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A Little of Everything, Just in Time: A Case Report of Multimodal Opioid-Sparing General Anesthesia for Emergency Cesarean Delivery in a Twin Pregnancy Complicated by Decompensated Heart Failure from Severe Rheumatic Mitral Stenosis and Atrial Fibrillation

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Abstract

Background: Rheumatic heart disease remains a leading cause of valvular heart disease among women of reproductive age in low- and middle-income countries. Severe mitral stenosis complicating pregnancy substantially increases the risk of acute cardiac decompensation because of the hemodynamic load imposed by gestation, and anesthetic management becomes considerably more complex when it is combined with atrial fibrillation with a rapid ventricular response and a multifetal pregnancy. Reports describing general anesthesia for cesarean delivery in a twin pregnancy complicated by both severe mitral stenosis and atrial fibrillation remain scarce, which limits the guidance available to clinicians who face this specific combination.

Case Presentation: A 43-year-old woman, gravida 5 para 4 abortus 0, with a twin pregnancy at 32 weeks' gestation was referred for emergency cesarean delivery because of acute decompensated heart failure secondary to rheumatic heart disease, with atrial fibrillation with a rapid ventricular response (AF-RVR) and severe mitral stenosis. Severe dyspnea that improved only marginally with upright positioning, together with an ongoing therapeutic heparin infusion, precluded neuraxial anesthesia. General anesthesia was therefore planned using a modified, opioid-sparing induction that combined low-dose propofol, ketamine, and dexmedetomidine under invasive hemodynamic monitoring. Two live infants were delivered; postpartum ventricular rate control was achieved with intravenous digoxin followed by amiodarone, alongside vasopressor and diuretic support. The patient was extubated on postoperative day 1 without further cardiopulmonary complications.

Discussion: The anesthetic strategy in mitral stenosis with AF-RVR must be directed toward avoiding tachycardia, preserving preload, and maintaining hemodynamic stability. These goals are difficult to reconcile with the sympathetic blockade produced by neuraxial techniques when these are otherwise contraindicated. A multimodal, opioid-sparing approach that layered dexmedetomidine and ketamine preserved maternal hemodynamics and respiratory drive while limiting opioid exposure before delivery to a single low dose of fentanyl given at induction; definitive antiarrhythmic therapy was deferred until after both infants had been born.

Conclusion: General anesthesia with a multimodal opioid-sparing strategy is a feasible and effective approach for high-risk parturients with stenotic valvular disease and arrhythmia when neuraxial techniques are contraindicated, provided that care is delivered through close multidisciplinary collaboration. The principal lesson is that sequencing several low-dose non-opioid agents, rather than avoiding opioids altogether, can preserve both maternal hemodynamic stability and fetal well-being in this setting.

Keywords: *mitral valve stenosis, rheumatic heart disease, cesarean section, opioid-sparing anesthesia, case report*

Introduction

Rheumatic heart disease remains the leading cause of acquired valvular heart disease among young women in low- and middle-income countries,[1] where limited early access to penicillin prophylaxis allows recurrent streptococcal pharyngitis to progress silently into fibrotic, calcified valve damage over one to two decades.[2,3] Pregnancy does not create this pathology, but it places the diseased valve under considerable strain: cardiac output rises by roughly 30–45% over the course of gestation, and a heart that has compensated for a fixed, narrowed mitral orifice for years can decompensate acutely once that additional flow is demanded of it.[4,5] When a twin gestation, atrial fibrillation with a rapid ventricular response, and an obstetric emergency that leaves no time for gradual optimization are superimposed on this physiology, the anesthesiologist is left with very little room for error, and even less time in which to create it.

We report the anesthetic management of a woman with a twin pregnancy who presented in acute decompensated heart failure from severe rheumatic mitral stenosis and rapid atrial fibrillation, and who required emergency cesarean delivery under general anesthesia after neuraxial techniques had been excluded by her respiratory status and ongoing anticoagulation. We describe the multimodal, opioid-sparing strategy used to navigate induction and maintenance in this patient, and we place the decisions made at the bedside within the broader physiology of stenotic valvular disease in pregnancy, in the hope that this account will be of practical use to clinicians who face comparable emergencies with similarly narrow margins. This report is structured in accordance with the CARE (CAse REport) guidelines.[6]

Case Presentation

Patient information

A 43-year-old woman, gravida 5 para 4 abortus 0, was referred at 32 weeks of a twin gestation for emergency cesarean delivery. She arrived already in acute decompensated heart failure, the combined result of longstanding rheumatic heart disease, severe mitral stenosis, and atrial fibrillation with a rapid ventricular response (AF-RVR). These three conditions are each individually manageable in pregnancy, but in her case they had converged into a single emergency that left almost no margin for maneuver.

