

General Anesthesia for Emergent Cesarean Delivery in a Parturient with Severe Left Ventricular Dysfunction: A Case Report with a Multidisciplinary, Rescue-Oriented Strategy

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Abstract

Background: Severe left ventricular dysfunction during pregnancy carries extreme maternal and fetal risk. Anesthetic management becomes challenging when rapid clinical deterioration coincides with an urgent need for therapeutic anticoagulation. **Case Presentation:** A 36-year-old woman at 32 weeks and 3 days of gestation with New York Heart Association class IV heart failure, atrial fibrillation with rapid ventricular response, and an ejection fraction of 25% underwent emergent cesarean delivery for rapidly worsening cardiac decompensation. Given severe ventricular dysfunction, persistent tachyarrhythmia, and anticipated immediate postoperative therapeutic anticoagulation, neuraxial anesthesia was deemed unsafe despite institutional preference for epidural techniques in cardiac parturients. A multidisciplinary team involving obstetrics, cardiology, anesthesiology, and cardiothoracic surgery established a rescue-oriented perioperative strategy. Femoral arterial guidewire placement was performed preemptively to facilitate rapid extracorporeal membrane oxygenation (ECMO) cannulation if required, and the cardiothoracic surgeon remained on standby throughout delivery. General anesthesia was administered with invasive monitoring. Intraoperative transesophageal echocardiography guided management and informed the decision to perform synchronized cardioversion for refractory atrial fibrillation; ECMO was not required. Left ventricular function improved from 25% preoperatively to 52% by postoperative day 4. **Conclusion:** Near-catastrophic obstetric cardiac cases benefit from structured risk stratification, anticoagulation-informed anesthetic selec-

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tion, real-time echocardiographic decision support, and predefined multidisciplinary rescue pathways, including readiness for mechanical circulatory support.

Keywords

Cesarean Section, Heart Failure, Ventricular Dysfunction, Atrial Fibrillation, Extracorporeal Membrane Oxygenation, Pregnancy, High-Risk

1. Introduction

Pregnancy induces profound cardiovascular adaptations, including increases in plasma volume, heart rate, and cardiac output [1]. While these physiologic changes are generally well tolerated in healthy women, they may precipitate decompensation in patients with significant structural or functional heart disease [1]. Women with severe left ventricular dysfunction are particularly vulnerable to abrupt alterations in preload, afterload, heart rate, and contractility during cesarean delivery, placing them at substantial risk of acute heart failure and cardiovascular collapse [1]-[3].

According to the modified World Health Organization (mWHO) classification of maternal cardiovascular risk, severe systemic ventricular dysfunction (ejection fraction <30% or New York Heart Association (NYHA) class III-IV symptoms) is considered to represent an extremely high-risk, near-prohibitive condition in pregnancy [2]. Contemporary prediction models such as the Cardiac Disease in Pregnancy Risk Index II (CARPREG II) further identify severe ventricular dysfunction and arrhythmia as major predictors of adverse peripartum cardiac events [4]. Nevertheless, tertiary referral centers increasingly encounter parturients with advanced cardiac disease, presenting complex anesthetic and perioperative challenges. International guidelines recommend multidisciplinary management and individualized delivery planning for pregnant patients with cardiovascular disease [2] [4].

We report the multidisciplinary perioperative management of an emergent cesarean delivery in a parturient with severe left ventricular dysfunction, atrial fibrillation (AF) with rapid ventricular response, and intracardiac thrombus. This case highlights the importance of structured risk stratification, anticoagulation-informed anesthetic selection, intraoperative echocardiographic guidance, and anticipatory mechanical circulatory support readiness.

2. Case Presentation

A 36-year-old woman at 32 weeks and 3 days' gestation was admitted to our institution for rapidly worsening dyspnea and suspected cardiac decompensation. She was 161 cm tall and weighed 99 kg (body mass index, 38.2 kg/m²).

In 2020, she was diagnosed with hyperthyroidism and was intermittently treated

with antithyroid medication, with poor adherence.

