease (RHD) and pulmonary hypertension (PH) represents an extremely high-risk condition, with maternal mortality rates reported to reach 20–50%. The combination of increased circulatory volume load, rapid hemodynamic changes during pregnancy and delivery, and limited anesthetic options makes perioperative management highly complex (Ahmadzadeh et al., 2025; Maisat & Yuki, 2024; Melaku, 2022; Meng et al., 2023). Physiological changes in pregnancy, such as up to a 50% increase in blood volume, increased cardiac output, and decreased systemic vascular resistance, can lead to decompensation in patients with valvular disease due to rheumatic heart disease or elevated pulmonary vascular resistance. Consequently, the risk of heart failure increases significantly, particularly during the intrapartum and postpartum periods (Adare et al., 2023; Ahmadzadeh et al., 2025).

Rheumatic heart disease commonly causes valvular abnormalities, especially mitral stenosis or regurgitation, which impose pressure and volume overload on both the left and right sides of the heart (Passos et al., 2021; Remenyi et al., 2016; Unger et al., 2018). In this condition, the increased circulating volume during pregnancy further elevates left atrial

pressure, leading to pulmonary congestion. If this persists, pulmonary circulation pressure rises and results in secondary pulmonary hypertension. On the other hand, pulmonary hypertension itself whether idiopathic or secondary to left heart disease plays a major role in increasing maternal mortality risk. In patients with PH, sudden changes in preload, afterload, and pulmonary vascular resistance during anesthesia or labor can trigger acute right heart failure or circulatory collapse. The postpartum period is also a critical phase due to autotransfusion from the uterus to the systemic circulation, which abruptly increases preload and predisposes to cardiac decompensation.

Perioperative management of cesarean section in patients with RHD and PH requires a multidisciplinary approach involving obstetricians, cardiologists, anesthesiologists, and intensive care specialists. Comprehensive preoperative evaluation is essential to assess disease severity, ventricular function, hemodynamic status, and cardiovascular risk classification during pregnancy, such as the WHO risk classification. Optimization before surgery includes blood pressure control, fluid balance management, and treatment of heart failure symptoms if present (Futier et al., 2017; Pichette et al., 2017; Zaza et al., 2026). Preparation for invasive monitoring such as an arterial line or central venous line should be considered, and in extreme-risk cases, circulatory support devices like extracorporeal membrane oxygenation (ECMO) may be prepared as an anticipatory measure in case of intraoperative cardiovascular collapse (Fraccaro et al., 2020; Hutt & Desai, 2020).

Anesthetic strategy plays a central role in preventing dangerous hemodynamic fluctuations. Regional anesthesia techniques, such as graded epidural anesthesia, are often preferred because they avoid sympathetic stimulation from intubation and minimize blood pressure fluctuations. However, regional anesthesia must be administered cautiously to prevent sudden reductions in preload and afterload that may decrease cardiac output. In certain circumstances, general anesthesia may be considered when regional anesthesia is contraindicated, provided that strict control of ventilation and oxygenation is maintained to avoid increased pulmonary vascular resistance. Intraoperative fluid management must be precise; excessive fluids can worsen right or left heart failure, whereas fluid deficit can impair systemic perfusion. Drugs such as vasopressin may be used to maintain blood pressure without increasing pulmonary vascular resistance, while uterotonic agents such as methylergometrine and carboprost should be avoided because they can exacerbate pulmonary hypertension. Oxytocin should be administered in low doses and infused slowly to reduce the risk of sharp systemic vasodilation.

During surgery, meticulous hemodynamic control should be maintained with monitoring of arterial pressure, heart rate, oxygen saturation, and, if possible, central venous pressure or intraoperative echocardiography. Excessive intra-abdominal pressure and positions that compress the inferior vena cava should be avoided. After delivery and placental separation, rapid circulatory volume changes must be anticipated with careful fluid management and diuretic use when necessary to prevent volume overload (Kachhwaha et al., 2025; Minhas et al., 2021).

