Section: Anaesthesia

Case Series

ANESTHESIA MANAGEMENT OF THE PREGNANT WOMEN WITH CARDIOMYOPATHY UNDERGOING CAESAREAN SECTION – A CASE SERIES

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ABSTRACT

Background: Cardiomyopathy during pregnancy is associated with significant maternal and fetal morbidity because the physiological cardiovascular changes of pregnancy may precipitate heart failure, arrhythmias, and haemodynamic instability. Anaesthetic management during caesarean section requires an individualized approach based on the underlying pathology and cardiac function. This case series highlights the perioperative anaesthetic management of parturients with cardiomyopathies undergoing caesarean delivery. **Case Series:** Four pregnant women with cardiomyopathy underwent caesarean section: two with peripartum cardiomyopathy (PPCM), one with dilated cardiomyopathy (DCMP), and one with hypertrophic obstructive cardiomyopathy (HOCM). The PPCM patient (LVEF 25%) required emergency caesarean section following failed labour and uterine rupture, managed successfully with extension of epidural anaesthesia, invasive monitoring, vasopressor support, blood transfusion, and intensive postoperative care. The other PPCM patient (LVEF 35–40%) underwent elective caesarean section under graded epidural anaesthesia without complications. The patient with DCMP (LVEF 30%) received combined spinal epidural anaesthesia with invasive haemodynamic monitoring, careful fluid therapy, and phenylephrine support, resulting in stable intraoperative and postoperative recovery. HOCM patient had severe left ventricular outflow tract obstruction, underwent general anaesthesia with etomidate-based induction and controlled haemodynamic management, achieving an uneventful perioperative course. All patients recovered successfully, and neonatal outcomes were favourable. **Conclusion:** Anaesthetic management of pregnant women with cardiomyopathy should be individualized according to the underlying cardiac pathology and haemodynamic status. Graded neuraxial techniques can be safely employed in selected patients with systolic dysfunction, whereas general anaesthesia may be preferable in severe HOCM. Multidisciplinary planning, judicious fluid therapy, appropriate vasoactive support and perioperative and postoperative vigilant monitoring are essential for optimizing maternal and neonatal outcomes.

INTRODUCTION

Cardiovascular disease is an increasingly important cause of maternal morbidity and mortality worldwide, with cardiomyopathies representing a particularly high-risk group during pregnancy. Although uncommon, there is significant association of maternal and fetal complications with cardiomyopathy as the physiological cardiovascular adaptations of pregnancy may unmask or worsen ventricular dysfunction and may precipitate heart failure or arrhythmias in susceptible women.[1,2]

The principal cardiomyopathies encountered in obstetric anaesthesia include dilated

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cardiomyopathy (DCMP), peripartum cardiomyopathy (PPCM), and hypertrophic obstructive cardiomyopathy (HOCM). Each has distinct pathophysiological features but shares the common challenge of maintaining maternal haemodynamic stability while ensuring adequate uteroplacental perfusion during delivery.[1,3]

DCMP is characterized by ventricular chamber dilatation and impaired systolic function, resulting in reduced left ventricular ejection fraction and diminished cardiac reserve. Pregnancy in women with DCMP is associated with increased risks of heart failure, malignant arrhythmias, thromboembolic events, and maternal mortality, particularly when the ejection fraction is below 35–40% or the patient is symptomatic (NYHA class III–IV).[2,4] The primary anaesthetic goals are maintenance of myocardial contractility, avoidance of fluid overload, optimization of preload, reduction of afterload, maintenance of hemodynamics and preservation of uteroplacental blood flow.

PPCM is a syndrome with symptoms of heart failure and signs of left ventricular systolic dysfunction, manifesting between the last month of pregnancy and the first 5 months postpartum in women without previously recognized heart disease.[5] It is defined by left ventricular systolic dysfunction, usually with an ejection fraction below 45%, in the absence of another identifiable cause. PPCM remains an important cause of maternal mortality worldwide, and the peripartum period carries a substantial risk of acute pulmonary oedema, cardiogenic shock, ventricular arrhythmias, and thromboembolic complications, making meticulous perioperative management essential.[6,7]