In 2022, she was diagnosed with AF and heart failure with reduced ejection fraction at a regional hospital and was prescribed digoxin and bisoprolol. Medication adherence was suboptimal, and she described intermittent lower extremity edema. During pregnancy, she reported taking bisoprolol 5 mg only intermittently and was not receiving anticoagulation therapy. On admission, thyroid function tests were consistent with uncontrolled hyperthyroidism, with a free thyroxine (FT4) level of 2.36 ng/dL and a thyroid-stimulating hormone (TSH) level of 0.005 μ IU/mL.

3. Pregnancy Course and Acute Deterioration

According to the patient, at approximately 28 weeks' gestation she began experiencing progressive dyspnea and was informed of polyhydramnios at a local hospital.

At 32 weeks and 2 days' gestation, she underwent amnioreduction of approximately 1 L at a local obstetric clinic for symptomatic relief. Although dyspnea transiently improved, respiratory distress reportedly recurred and progressively worsened.

Later that day, she presented to a regional referral center because of persistent dyspnea. During evaluation at the referring hospital prior to transfer, she experienced a syncopal episode, raising concern for significant hemodynamic instability. She was subsequently referred to our emergency department.

Upon arrival at our institution, she remained in AF with rapid ventricular response (heart rate 160 - 170 beats/min) and exhibited NYHA class IV symptoms. Preoperative N-terminal pro-B-type natriuretic peptide (NT-proBNP) was 733 pg/mL, and chest radiography demonstrated cardiomegaly, further supporting active cardiac decompensation.

Emergent transthoracic echocardiography demonstrated severe global left ventricular systolic dysfunction with an ejection fraction of 25% and revealed a right atrial thrombus. The presence of an intracardiac thrombus in the setting of uncontrolled AF indicated a very high embolic risk.

Given the syncopal event, persistent AF with rapid ventricular response, severely reduced ventricular function (EF 25%), and confirmed right atrial thrombus, multidisciplinary discussion was initiated immediately. The consensus was that urgent delivery was necessary both to prevent further maternal cardiovascular deterioration and to allow prompt initiation of therapeutic anticoagulation. Further delay in delivery was considered unsafe due to the risk of cardiovascular collapse. Emergent cesarean delivery at 32 weeks and 3 days' gestation was therefore recommended as a maternal rescue intervention, with a plan to initiate full-dose anticoagulation immediately postpartum. At that time, the fetal heart rate was 150 beats/min with moderate variability, indicating reassuring fetal status. Thus, fetal status did not independently drive the decision for cesarean delivery, which was undertaken primarily for maternal rescue.

4. Preoperative Multidisciplinary Planning

An urgent multidisciplinary discussion was held among anesthesiologists, obstetricians, cardiologists, and the cardiothoracic surgeon. From a hemodynamic standpoint, a carefully titrated epidural technique would ordinarily be preferred for cesarean delivery in a patient with severe ventricular dysfunction. However, the coexistence of persistent atrial fibrillation with rapid ventricular response and an intracardiac thrombus indicated an extremely high thromboembolic risk, and prompt initiation of therapeutic-dose anticoagulation immediately postpartum was planned. The preoperative profile, including severe systolic dysfunction, elevated NT-proBNP, and cardiomegaly on chest radiography, also heightened concern for perioperative decompensation. Moreover, given the substantial risk of perioperative cardiogenic shock, escalation to extracorporeal membrane oxygenation (ECMO) was considered a realistic contingency, necessitating systemic heparinization. In this context, neuraxial anesthesia and an indwelling epidural catheter were deemed unsafe due to the risk of neuraxial hematoma, and general anesthesia was therefore selected despite its potential hemodynamic challenges. The perioperative strategy incorporated ECMO readiness, including invasive arterial and central venous monitoring prior to induction, preparation for intraoperative transesophageal echocardiography (TEE), and close coordination with the cardiothoracic surgery team for immediate mechanical circulatory support in the event of cardiovascular collapse.