The postoperative period is the most critical phase for heart failure occurrence. Therefore, patients with RHD and PH after cesarean section should be managed in an intensive care unit for at least the first 24–72 hours. Close monitoring of vital signs, urine output, blood pressure, and oxygen saturation is essential. Adequate pain control is important to prevent tachycardia

and increased cardiac workload due to pain or stress. Patient activity should be gradually resumed with attention to hemodynamic stability. Furthermore, coordination with cardiologists for follow-up is necessary to assess cardiac function postpartum and determine further therapy, such as management of pulmonary hypertension or valve intervention if needed.

The primary objective of this case report is to present the successful perioperative management of a parturient with severe RHD, PH, and coagulopathy who required general anesthesia for an emergency cesarean section. By detailing the anesthetic strategy, intraoperative challenges, and multidisciplinary coordination, this report aims to fill the gap in literature regarding the safe execution of general anesthesia when regional techniques are contraindicated. The implications of this report are to provide a practical management framework for clinicians facing similar high-risk scenarios, ultimately contributing to improved maternal and fetal outcomes in this vulnerable patient population.

Recent case reports have shown that with a multidisciplinary approach and cautious anesthetic management, favorable maternal and neonatal outcomes can be achieved despite the high risk. Successful perioperative management has been reported in patients with severe mitral stenosis and pulmonary hypertension undergoing cesarean section with standby ECMO preparation, without fatal complications. Other studies have emphasized the importance of adapting anesthetic techniques to the patient's cardiovascular condition and ensuring postoperative ICU readiness. In developing countries like Indonesia, where rheumatic heart disease remains endemic and access to advanced cardiovascular facilities is limited, implementing evidence-based perioperative management protocols is crucial. Establishing cardio-obstetric teams, providing specialized training in obstetric anesthesia for high-risk cases, and developing referral systems to centers with interventional cardiology facilities should be prioritized to reduce maternal mortality associated with cardiac complications in pregnancy. Therefore, perioperative management of cesarean section in patients with rheumatic heart disease and pulmonary hypertension must be comprehensively and individually planned, encompassing thorough preoperative assessment, appropriate anesthetic strategy, intraoperative hemodynamic control, and intensive postoperative monitoring. A multidisciplinary approach and interdepartmental coordination are key to minimizing the risk of heart failure and maternal mortality, while ensuring the safety of both mother and baby (Simpson et al., 2023).

METHOD

CASE

A 25-year-old female patient, G3P2A0, at 32–33 weeks of gestation, was admitted to the hospital in obstetric and cardiovascular emergency condition. The patient was on mechanical ventilation. Based on the initial assessment, she was referred from GM Hospital with complaints of progressive shortness of breath over the past month, inability to lie flat, dyspnea on exertion, and paroxysmal nocturnal dyspnea, accompanied by bilateral lower limb edema. The patient had been taking cardiac medications regularly since being hospitalized in May and continued them as an outpatient but had never monitored her INR levels. She had been taking half a tablet of warfarin daily but stopped two days prior to hospital admission.

Her past medical history revealed moderate to severe mitral stenosis (MS), moderate tricuspid regurgitation (TR), and atrial fibrillation (AF) with a CHA_2DS_2-VASc score of 1 and

HAS-BLED score of 2. The patient was classified as ASA 4E, with obstetric status of gravida complicated by valvular heart disease secondary to rheumatic heart disease (moderate MS, trivial MR, mild PR, moderate TR, TVG 60 mmHg, suggesting high probability of pulmonary hypertension), AF with rapid ventricular response, and prolonged coagulation parameters (PT 2.12x, APTT 1.31x, INR 2.29x). Laboratory findings also showed hyponatremia (Na 125 mmol/L) and elevated transaminase enzymes. The duration of surgery was 1 hour, with a total anesthesia time of 1 hour and 20 minutes.