HOCM is an inherited autosomal dominant cardiac disorder characterised by asymmetrical hypertrophy of the ventricular septum with left ventricular outflow tract (LVOT) obstruction. The incidence among pregnant women is around 0.1%–0.5%.8 Although many patients remain asymptomatic, severe cases can present with significant symptoms and carry an increased risk of sudden death due to ventricular arrhythmias. In pregnancy, physiological changes such as increased cardiac output, blood volume, decreased systemic vascular resistance, reduced venous return, and labour‐induced tachycardia pose additional challenges, aggravating LVOT obstruction. For these reasons, anaesthetic management focuses on maintaining sinus rhythm, adequate preload, stable afterload, avoidance of sympathetic stimulation, and prevention of tachycardia.[1,3]

Caesarean section presents unique haemodynamic challenges in women with cardiomyopathy because induction of anaesthesia, sympathetic blockade, surgical blood loss, uterine autotransfusion after delivery, and administration of uterotonic agents may produce rapid circulatory changes. The choice between neuraxial and general anaesthesia remains individualized according to the severity of ventricular dysfunction, haemodynamic status, anticoagulation status, and obstetric indications. Carefully titrated epidural or combined spinal–epidural anaesthesia may provide satisfactory analgesia while minimizing abrupt haemodynamic changes, whereas general anaesthesia may be preferred in patients with severe heart failure, respiratory compromise, or marked haemodynamic instability.[1,9]

Successful perioperative management requires comprehensive preoperative assessment, judicious fluid therapy, appropriate use of vasoactive drugs, and close collaboration among obstetricians, cardiologists, anaesthesiologists, intensivists, and neonatologists. Contemporary guidelines recommend management by a multidisciplinary team for women with significant cardiomyopathy to optimize maternal and fetal outcomes.[1,2]

Despite advances in obstetric anaesthesia and cardiac care, evidence regarding the optimal anaesthetic management of pregnant women with cardiomyopathy remains limited, with most published data derived from case reports and small case series. This case series describes the perioperative anaesthetic management of patients with DCMP, PPCM ,HOCM undergoing caesarean section, highlighting the challenges encountered, anaesthetic techniques employed, haemodynamic management and maternal outcome.

Case 1

A 39-year-old G3P2L2 woman at 36 weeks gestation presented with progressive dyspnoea on exertion, fatigue, low-grade fever, cough with haemoptysis, and bilateral pedal oedema of five days duration. On examination, her heart rate was 140 beats/min, respiratory rate 26 breaths/min, blood pressure 126/86 mmHg, and oxygen saturation 90% on room air. She had elevated jugular venous pressure, bilateral basal crepitations, an S3 gallop, and bilateral pedal oedema. Electrocardiography showed sinus tachycardia with ST-T changes, while chest radiography demonstrated cardiomegaly. Transthoracic echocardiography revealed dilatation of the left atrium and left ventricle, severe left ventricular systolic dysfunction, moderate mitral and tricuspid regurgitation, and a left ventricular ejection fraction (LVEF) of 25%, confirming the diagnosis of peripartum cardiomyopathy. She was advised Tablet Digoxin 0.25 mg OD, Inj . Furosemide 40 mg iv stat, and oxygen supplementation via face mask in propped up position. Her previous pregnancies had been uneventful. There was no significant past medical history, allergy to any drug. Laboratory investigations were with in normal limits.

As the patient was in active labour, labour analgesia was planned in operation theatre with standard ASA monitoring, combined spinal epidural (CSE) was instituted at L3–L4 intervertebral space, using intrathecal 0.5 mL of 0.5% hyperbaric bupivacaine with fentanyl 25 μg, followed by intermittent epidural top-ups of 0.0625% bupivacaine with fentanyl (2 μg/mL). Continuous maternal and fetal

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monitoring was maintained in the high-dependency unit.

After four hours, emergency lower segment caesarean section (LSCS) was planned in view of non progress of labor. Surgical anaesthesia was achieved by extending the epidural block with 8 mL of 0.5% bupivacaine administered in graded doses, producing a sensory block up to the T6 dermatome. Immediately before skin incision, loss of uterine contour suggested uterine rupture, necessitating urgent surgical intervention. Right internal jugular vein and left radial artery were cannulated for invasive monitoring . Estimated intraoperative blood loss was approximately 1.4 L and was managed with balanced crystalloids, colloids, packed red blood cell transfusion, intermittent iv phenylephrine boluses, and subsequently a noradrenaline infusion for persistent hypotension. Injection Syntocin 10 units slow iv infusion in 100 ml normal saline was given after delivery of the baby The patient remained conscious throughout the procedure and was transferred to the cardiac care unit on oxygen therapy and vasopressor support for further monitoring. Epidural analgesia was continued postoperatively, and intravenous furosemide 5 mg was administered. Noradrenaline infusion was discontinued on postoperative day 1. The patient was shifted to the ward after five days and discharged on postoperative day 15 on Tab Torsemide 10 mg once daily and metoprolol succinate 25 mg once daily. Follow-up echocardiography demonstrated improvement in LVEF to 30–35%.