5. Intraoperative Management and Course

Upon arrival in the operating room, the patient remained in AF with rapid ventricular response (heart rate 160 - 170 beats/min), and systolic blood pressure ranged from 120 to 140 mmHg. Invasive arterial and central venous monitoring were established prior to induction as planned. Given the anticipated risk of sudden circulatory collapse and in accordance with the preoperative multidisciplinary strategy, the cardiothoracic surgeon placed femoral arterial and venous introducer sheaths (with guidewires in situ) under sterile conditions immediately after operating room arrival to facilitate rapid ECMO cannulation if required.

General anesthesia was induced immediately before skin incision with etomidate 10 mg and rocuronium 100 mg. The neonate (birth weight 2.29 kg) was delivered 4 minutes after induction, with an estimated blood loss of 400 mL at that time. Approximately 1 minute after delivery, oxytocin 10 IU was administered, after which profound hypotension developed, with systolic blood pressure falling to the 60-mmHg range. Repeated phenylephrine 100 µg boluses were administered, and a norepinephrine infusion was started at 0.05 µg/kg/min and titrated up to 0.15 µg/kg/min; however, arterial pressure remained unstable while AF with rapid ventricular response persisted. Dobutamine was initiated at 3 µg/kg/min for inotropic support, and amiodarone was administered as a 150-mg loading dose over 10 minutes for rhythm control. Despite escalation of vasoactive and antiarrhythmic therapy, the ventricular rate remained approximately 190 beats/min,

with ongoing hemodynamic instability. Because pharmacologic control was ineffective, intraoperative TEE was performed to exclude left atrial thrombus before cardioversion. TEE demonstrated severe biventricular dysfunction, severe mitral regurgitation, severe tricuspid regurgitation, and a right atrial thrombus, without definite evidence of left atrial thrombus. After confirming the absence of definite left atrial thrombus, synchronized cardioversion at 100 J was performed; sinus conversion was achieved, accompanied by a partial reduction in vasopressor requirement and improved hemodynamic stability.

Following cardioversion, systolic blood pressure was maintained around 120 mmHg with continuous norepinephrine infusion. After multidisciplinary discussion among anesthesiologists, obstetricians, and the cardiothoracic surgeon, ECMO support was deemed unnecessary. The femoral ECMO introducer sheaths and guidewires were removed, and the patient was transferred to the cardiac care unit under mechanical ventilation for continued management.

6. Postoperative Course

The patient was transferred to the cardiac intensive care unit (ICU) under mechanical ventilation immediately after surgery. Continuous intravenous amiodarone was maintained for rhythm control. Delayed emergence from anesthesia was observed postoperatively. Naloxone was administered as a diagnostic measure to assess whether residual sedative or opioid effects contributed; however, mental status did not normalize immediately. Given the substantial thromboembolic risk, urgent neurologic evaluation, including brain imaging, was performed and revealed no evidence of acute infarction or hemorrhage. The patient's level of consciousness subsequently improved. As planned, therapeutic anticoagulation with intravenous heparin was initiated promptly postpartum. Heparin infusion was started 2 hours after ICU admission with a target activated partial thromboplastin time (aPTT) of 50 - 60 seconds; the baseline aPTT before initiation was 37.4 seconds.

Over the first 24 postoperative hours, hemodynamics progressively deteriorated despite ongoing norepinephrine and inotropic support, requiring up-titration of vasoactive infusions. This culminated in further deterioration of cardiogenic shock with rising serum lactate levels, accompanied by acute kidney injury and worsening metabolic acidosis. Dobutamine was transitioned to dopamine, and vasopressor support was escalated. In the setting of volume overload and metabolic derangements, continuous renal replacement therapy (CRRT) was initiated to facilitate metabolic correction and careful volume management. Following initiation of CRRT and optimized hemodynamic support, the patient's metabolic status gradually improved, and vasopressor requirements decreased over the subsequent days. Amiodarone infusion was discontinued on postoperative day 1, and she was successfully extubated on postoperative day 2.