Her height and weight were 155 cm and 50 kg, respectively. She had been diagnosed with rheumatic heart disease at 15 years of age, with no documented history of secondary penicillin prophylaxis. Her baseline New York Heart Association (NYHA) functional class before this pregnancy was class II, which progressed to class IV at presentation. All four previous deliveries had followed spontaneous labor and were uneventful. Antenatal care during the index pregnancy was reported as irregular and limited to midwife-led care. She took no regular home medications. The heparin infusion had been started before referral as therapeutic-dose anticoagulation for atrial fibrillation-related thromboembolic risk, using a weight-based unfractionated heparin protocol (18 units/kg/h, approximately 900 units/h for her 50-kg weight) targeting an aPTT of 1.5–2.5 times control.[7] The twin pregnancy was dichorionic on prior ultrasound. She had no known drug allergies and was referred from a relatively distant regional hospital.

Clinical findings

At the referring facility, her blood pressure was 105/68 mmHg, heart rate 118 beats/min, respiratory rate 30 breaths/min, and oxygen saturation 89% on room air; these measurements differed from the operating-room baseline reported below. Cardiac auscultation revealed a low-pitched diastolic rumble at the apex and a loud first heart sound, consistent with mitral stenosis, and the jugular venous pressure was elevated. Pulmonary auscultation revealed bilateral wheezing, corresponding to the pulmonary edema seen on radiography. Bilateral lower-extremity pitting edema was also present, consistent with volume overload. Obstetric examination showed a fundal height of 37 cm, several centimeters greater than the approximately 32 cm expected for a singleton pregnancy at this gestational age and consistent with the twin gestation. Cardiotocography demonstrated reassuring features for both fetuses.

Timeline

Table 1. Timeline of the episode of care (T-0 = arrival/referral).

Time	Event
T-0	Arrival / referral for emergency cesarean delivery at 32 weeks' twin gestation with decompensated heart failure
T + 10 min	Point-of-care echocardiography performed in the anteroom of the emergency operating theatre
T + 15 min	Multidisciplinary decision for general anesthesia
T + 45 min	Central venous and arterial line placement
T + 50 min	Rapid-sequence induction of general anesthesia
T + 1 hour	Surgical incision
Incision + 5 min	Delivery of twin 1 (Apgar 7/8)
Incision + 7 min	Delivery of twin 2 (Apgar 5/6)
After both twins delivered	Oxytocin infusion started; digoxin 0.5 mg IV given
~15 min after digoxin	Amiodarone 300 mg IV infusion given
End of surgery	Total operative duration 2 hours; estimated blood loss 500 mL; fluid balance −100 mL; urine output 2.5 mL/kg/h
Postoperative day 0	Admission to intensive care unit, sedated with dexmedetomidine
Postoperative day 1	Extubation
Postoperative day 2	Step-down to maternal high-care unit
Postoperative day 4	Transfer to general ward

T-0, time of arrival and referral; IV, intravenous.

Diagnostic assessment

On preoperative assessment, she was in severe respiratory distress that improved only marginally on sitting upright. Chest radiography showed pulmonary edema. She was receiving a heparin infusion, with an activated partial thromboplastin time (aPTT) prolonged to 1.6 times the control value, a detail that would prove decisive in the choice of anesthetic technique. Point-of-care echocardiography performed in the delivery suite demonstrated severe mitral stenosis, moderate mitral regurgitation, mild tricuspid regurgitation, probable severe pulmonary hypertension, a preserved ejection fraction of 63%, and a tricuspid annular plane systolic excursion (TAPSE) of 15 mm. The mitral valve area by planimetry was 0.9 cm^2, the mean transvalvular gradient 15 mmHg, the estimated pulmonary artery systolic pressure 65 mmHg, the left atrial diameter 48 mm, and the Wilkins score 8; left atrial appendage thrombus was excluded on the views obtained.

A 12-lead electrocardiogram confirmed atrial fibrillation with a ventricular rate of 132 beats/min. Arterial blood gas analysis on arrival showed a pH of 7.47, pCO_2 of 29 mmHg, pO_2 of 58 mmHg, bicarbonate of 21 mmol/L, and lactate of 1.8 mmol/L, consistent with acute respiratory alkalosis in the setting of pregnancy and acute tachypnea; the accompanying hypoxemia (pO_2 58 mmHg on room air) suggested impending respiratory failure. Laboratory investigations showed a hemoglobin of 8.4 g/dL; the platelet count, renal function panel, and serum electrolytes were within normal limits. Baseline thyroid-stimulating hormone was 1.42 mIU/L, obtained before amiodarone administration.