During surgery, the patient received 500 mL of Ringer's Lactate as crystalloid infusion. Urine output was recorded at 700 mL, and estimated blood loss was approximately 400 mL. The patient was intubated at 12:45 PM on July 9, 2025, with a central venous catheter (CVC) placed in the right internal jugular vein at 12:15 PM, and an arterial line inserted in the left radial artery at 12:30 PM.

On objective examination, the patient weighed 60 kg with a height of 155 cm, and her general condition was severe. Respiratory system (B1): an ETT no. 7.5 non-kinking tube was in place with balloon pressure of 20 cmH_2O, respiratory rate 15 breaths/min, SpO_2 99% on SIMV mode ventilation (VT 380 mL, RR 15, PS 8, PEEP 5, FiO_2 50%). Cardiovascular system (B2): blood pressure 108/57 mmHg, heart rate 84 bpm with irregular rhythm, MAP 74 mmHg; the patient was on norepinephrine 0.017 mcg/kg/min, dobutamine 6 mcg/kg/min, and milrinone 0.2 mcg/kg/min. Neurological system (B3): the patient was sedated with midazolam 2 mg/h, fentanyl 20 mcg/h, and dexmedetomidine 0.2 mcg/kg/h; GCS prior to intubation was E4M5V6, RASS -2, and BPS 3. Urogenital system (B4): a urinary catheter was in place with urine output of 1.84 mL/kg/h, negative fluid balance (-951.55 mL), and creatinine clearance of 25 mL/min. Gastrointestinal system (B5): nasogastric tube in place with negative gastric residuals. Extremities were warm, capillary refill time <2 seconds, and no abnormalities noted.

Laboratory results on July 9, 2025 showed: Na 125 mmol/L, K 4.9 mmol/L, Cl 99 mmol/L, leukocytes 11,930/µL, erythrocytes 5.16 $\times 10^6$/µL, Hb 15.9 g/dL, hematocrit 45.4%, platelets 237 $\times 10^3$/µL. Coagulation profile: PT 29.7/14.0 = 2.12x, INR 2.39/1.04 = 2.30x, APTT 42.3/32.1 = 1.32x. eGFR 80 mL/min/1.73m², random blood glucose 70 mg/dL, HBsAg non-reactive, SGOT 67 U/L, SGPT 43 U/L, urea 41 mg/dL, creatinine 0.9 mg/dL, and anti-HIV non-reactive.

Follow-up laboratory examination at 18:21 WITA on July 9, 2025 revealed: Serum sodium 132 mmol/L, potassium 4.9 mmol/L, chloride 96 mmol/L, leukocytes 11.77 $\times 10^3$/µL, erythrocytes 5.14 $\times 10^6$/µL, hemoglobin 15.8 g/dL, hematocrit 45.8%, platelets 259 $\times 10^3$/µL, MCH 30.7 pg, MCHC 34.5 g/dL, and MCV 89.1 fL. Coagulation test results: PT 25.9 sec (control 13.9 sec), INR 2.05 (control 1.03), APTT 42.1 sec (control 30.9 sec). eGFR (CKD-EPI) was 71 mL/min/1.73m². Other results: magnesium 1.53 mg/dL, D-dimer 1.07 µg/mL, total bilirubin 1.99 mg/dL, direct bilirubin 1.49 mg/dL, SGOT (AST) 67 U/L, SGPT (ALT) 47 U/L, serum urea 41 mg/dL, serum creatinine 0.91 mg/dL, total calcium 7.9 mg/dL, and phosphorus 6.1 mg/dL. Chest X-ray revealed cardiomegaly.

