

CASE REPORT

A Challenge for Anaesthetist: LSCS of a Parturient with Peripartum Cardiomyopathy

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is a rare form of dilated cardiomyopathy presenting as new-onset heart failure in late pregnancy or early postpartum. It is characterized by reduced left ventricular ejection fraction ($\leq 45\%$) and carries significant maternal and fetal risk. Anaesthetic management is challenging due to susceptibility to arrhythmias, pulmonary edema, and hemodynamic instability, with limited evidence to guide optimal techniques.

This report describes two parturients with severe PPCM (ejection fraction 20–27%) requiring caesarean delivery. Both underwent a combined general anaesthesia and epidural approach after multidisciplinary evaluation. Induction with etomidate or propofol and succinylcholine resulted in hypotension requiring norepinephrine support. Maintenance was achieved with oxygen–nitrous oxide, and opioids were administered after delivery. Uterotonics were avoided to prevent abrupt cardiovascular changes. Neonatal outcomes were good, and maternal recovery was uneventful.

These cases demonstrate that a carefully titrated combined anaesthetic technique with meticulous hemodynamic management can facilitate safe delivery in severe PPCM.

KEYWORDS

- Peripartum Cardiomyopathy • Caesarean Section • Anaesthetic Management
- Heart Failure • Obstetric Anaesthesia • Left Ventricular Dysfunction

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INTRODUCTION

Peripartum cardiomyopathy (PPCM) is an uncommon subtype of dilated cardiomyopathy that presents as new-onset heart failure toward the end of pregnancy or within the first five postpartum months.¹ The diagnosis requires exclusion of pre-existing cardiac disease prior to the last month of gestation and elimination of alternative causes such as ischemic, metabolic, or viral cardiomyopathies.¹⁰ Echocardiography typically demonstrates a left ventricular ejection fraction (LVEF) $\leq 45\%$ with reduced fractional shortening.² Although its incidence is low around 0.1% of pregnancies the condition is associated with significant maternal morbidity and mortality.^{3,4}

Physiological changes of pregnancy, including increased cardiac output and decreased systemic vascular resistance, may unmask or exacerbate myocardial dysfunction in susceptible women.⁵ Anaesthetic management is complex due to the risk of arrhythmias, thromboembolism, pulmonary edema, and sudden cardiovascular collapse.⁹ Evidence guiding optimal anaesthetic strategies for obstetric or non-cardiac procedures in PPCM remains scarce.⁶

This report describes two cases of PPCM successfully managed with general anaesthesia supplemented by epidural analgesia for caesarean delivery.

CASE REPORTS

Case 1

A 26-year-old primigravida at 38 weeks presented with grade III dyspnoea for three days. She measured 157 cm and weighed 75.3 kg (BMI 30.5 kg/m²). Her heart rate (HR) was 102/min, blood pressure (BP) 112/78 mmHg, and oxygen saturation 99% on room air. Examination revealed pallor and a systolic ejection murmur; chest auscultation was normal.

Investigations were within normal limits except for hemoglobin 9 g/dL. ECG showed sinus tachycardia. Echocardiography revealed severe global LV hypokinesia, dilated chambers, EF 20–25%, and mild-moderate mitral and tricuspid regurgitation. Obstetric ultrasound showed a live fetus weighing approximately 2.8 kg.

She was commenced on torsemide and carvedilol but developed acute fetal distress within 48 hours, prompting emergency caesarean delivery. After obtaining high-risk consent, a combined general anaesthesia and epidural approach was chosen.

An epidural catheter was inserted at T12–L1, and 20 mL of 0.25% bupivacaine was administered. Following preoxygenation, anaesthesia was induced with etomidate 15 mg and succinylcholine 75 mg IV, and intubation was performed with a 7.0-mm endotracheal tube. Post-induction hypotension (80/60 mmHg) responded to norepinephrine infusion. Anaesthesia was maintained using oxygen nitrous oxide. Fentanyl 100 µg IV was given after delivery. Uterotonics were avoided.

Estimated blood loss was 700 mL. A healthy 2.2-kg neonate with Apgar 9 was delivered. The patient was extubated smoothly and recovered without complications. She was discharged after two weeks on beta-blockers and diuretics.