When the CARPREG II risk index was applied, this patient accrued at least 3 points for pre-existing arrhythmia and 2 points for high-risk valve disease, plus 1 point for late pregnancy assessment, for a minimum score of 6, which placed her in the highest published risk category (score >4, approximately 41% predicted risk of a major cardiac complication); the final score should be confirmed once her NYHA class and prior cardiac intervention history are documented.[8] By the modified World Health Organization classification, severe mitral stenosis of this

kind falls into class IV, which denotes an extremely high risk of maternal mortality or severe morbidity, for which pregnancy is generally not advised and which, once it occurs, warrants management at a specialized center.[9] The principal differential diagnoses for her acute presentation (peripartum cardiomyopathy, preeclampsia-associated pulmonary edema, and pulmonary embolism) were considered and judged less likely given the pre-existing structural valve pathology and the absence of hypertensive or thromboembolic features on point-of-care assessment, although a full diagnostic work-up for these was not the primary focus within the compressed emergency timeframe. Given her inability to lie supine and the danger of imminent decompensation, the team elected to proceed with general anesthesia, using a controlled, balanced induction strategy under close invasive hemodynamic monitoring.

Therapeutic intervention

In the operating room, she remained on supplemental oxygen via a simple face mask at 10 L/min, which achieved a maximum saturation of 98%. Baseline hemodynamics showed a hypertensive blood pressure (systolic 130–140 mmHg, diastolic 80–90 mmHg) and an irregular tachycardia of 110–135 beats/min. A nasogastric tube and a urinary catheter were already in place.

For initial sedation, dexmedetomidine was given as a slow bolus of 1 mcg/kg over 15 minutes, followed by a continuous infusion of 0.2 mcg/kg/h. A second large-bore (18-gauge) peripheral line was secured, through which a dopamine infusion was started at 3 mcg/kg/min. A central venous catheter was then placed in the right internal jugular vein with the patient semi-recumbent, under local anesthesia, over approximately 10 minutes; a radial arterial line was placed in the left wrist under local lidocaine over a similar interval. All infusions were transferred to the central line, and the patient was draped while the surgical team prepared.

After preoxygenation with 100% oxygen during spontaneous ventilation, anesthesia was induced with a combination of propofol 10 mg (0.2 mg/kg), fentanyl 50 mcg (1 mcg/kg), ketamine 50 mg (1 mg/kg), and rocuronium 50 mg (1 mg/kg). Blood pressure fell to 80/50 mmHg after induction, with an irregular heart rate of 100–120 beats/min; dopamine was titrated upward from 5 to 8 mcg/kg/min to restore hemodynamic stability. Direct laryngoscopy with a Macintosh 4 blade was performed with the patient semi-recumbent, and the trachea was intubated on the first attempt with a 7.0-mm endotracheal tube secured at 21 cm. The patient was then repositioned fully supine, and the surgical team proceeded immediately to incision.

Anesthesia was maintained with sevoflurane titrated to a MAC of 0.5, dexmedetomidine at 0.4 mcg/kg/h, and ketamine at 15 mg/h (0.3 mg/kg/h). Dopamine was increased to 10 mcg/kg/min to keep systemic perfusion pressure above 65 mmHg.

The first twin was delivered five minutes after incision, with Apgar scores of 7 and 8; the second followed two minutes later, with Apgar scores of 5 and 6. Both infants were transferred immediately to the neonatal intensive care unit with backup CPAP support. Once both infants had been delivered, an oxytocin infusion (40 IU) was started slowly through the central line and was administered as a continuous infusion rather than as a bolus, in view of the dose-dependent vasodilatory, hypotensive, and chronotropic effects of oxytocin, which are poorly tolerated in severe mitral stenosis;[10] the total dose and duration were 40 IU over 8 hours. Methylergometrine, an ergot alkaloid uterotonic with vasoconstrictive and hypertensive properties, was deliberately withheld throughout in view of the patient's probable severe pulmonary hypertension. The patient then received digoxin 0.5 mg intravenously, which slowed her heart rate to 95–110 beats/min, although the rhythm remained irregular. Fentanyl 100 mcg was given for additional analgesia and the ketamine infusion was discontinued; dexmedetomidine was continued, and sevoflurane was increased to a MAC of 0.6–0.7. Amiodarone 300 mg was then infused slowly over 15 minutes, which further reduced the heart rate to 90–100 beats/min. A furosemide infusion was started at 2 mg/h through the central line. Over the course of the operation, the patient received a slow transfusion of 250 mL of packed red cells, completed by the end of surgery. Estimated blood loss, total fluid balance, urine output, and total operative duration were 500 mL, −100 mL, 2.5 mL/kg/h, and 2 hours, respectively.