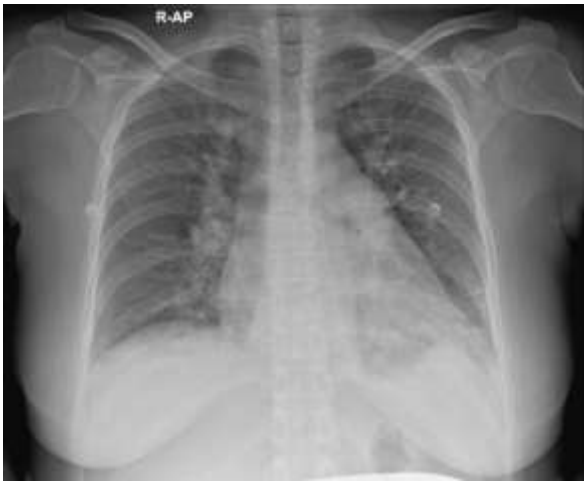

Figure 1. Chest Radiograph

Emergency echocardiography performed on July 9, 2025, revealed atrial fibrillation with rapid ventricular response (AF with RVR), reduced left ventricular ejection fraction (LVEF), moderate-to-severe mitral stenosis, and an inferior vena cava (IVC) diameter of 1.9 cm with less than 50% collapse. The 12-lead ECG from the IRDO showed atrial fibrillation with RVR (160–170 bpm), right axis deviation, and poor R-wave progression.

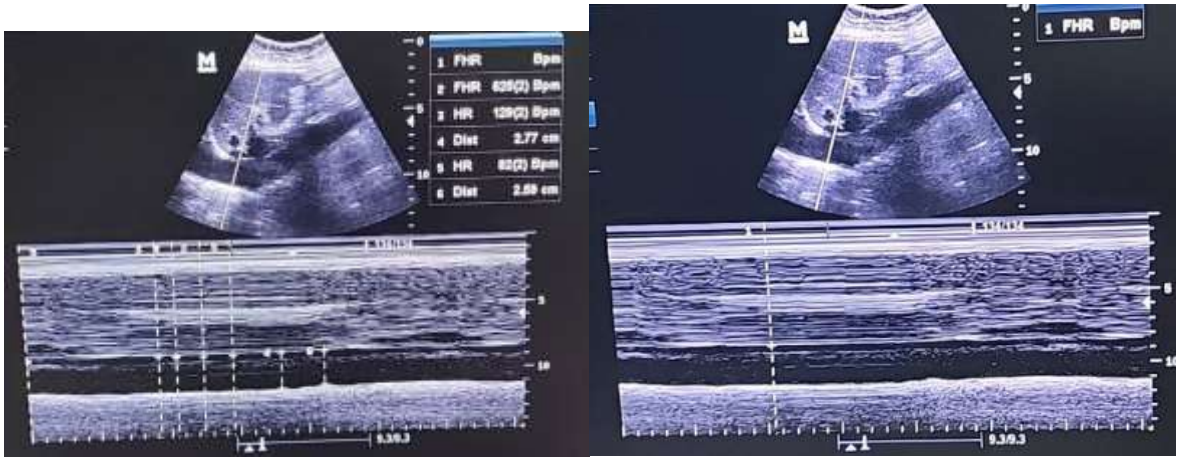

Figure 2. Echocardiography

Transthoracic echocardiography (TTE) performed on May 5, 2025, revealed left atrial (LA) and right ventricular (RV) dilatation (LAVI 47 mL/m², RV base 4.3 cm), a D-shaped left ventricle (LV) without LV hypertrophy, and normal LV systolic function with an ejection fraction (LVEF) of 57%. Global segmental motion was normokinetic. The aortic valve had three cusps with calcification noted on the non-coronary cusp (NCC), right coronary cusp (RCC), and left coronary cusp (LCC). Mild aortic regurgitation (AR) was present (AR PHT 640 ms). There was moderate-to-severe mitral stenosis (MVA planimetry 1.3 cm², MVA VTI 1.3 cm², mean pressure gradient 13.6 mmHg) with rheumatic etiology and a Wilkins score of 8–9 (mobility 2, valve thickness 2, subvalvular thickening 1–2, calcification 3). Trivial mitral regurgitation (MR), mild pulmonary regurgitation (PR PHT 665 ms), and moderate tricuspid regurgitation (TR) (vena contracta 0.4 cm, EROA 0.2 cm², TVG 60 mmHg) were also observed, indicating a high probability of pulmonary hypertension. RV contractility was reduced, with TAPSE 1.2 cm and RVS' 9 cm/s. The inferior vena cava (IVC) measured 2.2 cm with >50% collapse (estimated RAP 8 mmHg). Minimal pericardial effusion was observed at

the RV free wall (0.9 cm) and posterior wall (0.8 cm), with atrial fibrillation rhythm noted during the examination.

