

CASE REPORT

Peripartum Pulmonary Complications in High-Risk Cardiac Obstetric Patients: A Case Report

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ABSTRACT

Background: Managing pregnant females with significant cardiac comorbidities, including rheumatic heart disease (RHD) and pulmonary arterial hypertension (PAH), presents unique perioperative challenges. This case highlights the anesthetic management and associated complications in a high-risk obstetric patient with cardiac comorbidity undergoing emergency cesarean section.

Case Presentation: A 22-year-old female (G2P1L1) at 39 weeks and 8 days of gestation with a previous lower segment cesarean section (LSCS), RHD, post-balloon mitral valvotomy, scheduled for emergency cesarean delivery. Preoperative POCUS echocardiography revealed moderate AR, Grade I tricuspid regurgitation (TR), and moderate PAH. Given the significant hemodynamic risk, general anesthesia was selected as the mode of anaesthesia.

The anesthetic induction and intraoperative course were uneventful; however, at the end of the surgery, the patient developed severe desaturation despite adequate ventilation. Bilateral crepitations on auscultation suggested acute pulmonary edema. Patient was administered intravenous furosemide was transferred to the cardiac intensive care unit (ICU) on a ventilator for further management. By the following day, respiratory status improved, with resolution of crepitations. The patient was alert and extubated successfully without further complications.

Conclusion: This case underscores the importance of meticulous anesthetic planning and prompt recognition of perioperative complications in obstetric patients with significant cardiac comorbidities. Acute pulmonary edema can be effectively managed with timely intervention, emphasizing the need for a multidisciplinary approach in high-risk pregnancies.

How to cite this article: Kaur S, Kalal RH, Gogate V. Peripartum Pulmonary Complications in High-Risk Cardiac Obstetric Patients: A Case Report. *Int J Drug Deliv Technol.* 2026;16(36s): 1030-1032. DOI: 10.25258/ijddt.16.36s.121

Source of support: Nil., **Conflict of interest:** None

INTRODUCTION

Cardiovascular disease in pregnancy significantly contributes to maternal morbidity and mortality worldwide (9). Physiological adaptations of pregnancy can exacerbate pre-existing cardiac disease and precipitate acute decompensation, necessitating a multidisciplinary approach. (3).

Valvular heart disease remains a leading cause of maternal mortality (6). Patients with rheumatic heart disease are especially vulnerable as the peripartum period is marked by sudden fluid shifts, fluctuations in systemic vascular resistance, and heightened sympathetic activity. This case illustrates these challenges, in the management of acute pulmonary edema in a parturient with rheumatic valvular heart disease and pulmonary arterial hypertension.

CASE PRESENTATION

Preoperative Assessment

A 22-year-old gravida 2, para 1 (living 1) woman at 39+6 weeks gestation presented in active labor with a history of prior cesarean section with rheumatic heart

disease status post balloon mitral valvotomy. She had received consistent antenatal care and was taking Tab. Torsemide 2.5 mg daily and Tab. Bisoprolol 2.5 mg daily throughout pregnancy.

Upon admission, emergency cesarean delivery was indicated due to labor onset in the context of previous cesarean section. Physical examination revealed hemodynamic stability, with heart rate of 100 beats/min, Blood Pressure of 110/70 mmHg, and oxygen saturation of 98% on room air. General examination showed bilateral pedal edema but no respiratory distress. Cardiovascular examination revealed murmurs consistent with mitral and aortic regurgitation.

Airway evaluation was normal. Routine laboratory studies were unremarkable. Preoperative POCUS echocardiography showed moderate aortic regurgitation with a thickened aortic valve, Grade I tricuspid regurgitation, moderate pulmonary arterial hypertension, and preserved systolic function (EF 60%).

Intraoperative Management

Given the patient's cardiovascular status, general anesthesia was selected to avert hemodynamic

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perturbations associated with neuraxial techniques. This decision was prompted by moderate pulmonary arterial hypertension and concerns about severe hypotension after neuraxial blockade, which could impair uteroplacental perfusion and increase right ventricular strain.

General anaesthesia induced via rapid sequence induction and maintained with inhalation agents and intermittent atracurium. Post-delivery, Oxytocin 10 IU was infused intravenously slowly, followed by 10 IU intramuscularly. Surgical duration approximated one hour with minimal blood loss.

The intraoperative course was uneventful.