By postoperative day 4, repeat transthoracic echocardiography demonstrated marked recovery of ventricular function, with improvement of the left ventricular

ejection fraction to 52%. The patient continued to stabilize clinically and was discharged on postoperative day 12 with optimized guideline-directed medical therapy for heart failure. At discharge, medical therapy included carvedilol 18.75 mg twice daily, sacubitril/valsartan 50 mg twice daily, spironolactone 12.5 mg daily, amiodarone 100 mg daily, digoxin 0.25 mg daily, and apixaban 5 mg twice daily. At the 1-month follow-up, atrial fibrillation persisted, with the ventricular rate maintained in the low 100s, and anticoagulation therapy was continued. Although follow-up echocardiographic data were unavailable, NT-proBNP decreased from 733 pg/mL preoperatively to the low 200 pg/mL range postoperatively, suggesting sustained improvement in cardiac status.

7. Neonatal Outcome

The initial cry was weak and cyanosis was noted; the neonatology team initiated positive-pressure ventilation, with improvement in the Apgar score from 6 at 1 minute to 9 at 5 minutes. The neonate was transferred directly to the neonatal intensive care unit for ongoing care. The neonate received high-flow nasal cannula (HFNC) support (FiO₂ 0.21 at 4 L/min) for 2 hours and was subsequently weaned to room air. Due to poor sucking, gavage feeding via a gastric tube was initiated. Urine output and defecation were appropriate. Neonatal echocardiography and cranial ultrasonography were unremarkable except for a patent foramen ovale. On day 12 of life, following maternal discharge, the neonate was transferred to a local neonatal intensive care unit closer to the family's residence for continuity of care. At final discharge, the neonate weighed 2.4 kg.

8. Discussion

Anesthetic management of cesarean delivery in patients with severe left ventricular dysfunction represents one of the most challenging scenarios in obstetric anesthesia. These patients possess markedly reduced cardiac reserve and limited tolerance for abrupt alterations in preload, afterload, heart rate, and contractility [1] [3]. Even modest hemodynamic perturbations during induction of anesthesia, delivery, or uterotonic administration may precipitate acute decompensated heart failure or cardiovascular collapse [5] [6]. In addition, anesthetic interventions directly influence uteroplacental perfusion and neonatal outcome, further complicating clinical decision-making [7] [8].

In the present case, the clinical situation approached a near-prohibitive-risk pregnancy. The patient exhibited NYHA class IV symptoms, severe systemic ventricular dysfunction with an ejection fraction of 25%, persistent AF with rapid ventricular response, and intracardiac thrombus. According to established maternal cardiovascular risk frameworks, including the modified WHO classification and contemporary prediction models such as CARPREG II, she would be categorized as being at extremely high risk for peripartum cardiac events [2] [4]. Importantly, the decision to proceed with delivery was driven not only by obstetric considerations but also by a rapidly worsening maternal cardiac status. Cardiol-

ogy consultants expressed concern that further delay might result in cardiovascular collapse, and cesarean delivery was therefore undertaken as a maternal rescue measure.

Although neuraxial anesthesia is often favored for cesarean delivery in women with cardiac disease, there is no single universally applicable recommendation for choosing neuraxial versus general anesthesia in severe cardiac dysfunction [3] [5] [6] [9]; the optimal approach should be individualized according to patient characteristics and the evolving clinical context. At our institution, epidural anesthesia is typically preferred in cardiac parturients because a carefully titrated epidural may provide relatively stable hemodynamics while avoiding airway manipulation and attenuating sympathetic stress responses [3] [9]. However, neuraxial techniques can also produce clinically significant sympathetic blockade, with abrupt reductions in systemic vascular resistance and preload that may be poorly tolerated in advanced cardiomyopathy [2] [3]; therefore, gradual dosing and meticulous titration are essential. General anesthesia offers advantages such as controlled ventilation and reduced patient distress, but it may be associated with abrupt hemodynamic responses during induction and airway instrumentation and may adversely affect uterine tone, potentially increasing obstetric bleeding risk. In the present case, the planned initiation of systemic heparinization immediately postpartum—particularly if escalation to ECMO became necessary—rendered neuraxial anesthesia unsafe because of the risk of spinal or epidural hematoma [10]. Consequently, general anesthesia was selected despite institutional preference for neuraxial techniques. This decision underscores that anesthetic technique selection in advanced cardiomyopathy must be individualized and incorporate anticoagulation strategy, rather than adhere rigidly to protocol.