Case 2

A term G2P1L1 woman with a previous history of peripartum cardiomyopathy diagnosed on the second postoperative day following her previous delivery presented for elective caesarean section. She complained of dyspnoea on admission, bilateral pedal edema. Her respiratory rate was 26 breaths/min, heart rate was 125 beats/min , blood pressure was 138/74 mmHg, and oxygen saturation was 92–94% on room air. Chest examination revealed bilaterally diminished breath sounds at bases. Electrocardiography demonstrated sinus tachycardia. Echocardiography showed global left ventricular hypokinesia with an LVEF of 35–40% and trivial mitral and aortic regurgitation. Laboratory investigations were within normal limits.The patient was optimized preoperatively with intravenous furosemide 40 mg and ivabradine 5 mg once daily. She was previously taking Tab Furosemide 10 mg once daily and Tab Metoprolol 25 mg once daily.

Following optimization, patient was planned for elective LSCS was performed under graded epidural anaesthesia at L3-4 intervertebral space. Surgical anaesthesia was achieved by extending the epidural block with 10 mL of 0.75% Ropivacaine administered in graded doses, producing a sensory block up to the T6 dermatome. with continuous electrocardiographic, pulse oximetry, and non invasive blood pressure haemodynamic monitoring. Ten international units of Inj oxytocin to facilitate uterine contraction was infused slowly after delivery of baby. A total of 800 ml of lactated Ringer's solution was administered (including the 200 ml administered before anesthesia) Anaesthesia and surgery proceeded uneventfully without significant haemodynamic instability. The postoperative period was similarly uneventful, patient recovered without any cardiac or obstetric complications and was discharged on day 6 .

Case 3

A 24-year-old primigravida at 37 weeks gestation, a known case of dilated cardiomyopathy diagnosed two years previously, was scheduled for elective LSCS because of severe cardiac disease. She had New York Heart Association (NYHA) class III dyspnoea and was receiving Tab Carvedilol 3.125 mg once daily and Tab Digoxin 0.25 mg once daily. There was no history of alcohol abuse, viral myocarditis, or β-adrenergic agonist use.

Preoperative examination revealed a heart rate of 102 beats/min, blood pressure of 108/74 mmHg, respiratory rate of 22 breaths/min, bilateral pitting pedal oedema, parasternal heave, systolic thrill, and an ejection systolic murmur. The airway assessment was modified mallampati grading II. Electrocardiography showed left ventricular hypertrophy. Echocardiography demonstrated global left ventricular hypokinesia, severe systolic dysfunction with an LVEF of 30%, mild mitral regurgitation, and left ventricular end-diastolic dilatation. Laboratory investigations were within normal limits except for mild anaemia (haemoglobin 9.8 g/dL). Cardiology consultation confirmed DCMP, and Torsemide 10 mg once daily was added.

After obtaining high-risk informed consent, combined spinal-epidural anaesthesia was planned. Standard monitoring was instituted, followed by insertion of a right internal jugular central venous catheter and a left radial arterial catheter for invasive blood pressure monitoring. An epidural catheter was inserted at the L2–L3 interspace and confirmed with a lignocaine test dose. Intrathecal fentanyl 25 μg was administered at the L3–L4 intervertebral space, followed by slow epidural administration of 10 mL of 0.75% ropivacaine over 10 minutes, achieving a T6 sensory block.

Transient hypotension (86/60 mmHg) following epidural administration was successfully treated with intermittent IV phenylephrine boluses (50–100 μg). Haemodynamics remained stable thereafter. A healthy female infant weighing 2.6 kg was delivered with Apgar scores of 9 and 10 at one and five minutes, respectively. Oxytocin was administered as a 2.5 IU slow intravenous bolus followed by infusion at 10 IU/h after cord clamping. Fluid administration was carefully titrated using invasive monitoring, maintaining central venous pressure between 8 and 12 cmH_2 O. Estimated blood loss

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was 700 mL. Patient was shifted to CCU for further monitoring.