Peri-Extubation Acute Decompensation

Towards the end of surgery, profound desaturation ensued (SpO₂ plummeting to 60%). Auscultation disclosed bilateral coarse crepitations, with copious frothy pink secretions in the endotracheal tube, diagnostic of acute pulmonary edema. In view of the acuity, re-induction with thiopentone and atracurium was done to enable controlled ventilation and prevent further compromise.

Intravenous furosemide 60 mg was administered to alleviate pulmonary congestion. With sustained controlled ventilation and diuresis, SpO₂ improved progressively, and secretions diminished markedly. The patient was transferred intubated to the cardiac intensive care unit for further management.

Postoperative Course

In the intensive care unit, controlled ventilation with positive end-expiratory pressure was instituted. Immediate postoperative chest radiography revealed near-complete bilateral opacification, corroborating extensive pulmonary edema.

Over 12–18 hours, diuresis with furosemide and positive-pressure ventilation facilitated rapid resolution. Repeat radiography the subsequent day evidenced substantial pulmonary congestion clearance, mirrored clinically by absent crepitations and stable oxygenation. Postoperative echocardiography revealed moderate mitral regurgitation, mild aortic regurgitation, moderate pulmonary arterial hypertension, and preserved biventricular function, with no acute structural deterioration, supporting a hemodynamic cause of pulmonary edema.

Recovery

Ventilatory weaning commenced the ensuing evening, culminating in successful extubation the following day with sustained oxygenation stability and appropriate responsiveness.

DISCUSSION

This case underscores the importance of rigorous monitoring and expeditious intervention for peripartum pulmonary complications, especially acute pulmonary edema, in obstetric patients with high-risk cardiac

profiles (4).

The postpartum period imposes peak cardiovascular stress, with cardiac output increasing by up to 100% due to uterine involution and autotransfusion from resolving edema. This burden can exacerbate underlying disorders and valvular gradients, and—combined with acute volume overload post-delivery—precipitate pulmonary edema or right ventricular failure, particularly in patients with pre-existing pulmonary hypertension or right heart dilatation. Thus, careful fluid management and hemodynamic stability are essential.

General anesthesia is preferred over neuraxial techniques as it provides a better control of the hemodynamics along with careful drug titration to maintain hemodynamic stability and minimize myocardial depression, a crucial consideration for patients with compromised cardiovascular function (5).

Multiple mechanisms contributed to the acute pulmonary edema. Pre-existing moderate pulmonary arterial hypertension and moderate mitral and aortic regurgitation predisposed the patient to elevated left atrial and pulmonary capillary wedge pressures. The shift from positive-pressure ventilation to spontaneous breathing during extubation suddenly increased venous return and pulmonary blood flow, while extubation stress—via pain and catecholamine release—increased afterload and myocardial demand. These changes precipitated flash pulmonary edema, manifesting as sudden hypoxemia with frothy secretions.

Distinguishing pulmonary edema from other causes of acute respiratory distress—such as negative-pressure edema, acute valvular worsening, stress cardiomyopathy, pulmonary embolism, or hypervolemia—is essential. Postoperative findings such as preserved biventricular function, no new structural changes, and rapid improvement with diuretics and positive-pressure ventilation, confirmed a hemodynamic rather than structural cause. Conservative intraoperative fluid management excluded volume overload.

Quick identification and prompt active intervention prevented worsening of pulmonary edema. Positive-pressure ventilation reduced venous return and lung congestion, while diuretics lowered hydrostatic pulmonary capillary pressures. This case emphasizes tailored anesthetic plans, cautious uterotonic use, and close postoperative monitoring in pregnant women with heart disease, enabling full recovery through timely intervention. Intensive postpartum monitoring is vital during this period of peak cardiovascular stress.

CONCLUSION

This case illustrates the delicate hemodynamic balance in pregnant patients with combined valvular regurgitant lesions and pulmonary arterial hypertension, particularly during the peri-extubation period. Despite stable intraoperative hemodynamics, the abrupt physiological changes during emergence from anesthesia—including the transition from positive-pressure ventilation to

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spontaneous respiration, extubation-associated stress with catecholamine release, and superimposed peripartum volume shifts—precipitated flash pulmonary edema (1,2,7).

Early recognition of respiratory distress, immediate airway control with positive intrathoracic pressure, and timely diuretics proved crucial to the patient's favorable outcome.

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