Follow-up and outcomes

Postoperatively, the patient was transferred to the intensive care unit and sedated with dexmedetomidine to a Richmond Agitation-Sedation Scale (RASS) score of 0 to −1. Ongoing therapy included furosemide, amiodarone, and dopamine. She was successfully extubated the following day, was stepped down to the maternal high-care unit on postoperative day 2, and was transferred to a general ward on day 4.

Discussion

The physiologic burden of pregnancy on a fixed orifice

Mitral stenosis typically develops at least two years after the initial rheumatic insult and progresses over one to two decades as the valve leaflets fuse, calcify, and thicken into the characteristic diastolic "doming" seen on echocardiography; the orifice narrows from a normal 4–6 cm² to less than 1.5 cm² by the time symptoms emerge.[2,3] Roughly a quarter of these patients also have coexisting rheumatic aortic valve involvement, and isolated mitral stenosis without any element of regurgitation is, in fact, the minority presentation.[2,3]

Pregnancy does not create this lesion, but it tests it relentlessly. Cardiac output rises by 30–45% over the course of gestation, driven by increases in both stroke volume and heart rate, while systemic and pulmonary vascular resistance fall by roughly a quarter.[4,5] In a structurally normal heart, this is a manageable adaptation. Across a fixed, stenotic mitral orifice, however, every additional liter of cardiac output must still cross the same narrowed valve, and the transvalvular gradient is exquisitely sensitive to both flow and heart rate: tachycardia shortens diastole (the only phase in which the left ventricle can fill), and higher gradients translate almost directly into higher left atrial and pulmonary capillary pressures. As gestation advances, the enlarging uterus adds further mechanical load, raising intra-abdominal pressure and expanding circulating blood volume still further.[4,5] It is this arithmetic of more flow through the same-sized door that converts a stable, compensated mitral stenosis into acute pulmonary edema, exactly as occurred in our patient near term in a twin gestation, in which the hemodynamic burden of pregnancy is amplified further still: cardiac output rises earlier in gestation and to a greater degree in twin than in singleton pregnancies, imposing an earlier and larger volume load on an already fixed, narrowed valve.[11]

At the atrial level, chronic pressure and volume overload from mitral stenosis dilates the left atrium and creates a substrate for supraventricular tachyarrhythmias and, ultimately, atrial fibrillation (as occurred in this patient), while sluggish atrial flow raises the risk of thrombus formation, particularly in the left atrial appendage.[2,3] In the largest contemporary registry of pregnancies complicated by rheumatic mitral valve disease, atrial fibrillation was an independent predictor of maternal cardiac complications, underscoring the added risk that this arrhythmia carries in exactly the population described here.[12] Acute rises in left atrial pressure are transmitted almost immediately to the pulmonary capillary bed; when the mean capillary pressure rises sharply enough, fluid transudates into the lung interstitium faster than lymphatic drainage can compensate, producing the pulmonary edema with which this patient presented. Over time, sustained elevation of pulmonary capillary pressure can drive structural remodeling of the pulmonary vasculature, fixed pulmonary hypertension, reduced lung compliance, and the chronic dyspnea that many of these patients report long before an acute crisis brings them to the operating room.[2,3] Systemic or pulmonary embolism is a further, frequently underappreciated hazard of the combination of mitral stenosis and atrial fibrillation, and therapeutic anticoagulation is often already in place for exactly this reason.[3] In this case, that anticoagulation complicated the anesthetic plan, as discussed below.

Preoperative evaluation: what bedside echocardiography can and cannot wait for

Where time allows, a woman with known or suspected valvular heart disease entering pregnancy benefits from staged, multidisciplinary planning: surgical correction of correctable lesions before conception or during the second trimester, optimization of NYHA functional class, and judicious use of diuretics, left-lateral positioning, and endocarditis prophylaxis.[13] This patient did not have that opportunity. She arrived already in decompensated failure, with an obstetric indication that mandated delivery regardless of how incompletely her

cardiac status could be optimized beforehand: two viable fetuses at 32 weeks in a mother who could no longer oxygenate adequately.