A central feature of this case was the presence of AF with rapid ventricular response in the setting of severe systolic dysfunction. Tachycardia reduces diastolic filling time and may substantially impair cardiac output in patients with limited contractile reserve [11]. Because hemodynamic instability persisted despite escalation of vasoactive support and administration of amiodarone, pharmacologic rate or rhythm control was judged insufficient, necessitating synchronized electrical cardioversion. In this context, a delayed pharmacologic response was unacceptable in the face of ongoing shock. Because cardioversion in the presence of left atrial thrombus carries a potential embolic risk, intraoperative TEE was performed by the anesthesiology team to evaluate for left atrial thrombus before proceeding [12] [13]. Although right atrial thrombus was identified, no definite left atrial thrombus was visualized. Given persistent hemodynamic instability and progressive shock, the immediate threat posed by sustained tachyarrhythmia was judged to outweigh the theoretical embolic risk. Real-time TEE was instrumental in guiding this high-stakes decision and highlights the expanding role of advanced perioperative imaging in critically ill obstetric patients.

Another defining element of management was anticipatory ECMO preparedness [14]. Severe ventricular dysfunction combined with uncontrolled tach-

yarrhythmia placed the patient at substantial risk for refractory cardiogenic shock. Rather than waiting for collapse to occur, a structured rescue pathway was established preoperatively. The cardiothoracic surgery team was formally involved in multidisciplinary deliberation; femoral arterial and venous introducer sheaths were placed under sterile conditions to facilitate rapid cannulation if necessary, and the cardiothoracic surgeon remained physically present on standby throughout delivery [14]. Although cannulation was not performed and ECMO was ultimately not required, this proactive preparation significantly reduced escalation time and provided a meaningful safety margin during a period of extreme instability. In large-volume tertiary centers, predefined mechanical support strategies and interdepartmental coordination may be as critical as pharmacologic management in determining outcome.

Interestingly, left ventricular function improved substantially by postoperative day 4, with ejection fraction increasing from 25% to 52%. This rapid recovery, together with uncontrolled hyperthyroidism on admission, suggests that tachycardia-induced cardiomyopathy related to poorly controlled thyrotoxicosis and AF was the most plausible primary mechanism of ventricular dysfunction, although acute pregnancy-related hemodynamic stress may also have aggravated the presentation [15] [16]. Restoration of rate control and delivery-associated reduction in cardiovascular load likely facilitated myocardial recovery. This observation underscores the dynamic and potentially reversible nature of peripartum cardiac dysfunction when timely intervention is achieved.

Finally, this case emphasizes that the management of critically ill parturients extends beyond intraoperative stabilization. The coexistence of AF, intracardiac thrombus, cardioversion, and postpartum hypercoagulability created a substantial thromboembolic risk [17]-[19]. Prompt neurologic evaluation and early therapeutic anticoagulation were therefore essential components of comprehensive care.

In summary, this case demonstrates that successful anesthetic management of emergent cesarean delivery in patients with severe ventricular dysfunction is determined not by a single anesthetic modality but by comprehensive risk stratification, integration of anticoagulation planning into anesthetic decision-making, real-time echocardiographic guidance, anticipatory mechanical circulatory support readiness, and coordinated multidisciplinary execution. In near-catastrophic obstetric cardiac scenarios, outcome depends on structured preparation, rapid escalation capability, and continuous physiologic reassessment.

Informed Consent

Informed consent was obtained from the patient for this case report.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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