Case 2

A 28-year-old primigravida at 38 weeks reported one day of grade III dyspnoea. She weighed 80 kg and was 159 cm tall. Her HR was 154/min, BP 170/110 mmHg, RR 24/min, and SpO₂ 93% on room air. Basal crepitations and an ejection systolic murmur were present.

Laboratory investigations were normal. ECG revealed sinus tachycardia. Echocardiography showed global LV hypokinesia, a dilated LV and LA, mild mitral regurgitation, and EF 27%. Ultrasound demonstrated a fetus weighing about 3 kg.

She was treated with lactic acid and labetalol for one week, improving her symptoms. An elective caesarean section was scheduled after obtaining high-risk consent.

An epidural catheter was inserted at T12–L1, followed by administration of 20 mL of 0.25% bupivacaine. After preoxygenation, induction was achieved with propofol 15 mL and succinylcholine 75 mg IV. Intubation was performed with a 7.0-mm tube. Hypotension following induction (90/60 mmHg) required norepinephrine infusion. Maintenance involved oxygen–nitrous oxide with isoflurane.

Estimated blood loss was 600 mL. A 3.2-kg newborn with an Apgar score of 9

was delivered. Postoperative recovery was uneventful, and she was discharged two weeks later on diuretics and beta-blockers.

DISCUSSION

PPCM is a rare yet potentially fatal form of heart failure that emerges late in pregnancy or shortly after delivery.¹¹ Diagnosis requires exclusion of other causes of myocardial impairment such as valvular disease, ischemia, pulmonary embolism, metabolic pathology, or infectious etiologies.¹² Proposed mechanisms include viral myocarditis, autoimmune injury, cytokine-mediated myocardial damage, nutritional deficiencies, and abrupt fluid shifts.¹² Risk factors include multiparity, maternal age >30 years, obesity, hypertension, preeclampsia, malnutrition, and African ancestry.¹²

Symptoms vary from mild exertional dyspnoea to severe congestive failure. Severe dysfunction may manifest as orthopnoea, paroxysmal nocturnal dyspnoea, pulmonary crepitations, murmurs, raised jugular venous pressure, or hepatomegaly.¹²

Management focuses on optimizing preload and afterload, improving contractility, and preventing thromboembolism. Standard therapy includes diuretics, beta-blockers, vasodilators, digoxin, anticoagulation when appropriate, and inotropes for severe heart failure. Delivery planning should be multidisciplinary.^{11,12}

Anaesthetic decisions depend on the urgency of delivery and the patient's NYHA classification.¹¹ Major goals include avoiding myocardial depression, maintaining stable hemodynamics, minimizing tachycardia, and preventing abrupt shifts in systemic vascular resistance.¹⁵ Graded neuraxial techniques may enhance cardiac output by reducing afterload but must be used carefully in patients with markedly impaired LV function.¹⁴

Etomidate is preferred due to its haemodynamic stability. Ketamine increases SVR and is relatively unsuitable. Propofol may depress myocardial function and lower SVR. Volatile agents in high concentrations depress contractility, whereas opioids have minimal cardiovascular impact.⁸

Oxytocin can cause hypotension by lowering SVR, while methylergometrine markedly increases afterload; both require cautious use. Sudden haemodynamic deterioration may

occur post-delivery due to autotransfusion and resolution of aortocaval compression.¹⁴ General anaesthesia is typically preferred when EF <30%, whereas graded neuraxial techniques may be safe when EF is between 35–45%.¹¹

Both patients in this report had severe LV dysfunction but were successfully managed using a combined epidural general anaesthesia technique with meticulous haemodynamic control, restricted fluid administration, appropriate coverage of pain and avoidance of uterotonics. Placement of epidural catheter and giving local anaesthetic intraoperatively helped us in reducing the requirement of excess of iv opioids and its side effects.

CONCLUSION

PPCM is a rare but serious cardiac disorder with high maternal risk. Accurate diagnosis requires excluding other cardiovascular conditions. Anaesthetic management is demanding and must prioritize haemodynamic stability, avoidance of myocardial depressants, and prevention of abrupt preload or afterload changes. Severe LV dysfunction (EF <25%), persistent ventricular impairment, and associated valvular regurgitation are predictors of poorer outcomes.⁵ Multidisciplinary coordination is crucial for achieving favourable maternal and neonatal outcomes.

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