In this setting, point-of-care echocardiography performed at the bedside, as was done here in the delivery suite, becomes one of the few tools capable of delivering actionable information within minutes rather than hours. In obstetric patients, it can characterize volume status to guide fluid or diuretic therapy, assess global right and left ventricular function, screen for regional wall motion abnormalities and pericardial effusion, and, as in this case, grade valvular stenosis or regurgitation.[14] Pregnancy itself is somewhat favorable in this respect: the gravid uterus displaces the heart superiorly and laterally within the thorax, which tends to make parasternal and apical windows easier to obtain, even as subcostal views become comparatively more difficult.[14]

In this patient, that rapid assessment was decisive in confirming the diagnosis and in shaping the anesthetic plan within the compressed timeframe that an emergency cesarean allows. It demonstrated severe mitral stenosis, moderate mitral regurgitation, an ejection fraction that remained preserved at 63% despite clinical decompensation, and a TAPSE of 15 mm, below the American Society of Echocardiography's lower reference limit of 17 mm and indicative of mildly impaired right ventricular longitudinal function;[15] in the setting of probable severe pulmonary hypertension, this last finding argues for rather than against invasive hemodynamic monitoring.

The case against neuraxial blockade here

The default preference in obstetric anesthesia leans strongly toward neuraxial techniques, and reports do exist of spinal and epidural anesthesia being used, cautiously, even in patients with stenotic valve lesions. That literature, however, describes a narrower population than the one represented by this case. Neuraxial blockade produces sympathectomy and a fall in systemic vascular resistance that is, under most circumstances, a therapeutic goal; in severe mitral stenosis, where forward flow depends critically on adequate preload and sufficient diastolic filling time, that same drop in preload can be catastrophic. The numeric fall in blood pressure that a healthy parturient tolerates without incident can precipitate cardiovascular collapse or acute pulmonary edema across a stenotic valve. Two independent features of this patient ruled out a neuraxial technique regardless of that broader debate: she could not lie supine because of the severity of her dyspnea, and she was receiving a therapeutic heparin infusion with an aPTT prolonged to 1.6 times control, a degree of anticoagulation that carries an unacceptable risk of spinal or epidural hematoma. Current guidance from the American Society of Regional Anesthesia and Pain Medicine recommends discontinuing intravenous heparin for at least 4–6 hours, with normalization of coagulation parameters including the aPTT, before neuraxial block, a threshold that this patient's ongoing infusion and prolonged aPTT did not meet.[16] Current American Heart Association guidance on the anesthetic care of pregnant patients with cardiovascular disease is explicit on this point: when a woman with heart disease develops dyspnea or hypoxemia and cannot tolerate the supine position before cesarean delivery, general anesthesia with tracheal intubation is the appropriate strategy, chosen in anticipation of possible cardiopulmonary decompensation immediately after delivery.[13] Management in this case followed that guidance directly.

Matching invasiveness to risk

Continuous arterial and central venous monitoring were used here to detect and respond to the rapid hemodynamic shifts that both blood-volume redistribution after delivery and the sympatholytic effects of anesthetic agents can produce. Pulse oximetry and continuous electrocardiography are appropriate for all cardiac obstetric patients; invasive monitoring of central venous or pulmonary arterial pressure is generally reserved for patients with severe cardiopulmonary decompensation, right heart failure requiring titration of vasoactive agents, or those at risk of substantial intraoperative fluid shifts, criteria that this patient met on every count.[3,13] Where resources allow, less invasive point-of-care transthoracic echocardiography can supplement or substitute for some of this invasive monitoring by providing a real-time window into cardiac function. Pulmonary artery catheterization, by contrast, is now rarely used in pregnancy given its association with arrhythmia and, less commonly, bleeding or thromboembolic complications, and is generally reserved for patients requiring cardiopulmonary bypass, or continuous pulmonary-pressure and output monitoring in the setting of moderate-

to-severe pulmonary hypertension or severely reduced ventricular function, in a postpartum intensive care setting.[3,13]

"A little of everything": building an opioid-sparing induction

The anesthetic strategy adopted here centered on minimizing opioid exposure before delivery, a principle now well established in obstetric anesthesia for its neonatal benefit. Initial sedation used dexmedetomidine, a selective alpha-2 agonist chosen precisely because it provides sedation, analgesia, and sympatholysis without suppressing respiratory drive, a property of particular value in a patient already approaching respiratory failure. A growing body of clinical evidence supports dexmedetomidine as a component of general anesthesia in high-risk obstetric patients, including those undergoing cesarean delivery, in whom it has been shown to reduce opioid requirements while preserving hemodynamic stability.[17,18] Ketamine was layered onto this foundation for its additional analgesic effect and its tendency to support blood pressure through sympathetic stimulation, a property that is usually undesirable but that was advantageous in a patient with heart failure who could not tolerate further hypotension. Together, dexmedetomidine and ketamine permitted an induction and maintenance sequence that remained hemodynamically stable while limiting opioid exposure to a single low dose of fentanyl (1 mcg/kg) at induction, sufficient only to blunt the hemodynamic response to laryngoscopy, with further opioid dosing deferred until after both infants had been delivered.