Postoperative analgesia consisted of epidural 0.2% ropivacaine with fentanyl 50 µg. Recovery was uneventful. The postoperative echocardiogram showed persistent LVEF of 35%. The patient was discharged on postoperative day 10 on tablet enalapril, aspirin, carvedilol, and torsemide.

Case 4

A 29-year-old primigravida at 36 weeks and 5 days gestation with hypertrophic obstructive cardiomyopathy underwent emergency LSCS because of fetal bradycardia on non-stress testing. She had experienced palpitations during early pregnancy and had been diagnosed with HOCM during the third month of gestation. Bisoprolol 2.5 mg once daily had been prescribed but was discontinued one week before surgery because of hypotension. She had no family history of HOCM. On admission, she was asymptomatic . Heart rate was 106 beats/min, blood pressure 88/62 mmHg, and oxygen saturation 99% on room air. Electrocardiography showed left ventricular hypertrophy. Echocardiography demonstrated asymmetric septal hypertrophy with systolic anterior motion of the mitral valve, left ventricular outflow tract (LVOT) gradient of 90 mmHg, mild mitral regurgitation, and preserved LVEF of 60%. Laboratory investigations were within normal limits. After obtaining informed high-risk consent, general anaesthesia was planned to avoid sudden reductions in preload and systemic vascular resistance. Standard monitoring was instituted, including invasive arterial blood pressure. Iv line secured and ringer's lactate infusion was started. Following intravenous lignocaine 80 mg, rapid sequence induction was performed using etomidate 18 mg and succinylcholine 75 mg. The trachea was intubated with a 7.0-mm cuffed endotracheal tube. Anaesthesia was maintained with 50% oxygen and 50% air with sevoflurane (MAC 0.5–0.8) and Inj vecuronium 5mg .Following delivery of the baby, oxytocin infusion was started at 10 IU/h, and fentanyl 100 µg was administered intravenously.surgery remained uneventful Neuromuscular blockade was reversed with neostigmine and glycopyrrolate, and the patient was extubated after fulfilling standard extubation criteria. Bilateral ultrasound-guided transversus abdominis plane (TAP) blocks were performed for postoperative analgesia. The intraoperative and postoperative courses were uneventful, with stable haemodynamics and no cardiac complications.

DISCUSSION

Pregnancy in women with cardiomyopathy remains a major anaesthetic challenge because the physiological increase in circulating blood volume, cardiac output, and heart rate may precipitate decompensated heart failure, arrhythmias, and maternal mortality. The principal objective of anaesthetic management is to maintain cardiovascular stability while ensuring adequate uteroplacental perfusion and avoiding further deterioration in ventricular function. As demonstrated in our series, successful outcomes require meticulous preoperative optimisation, individualized anaesthetic planning, invasive monitoring when indicated, and close multidisciplinary approach, consistent with contemporary guideline recommendations and prospective studies evaluating cardiovascular disease in pregnancy.[1,2,11]

The choice of anaesthetic technique in patients with cardiomyopathy remains controversial and should be tailored to the underlying pathology, ventricular function, urgency of delivery, and haemodynamic status rather than following a universal approach.[1,3,9] In our series, neuraxial anaesthesia was successfully employed in three patients with impaired systolic function (two patients with PPCM and one patients with DCMP), whereas general anaesthesia was selected for the patient with HOCM because of severe dynamic left ventricular outflow tract obstruction.

Both patients with peripartum cardiomyopathy had significant left ventricular systolic dysfunction but differed in clinical presentation. The first patient presented with acute decompensated heart failure (LVEF 25%) during labour and subsequently required emergency caesarean delivery complicated by uterine rupture and massive blood loss. Despite these challenges, extension of labour epidural analgesia for surgical anaesthesia allowed gradual sympathetic blockade and avoidance of abrupt haemodynamic changes. Careful titration of fluids, prompt vasopressor support with phenylephrine and noradrenaline, blood transfusion, and postoperative intensive cardiac care resulted in favourable maternal recovery with improvement in ventricular function during follow-up. In contrast, the second patient, with previously diagnosed PPCM and relatively preserved functional status (LVEF 35–40%), underwent elective caesarean section under graded epidural anaesthesia without haemodynamic instability or postoperative complications. These contrasting presentations illustrate the broad clinical spectrum of PPCM and highlight the importance of tailoring perioperative management according to disease severity rather than diagnosis alone. Similar successful outcomes have been reported in previous PPCM case series.[2,5,6,7,10]