The patient was diagnosed with respiratory failure on mechanical ventilation, P3A0 post–cesarean section due to 32–33 weeks of gestation with first-stage labor and a high-risk pregnancy (due to maternal age and obstetric history — 11-year interpregnancy interval), atrial fibrillation with rapid ventricular response (RVR), CHA_2DS_2-VASc score 1, HAS-BLED score 2, heart failure functional class II–III secondary to valvular heart disease, hyponatremia, moderate-to-severe mitral stenosis, moderate tricuspid regurgitation, minimal pericardial effusion, hypomagnesemia, hypocalcemia, and hepatopathy.

Antibiotic therapy was initiated with ceftriaxone 1 gram IV every 12 hours. Other treatments included lansoprazole 30 mg IV every 24 hours, tranexamic acid 500 mg IV every 8 hours, paracetamol 500 mg IV every 8 hours PRN, furosemide 20 mg IV twice daily, oxytocin 20 IU in 50 mL D5% every 8 hours IV via syringe pump for the first 24 hours, magnesium sulfate 7.5 grams in 50 mL infused over 6 hours, human albumin 100 mL IV every 24 hours, N-acetylcysteine 200 mg via NGT every 8 hours, vitamin B complex 1 tablet via NGT every 8 hours, vitamin C 200 mg via NGT every 24 hours, zinc 20 mg via NGT every 24 hours, bisoprolol 2.5 mg via NGT once daily, digoxin 0.125 mg via NGT once daily, and warfarin 2 mg via NGT once daily at night.

The patient was monitored jointly by the obstetrics-gynecology and cardiology teams, with plans for ventilator and central line bundles, periodic complete laboratory tests, and arterial blood gas analyses. Nutritional support provided 1,500–1,800 kcal/day. Analgesia was maintained with paracetamol 500 mg IV every 8 hours, and sedation was achieved with fentanyl at 0.5–1 µg/kg/min.

Figure 3. Patient monitoring observation

RESULT AND DISCUSSION

Pregnancy in patients with rheumatic heart disease represents a high-risk condition that can result in serious cardiovascular complications for both the mother and the fetus. Physiologically, pregnancy increases plasma volume by up to 40–50% and cardiac output by 30–50%, which imposes a greater burden on the heart particularly in patients with valvular abnormalities.[7–8] In this case, a 25-year-old woman, G3P2A0, 32–33 weeks pregnant, presented with progressive shortness of breath, orthopnea, and paroxysmal nocturnal dyspnea, which are classic manifestations of left-sided heart failure due to mitral stenosis. The inability to lie flat and the presence of leg edema indicated elevated pulmonary venous pressure and systemic congestion resulting from severe valvular dysfunction.

The patient's mitral stenosis was classified as moderate to severe, with a mitral valve area of 1.3 cm^2 and a mean pressure gradient of 13.6 mmHg. Pathophysiologically, mitral stenosis leads to obstruction of blood flow from the left atrium to the left ventricle, increasing left atrial pressure and eventually causing pulmonary hypertension and left atrial dilatation. These mechanisms explain the patient's atrial fibrillation (AF) with rapid ventricular response and clinical signs of pulmonary congestion. Pulmonary hypertension, reflected by a tricuspid gradient of 60 mmHg, exacerbates right heart failure and results in peripheral edema. Echocardiography also revealed reduced right ventricular contractility (TAPSE 1.2 cm), indicating chronic pressure overload secondary to long-standing mitral stenosis.