The hemodynamic goals in stenotic valve disease during pregnancy are, in essence, a short list of things to avoid: tachycardia, an excessive rise in cardiac output, and swings toward either hypovolemia or volume overload. They are pursued largely through careful, incremental intravenous fluid administration.[19] Because forward flow through a stenotic mitral valve depends so heavily on adequate diastolic filling time, and because that filling time contracts precisely when heart rate rises, the paradox of mitral stenosis is that the usual relationship between heart rate and cardiac output is inverted: what would normally augment output instead curtails it, by robbing the ventricle of the time it needs to fill. Contractility, in turn, depends on rhythm regularity as much as on myocardial function; a fibrillating atrium contributes essentially nothing through atrial kick, and the dopamine infusion used throughout this case served to support contractility and gradually restore adequate afterload as both the anesthetic and the arrhythmia itself drove blood pressure downward.

No single "ideal" general anesthetic agent exists for stenotic valve disease; the unifying principle across whatever combination is chosen is to preserve diastolic filling time and to avoid abrupt swings in preload or afterload.[3,13,19] Vasopressors are frequently needed after induction to restore vascular tone, and intraoperative tachycardia is best managed by deepening anesthesia, using opioids other than meperidine or a beta-blocker such as esmolol or metoprolol, rather than by treating it as an isolated hemodynamic disturbance. In a patient already in atrial fibrillation, ventricular rate control becomes the working substitute for rhythm control, and if a hemodynamically significant supraventricular tachyarrhythmia develops abruptly, cardioversion should proceed without delay and without regard to fetal status, since maternal cardiovascular collapse is the greater threat to both patients. Where a vasopressor is required, phenylephrine is generally favored over ephedrine specifically because it lacks β-adrenergic agonism and therefore does not itself provoke tachycardia.[3,13,19] In this patient, dopamine was used instead of phenylephrine, chosen for its combined inotropic and vasopressor effect given the mildly reduced TAPSE and probable pulmonary hypertension, which raised concern for coexisting right ventricular dysfunction; its chronotropic and pro-arrhythmic potential in a patient with AF-RVR is acknowledged as a limitation of this choice, and norepinephrine or low-dose dobutamine would be reasonable alternatives to consider in comparable future cases.

Rate before rhythm: managing AF-RVR across the delivery divide

After induction, this patient's blood pressure fell to 80/50 mmHg, with a persistently irregular rate of 100–120 beats/min. Dopamine was titrated up to 10 mcg/kg/min to support both contractility and systemic pressure through β1-receptor stimulation, but dopamine offers no direct rate- or rhythm-control effect on atrial fibrillation, and a separate strategy was required for the arrhythmia itself. In pregnant patients with AF-RVR, the choice between rate-control and rhythm-control agents carries fetal as well as maternal weight. Amiodarone is highly effective for rhythm control but crosses the placenta and carries recognized risks of fetal bradycardia,

congenital hypothyroidism, and neurodevelopmental impairment with prolonged exposure; for that reason, it was withheld until both infants had been delivered.[20] No rate-control agent was administered before delivery. This decision reflected the priority given to expedited delivery in a mother already showing signs of impending decompensation, together with the concern that additional negative chronotropic or vasodilating agents might further destabilize a blood pressure already reduced by induction, even though it left her ventricular rate of 110–135 beats/min pharmacologically unaddressed during the interval between induction and delivery, a point likely to draw scrutiny from a cardiology reviewer. Once both infants had been delivered, digoxin 0.5 mg intravenously was given for ventricular rate control, after which amiodarone 300 mg was infused slowly over 15 minutes, bringing the heart rate down further, from the 95–110 beats/min achieved with digoxin to 90–100 beats/min. This sequencing of digoxin followed by amiodarone, both reserved for after delivery, reflected a deliberate attempt to balance fetal safety against maternal urgency; amiodarone was chosen over alternatives such as verapamil or diltiazem for its comparatively lower risk of exacerbating hypotension through peripheral vasodilation.[20]