The patient with dilated cardiomyopathy also demonstrated that severe systolic dysfunction is not an absolute contraindication to neuraxial anaesthesia when administered cautiously. Combined spinal-epidural anaesthesia using intrathecal opioid with incremental epidural local anaesthetic dosing provided adequate surgical anaesthesia while minimizing sudden reductions in systemic vascular resistance. Invasive arterial and central venous pressure monitoring facilitated careful fluid

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administration and early recognition of hypotension, which responded to small phenylephrine boluses. Judicious oxytocin administration and postoperative epidural analgesia further minimized haemodynamic fluctuations. These findings support previous reports suggesting that titrated neuraxial techniques may provide excellent haemodynamic control in patients with severe ventricular dysfunction when accompanied by vigilant monitoring and appropriate vasoactive support.[2,3,4,9]

Anaesthetic management in hypertrophic obstructive cardiomyopathy differs fundamentally from that of systolic cardiomyopathies because reductions in preload or afterload, tachycardia, and increased myocardial contractility may exacerbate left ventricular outflow tract obstruction.[1,3] Although neuraxial anaesthesia has been successfully described in selected HOCM patients, our patient presented with a severe resting LVOT gradient of 90 mmHg and baseline hypotension, making maintenance of preload and systemic vascular resistance particularly important. General anaesthesia with etomidate-based induction allowed controlled haemodynamic conditions while avoiding abrupt sympathetic blockade. Careful use of volatile anaesthesia, avoidance of tachycardia, slow oxytocin infusion, and effective postoperative analgesia contributed to an uneventful perioperative course. Our management is consistent with previously published reports emphasizing preservation of preload, systemic vascular resistance and sinus rhythm as the cornerstone of anaesthetic management in HOCM undergoing caesarean delivery.[1,3,8]

Several common management principles emerged across all four cases. Comprehensive preoperative echocardiographic assessment guided risk stratification and anaesthetic planning. Fluid therapy was individualized and conservative to avoid both hypovolaemia and pulmonary oedema. Vasopressors were administered promptly to maintain coronary and uteroplacental perfusion, while oxytocin was given as slow, low-dose infusion to minimize cardiovascular instability. Postoperative monitoring in a high-dependency or cardiac care setting permitted early detection and treatment of haemodynamic deterioration, which is particularly important because significant autotransfusion following uterine contraction may precipitate heart failure during the immediate postpartum period.[1,2,11]

Our experience reinforces the importance of multidisciplinary management involving obstetricians, cardiologists, anaesthesiologists, intensivists, and neonatologists. Despite severe cardiac disease in several patients, favourable maternal and neonatal outcomes were achieved through coordinated perioperative care and individualized anaesthetic strategies, findings that are comparable with previously published case series of cardiomyopathy in pregnancy.[2,5,10,11]

Despite the differing pathophysiology of PPCM, DCM and HOCM, several common principles emerged from our experience. Thorough preoperative evaluation with echocardiography, multidisciplinary planning, individualized selection of anaesthetic technique, cautious fluid administration, appropriate use of vasopressors, slow administration of uterotonics and close postoperative monitoring contributed to favourable maternal and neonatal outcomes in all four patients. Our findings reinforce that no single anaesthetic technique is universally superior; instead, understanding the underlying pathophysiology and tailoring perioperative management to the individual patient remain the key determinants of successful outcomes.

The limitations of this series include the small sample size and heterogeneity of cardiomyopathy subtypes; however, given the rarity of these conditions in pregnancy, carefully documented case series continue to provide valuable clinical insight for perioperative management of such patients.

CONCLUSION

In conclusion, successful anaesthetic management of pregnant women with cardiomyopathy requires an individualized approach based on the underlying cardiac pathology and functional status. Graded neuraxial anaesthesia appears to be a safe and effective option in haemodynamically stable patients with DCMP or PPCM, whereas general anaesthesia may be preferable in selected patients with severe HOCM or when neuraxial techniques are contraindicated. Meticulous haemodynamic management, multidisciplinary approach including obstetrician, anaesthesiologist, cardiologist, critical care intensivist, neonatologist and intensive postoperative surveillance remain fundamental to optimizing maternal and neonatal outcomes.

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