Atrial fibrillation is a common complication of mitral stenosis caused by left atrial enlargement. AF with rapid ventricular response increases myocardial oxygen demand and decreases effective cardiac output, worsening heart failure symptoms and elevating the risk of thromboembolism.9,10 This patient had a CHA_2DS_2-VASc score of 1 and a HAS-BLED score of 2, suggesting a moderate risk of thromboembolism and a low-to-moderate risk of bleeding. However, irregular INR control and discontinuation of warfarin two days prior to hospital admission led to hemostatic imbalance, as reflected in laboratory findings of PT 2.12×, APTT 1.31×, and INR 2.29×. This further increased the risk of intraoperative and postoperative bleeding, particularly in an obstetric patient undergoing cesarean section.

Anesthetic management in patients with valvular heart disease and pulmonary hypertension requires meticulous hemodynamic control.9 In this case, the patient was classified as ASA 4E, indicating high perioperative mortality risk. Because of anticoagulant therapy and prolonged coagulation time, regional anesthesia (spinal/epidural) was relatively contraindicated. Therefore, general anesthesia with gradual induction, invasive monitoring (central venous and arterial lines), and protective ventilation was selected to maintain cardiopulmonary stability. Mechanical ventilation was set to SIMV mode with a tidal volume of 380 mL, PEEP of 5 cmH_2O, and FiO_2 of 50% to ensure adequate oxygenation while minimizing intrathoracic pressure that could reduce venous return.

Inotropic support with titrated doses of dobutamine, milrinone, and norepinephrine was administered to maintain systemic perfusion and stabilize blood pressure. Dobutamine enhances myocardial contractility, milrinone improves right ventricular function and reduces pulmonary vascular resistance, while norepinephrine maintains systemic perfusion pressure without significantly increasing pulmonary afterload.10,11 Laboratory findings showed

hyponatremia (125–132 mmol/L) and mild elevations in SGOT/SGPT, which could be attributed to hepatic congestion from right heart failure (congestive hepatopathy). Blood urea was elevated (41 mg/dL) with normal creatinine (0.9 mg/dL), indicating mild renal hypoperfusion without acute kidney injury.

The cesarean section was performed under close collaboration among the obstetrics, anesthesiology, and cardiology teams. The main management principles included maintaining optimal preload, avoiding tachycardia, preventing increases in pulmonary pressures, and ensuring rhythm stability. Postoperatively, the patient was admitted to the ICU for mechanical ventilation and pharmacologic support to control AF, improve ventricular function, and correct electrolyte imbalances. Continued therapy included diuretics to relieve congestion, digoxin and bisoprolol for rate control, and warfarin for thromboembolism prevention. Gradual correction of hyponatremia, hypocalcemia, and hypomagnesemia was undertaken to prevent further arrhythmias.

This case illustrates the complexity of perioperative management in a patient with rheumatic heart disease, pulmonary hypertension, and atrial fibrillation during pregnancy. The combination of pregnancy-induced physiological changes and structural cardiac abnormalities places such patients at high risk of heart failure, arrhythmia, and obstetric complications. A comprehensive multidisciplinary approach including strict hemodynamic control, protective ventilation, correction of electrolyte disturbances, and meticulous anticoagulant management is essential to prevent maternal mortality and fetal complications.

CONCLUSION

Pregnancy in patients with rheumatic heart disease and pulmonary hypertension presents a complex clinical challenge with a high risk of maternal and fetal morbidity and mortality. The physiological changes of pregnancy that increase hemodynamic load exacerbate mitral stenosis and pulmonary hypertension, leading to heart failure, atrial fibrillation, and impaired perfusion of vital organs. In the perioperative context, the use of general anesthesia with invasive monitoring, inotropic support, and protective ventilation strategies is crucial to maintain cardiopulmonary stability and prevent acute decompensation. Successful management relies heavily on multidisciplinary collaboration among obstetric, anesthesiology, and cardiology teams, focusing on hemodynamic optimization, heart rhythm control, electrolyte balance, and strict anticoagulation management. A comprehensive and individualized therapeutic approach based on the patient's clinical condition is essential to minimize intraoperative complications and improve both maternal and perinatal outcomes.

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