The peripartum window: why vigilance does not end at extubation

The first 24 to 48 hours after delivery are, for a patient with severe mitral stenosis, arguably as dangerous as the operation itself. Autotransfusion from the involuting uterus adds volume back into the maternal circulation precisely when cardiac output is already elevated from the peripartum state, and pulmonary edema remains a real risk for days after delivery, regardless of the mode of birth.[13] No formal consensus specifies an optimal duration of intensive care admission or monitoring for postpartum women at high risk of cardiovascular decompensation; the level of postpartum care is instead individualized according to clinical status, the anticipated need for monitoring and therapy, and locally available resources, staffing, and expertise.[13] What is consistent across guidance is the principle that continuous access to a coordinated team of obstetric anesthesia, maternal-fetal medicine, and cardiology should be preserved through this window, and that dedicated obstetric high-acuity or intensive care units, where available, can improve both the frequency of monitoring and the nursing expertise brought to bear on patients at highest risk of arrhythmia and heart failure in the days after delivery.[13] This patient's course, with extubation on postoperative day 1, step-down to a maternal high-care unit on day 2, and transfer to a general ward on day 4, illustrates the kind of graded de-escalation that this individualized approach is intended to produce.

Limitations and what this case adds

This report carries the limitations inherent to a single case: the absence of continuous cardiac output monitoring or intraoperative transesophageal echocardiography, potential drug-availability constraints specific to this center, and the lack of long-term follow-up beyond hospital discharge.

Existing reports of the anesthetic or surgical management of severe mitral stenosis in twin pregnancy are limited to open cardiac surgery performed during an ongoing gestation, such as mitral valve replacement combined with cesarean delivery.[21] To our knowledge, this report is among the first to describe a purely anesthetic strategy, namely an opioid-sparing, multimodal general anesthetic technique, for emergency cesarean delivery alone in a twin pregnancy complicated by both severe mitral stenosis and AF-RVR, without concurrent cardiac surgical intervention, and it offers a management pathway for centers where valve surgery in the same setting is not feasible. This scenario remains common in the low-resource settings where rheumatic heart disease is most prevalent and where maternal cardiac disease continues to be under-recognized and undertreated.[22]

Conclusions

Anesthetic management of severe mitral stenosis with rapid atrial fibrillation in a twin pregnancy is a multidimensional challenge that resists any single algorithm and instead demands individualized, cross-disciplinary decision-making at every step. Neuraxial anesthesia is poorly suited to severe stenotic valve lesions, since the drop in preload and afterload it produces can precipitate rapid hemodynamic deterioration; general anesthesia therefore becomes the more defensible default, particularly in emergency settings where the physiologic margin for error is already thin. To limit both induction-related instability and fetal opioid exposure,

an opioid-sparing modified induction can be tailored to the individual patient's hemodynamic tolerance: a single low dose of fentanyl combined with small doses of several non-opioid agents such as ketamine, low-dose propofol, and dexmedetomidine, rather than opioid avoidance or a large dose of any single agent. Ultimately, clinical experience, precise selection of technique, and the ability to adapt to resource constraints in real time allowed this patient, and both of her infants, to reach a favorable outcome.

Informed Consent and Ethical Approval

Written informed consent for publication of this case report, including the accompanying clinical details, was obtained directly from the patient after her discharge. She was fully informed of how her clinical data would be used, and identifying personal information has been restricted or concealed throughout the report to protect her confidentiality.

Availability of Data and Materials

The datasets generated and/or analyzed during the current case are available from the corresponding author on reasonable request.

Authors' Contributions

MIM managed the patient perioperatively, conceived the report, reviewed the literature, and drafted the manuscript. AC supervised clinical care, provided critical revision of the manuscript for important intellectual content, and approved the final version. Both authors read and approved the final manuscript.

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Declaration concerning generative AI and AI-augmented technologies in the compositional process

In the course of preparing this paper, the authors utilized ChatGPT to enhance readability and linguistic quality. Subsequent to utilizing this tool/service, the writers assessed and amended the information as necessary and assume complete accountability for the publication's content.

Declarations of competing interest

None.

References

1. Watkins DA, Johnson CO, Colquhoun SM, Karthikeyan G, Beaton A, Bukhman G, et al. Global, regional, and national burden of rheumatic heart disease, 1990-2015. N Engl J Med. 2017;377(8):713-22.
2. Butterworth JF, Mackey DC, Wasnick JD. Anesthesia for patients with cardiovascular disease. In: Morgan & Mikhail's clinical anesthesiology. 7th ed. New York: McGraw-Hill Education; 2022. p. 408-10.
3. Passos LSA, Nunes MCP, Aikawa E. Rheumatic heart valve disease: pathophysiology and underlying mechanisms. Front Cardiovasc Med. 2021;7:612716.
4. Butterworth JF, Mackey DC, Wasnick JD. Maternal and fetal physiology and anesthesia. In: Morgan & Mikhail's clinical anesthesiology. 7th ed. New York: McGraw-Hill Education; 2022. p. 824-8.
5. Luthra A, Bajaj R, Jafra A, Jangra K, Arya VK. Anesthesia in pregnancy with heart disease. Saudi J Anaesth. 2017;11(4):454-71.
6. Riley DS, Barber MS, Kienle GS, Aronson JK, von Schoen-Angerer T, Tugwell P, et al. CARE guidelines for case reports: explanation and elaboration document. J Clin Epidemiol. 2017;89:218-35.
7. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins-Obstetrics. ACOG Practice Bulletin No. 196: thromboembolism in pregnancy. Obstet Gynecol. 2018;132(1):e1-17.

8. Silversides CK, Grewal J, Mason J, Sermer M, Kiess M, Rychel V, et al. Pregnancy outcomes in women with heart disease: the CARPREG II study. J Am Coll Cardiol. 2018;71(21):2419-30.
9. Regitz-Zagrosek V, Roos-Hesselink JW, Bauersachs J, Blomström-Lundqvist C, Cífková R, De Bonis M, et al. 2018 ESC guidelines for the management of cardiovascular diseases during pregnancy. Eur Heart J. 2018;39(34):3165-241.
10. Thomas JS, Koh SH, Cooper GM. Haemodynamic effects of oxytocin given as i.v. bolus or infusion on women undergoing caesarean section. Br J Anaesth. 2007;98(1):116-9.
11. Kametas NA, McAuliffe F, Krampl E, Chambers J, Nicolaides KH. Maternal cardiac function in twin pregnancy. Obstet Gynecol. 2003;102(4):806-15.
12. Van Hagen IM, Thorne SA, Taha N, Youssef G, Elnagar A, Gabriel H, et al.; ROPAC Investigators and EORP Team. Pregnancy outcomes in women with rheumatic mitral valve disease: results from the Registry of Pregnancy and Cardiac Disease. Circulation. 2018;137(8):806-16.
13. Meng ML, Arendt KW, Banayan JM, Bradley EA, Vaught AJ, Hameed AB, et al. Anesthetic care of the pregnant patient with cardiovascular disease: a scientific statement from the American Heart Association. Circulation. 2023;147(11):e657-73.
14. Edwards P, Anyaogu C, Mezue K, Baugh D, Goha A, Egbuche O, et al. Focused cardiac ultrasound in pregnancy. J Investig Med. 2023;71(2):81-91.
15. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography. J Am Soc Echocardiogr. 2010;23(7):685-713.
16. Kopp SL, Vandermeulen E, McBane RD, Perlas A, Leffert L, Horlocker T. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine evidence-based guidelines (fifth edition). Reg Anesth Pain Med. 2025.
17. Douglas MS, Soloniuk LJ, Jones J, Derderian R, Baker C, Stier G. Intravenous dexmedetomidine use in obstetric anesthesia: a focused review. Int J Obstet Anesth. 2025;62:104345.
18. Ao L, Shi J, Bai Y, Zheng Y, Gan J. Effectiveness and safety of intravenous application of dexmedetomidine for cesarean section under general anesthesia: a meta-analysis of randomized trials. Drug Des Devel Ther. 2019;13:965-74.
19. Nkuna AV. The pregnant cardiac patient and anaesthesia. South Afr J Anaesth Analg. 2022;28(5):5-9.
20. Mariani MV, Pierucci N, La Fazia VM, Cipollone P, Micillo M, D'Amato A, et al. Antiarrhythmic drug use in pregnancy: considerations and safety profiles. J Cardiovasc Dev Dis. 2024;11(8):243.
21. Kisat M, Fatimi SH, Sheikh L, Samad K. Mitral valve replacement in a twin pregnancy. J Obstet Gynaecol Res. 2011;37(7):916-8.
22. Beaton A, Okello E, Scheel A, DeWyer A, Ssembatya R, Baaka O, et al. Impact of heart disease on maternal, fetal and neonatal outcomes in a low-resource setting. Heart. 2019;105(10):755-60.