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Anesthesia Management of Cesarean Section in Women with Peripartum Cardiomyopathy: A Case Series

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ABSTRACT

Introduction: Peripartum cardiomyopathy (PPCM) is a rare but serious heart condition that occurs during late pregnancy or within the first few months after delivery. It can lead to significant maternal and fetal morbidity and mortality. Anesthetic management of PPCM patients undergoing cesarean section (Csection) is challenging due to the hemodynamic instability and potential for complications. Case presentation: This case series describes the anesthetic management of four women with PPCM undergoing C-section. Various anesthetic techniques were employed, including combined spinal-epidural (CSE) and epidural anesthesia, with careful monitoring and individualized management strategies. Case 1 present a 34-year-old female with mild mitral regurgitation, mild tricuspid regurgitation, mild pulmonary regurgitation, intermediate probability pulmonary hypertension, hypertensive heart failure, and obesity underwent C-section under CSE anesthesia with ropivacaine and bupivacaine. Case 2 present a 26-year-old female with PPCM and mild mitral regurgitation underwent C-section under CSE anesthesia with ropivacaine and bupivacaine. Case 3 present a 26-year-old female with PPCM, thrombocytosis, and hypoalbuminemia underwent C-section under epidural anesthesia with ropivacaine and fentanyl. Case 4 present a 30-year-old female with PPCM, marginal placenta previa, uterine myoma, and severe myopia underwent Csection under epidural anesthesia with ropivacaine and fentanyl. Conclusion: Regional anesthesia, particularly CSE and epidural techniques, appears to be a safe and effective anesthetic approach for C-sections in women with PPCM. Meticulous hemodynamic monitoring and individualized management are crucial for successful outcomes.

1. Introduction

Peripartum cardiomyopathy (PPCM) is a rare and often devastating cardiac condition that affects women during the peripartum period, typically manifesting in the final month of pregnancy or within five months following delivery. It is characterized by the development of left ventricular systolic dysfunction and heart failure in women with no prior history of cardiac disease. The incidence of PPCM varies geographically, with reported rates ranging from 1 in 1000 live births to 1 in 3000 live births in the United States. Despite increasing awareness and research efforts, the precise

etiology of PPCM remains elusive. However, several factors have been implicated in its pathogenesis, including; Genetic Predisposition: Studies have identified genetic variants that may increase susceptibility to PPCM, suggesting a hereditary component in some cases; Inflammation and Immune Dysregulation: Elevated levels of inflammatory markers and autoantibodies against cardiac proteins have been observed in PPCM patients, suggesting a potential role for immune system dysfunction in the development of the disease; Hormonal Changes: The dramatic hormonal shifts that occur during pregnancy and the

postpartum period may contribute to the development of PPCM in susceptible individuals; Oxidative Stress: Increased oxidative stress has been implicated in the pathogenesis of PPCM, potentially leading to myocardial damage and dysfunction; Other Factors: Additional factors that may play a role in PPCM development include nutritional deficiencies, infections, and preeclampsia.¹⁻³

The clinical presentation of PPCM can vary, but common symptoms include fatigue, shortness of breath, edema, and palpitations. The diagnosis is typically confirmed through echocardiography, which reveals left ventricular systolic dysfunction with an ejection fraction (EF) of less than 45%. PPCM poses significant challenges for anesthesiologists due to the potential for hemodynamic instability and lifethreatening complications such as pulmonary edema, cardiogenic shock, arrhythmias, and thromboembolism. Anesthetic management of women with PPCM undergoing cesarean section requires a comprehensive understanding of the pathophysiology of the disease, meticulous preoperative assessment, and careful intraoperative and postoperative monitoring. anesthetic The primary goals of management in this setting are to; Maintain Hemodynamic Stability: This involves optimizing preload, afterload, and contractility to ensure adequate cardiac output and tissue perfusion; Ensure Adequate Oxygenation: Preventing hypoxemia is crucial in PPCM patients, as it can exacerbate cardiac dysfunction and lead to complications; Provide Effective Analgesia: Pain management is essential to minimize stress and sympathetic activation, which can worsen hemodynamic instability; Minimize Risk Complications: Careful monitoring and prompt intervention are necessary to prevent and manage potential complications such as pulmonary edema, arrhythmias, and thromboembolism.4-6

The choice of anesthetic technique for cesarean section in women with PPCM is a critical decision that should be individualized based on the patient's clinical condition, preferences, and the anesthesiologist's expertise. Regional anesthesia, including spinal, epidural, and combined spinal-epidural (CSE) techniques, is generally preferred over general

anesthesia in this population. Regional anesthesia offers several advantages, including; Hemodynamic Stability: By avoiding the myocardial depressant effects of general anesthetics and reducing afterload, regional anesthesia can help maintain hemodynamic stability in PPCM patients; Rapid Recovery: Regional anesthesia allows for a faster recovery and earlier ambulation, which can reduce the risk of thromboembolic complications; Avoidance of Airway Manipulation: In patients with potential for pulmonary edema, regional anesthesia avoids the risks associated with airway manipulation and intubation. However, regional anesthesia is not without its challenges in PPCM patients. Hypotension is a common side effect, and careful fluid management and vasopressor support may be required. Additionally, the presence of coagulopathy or thrombocytopenia may increase the risk of bleeding complications with regional techniques.^{7,8}

General anesthesia may be considered in certain situations, such as when regional anesthesia is contraindicated or when the patient's hemodynamic instability precludes its use. However, general anesthesia requires careful titration of anesthetic avoid myocardial depression agents to hemodynamic fluctuations. In this case series, we present the anesthetic management of four women with PPCM undergoing cesarean section at our institution. We describe the clinical presentation, anesthetic considerations, and outcomes of each case, highlighting the importance of individualized management and multidisciplinary collaboration in optimizing care for this high-risk patient population.9,10 Our aim is to contribute to the growing body of literature on anesthetic management of PPCM and provide insights that may help guide clinical practice in this challenging area.

2. Case Presentation

This case series describes the anesthetic management of four women diagnosed with peripartum cardiomyopathy (PPCM) who underwent cesarean section. Each case highlights the unique challenges and considerations involved in providing safe and effective anesthesia for this high-risk patient population.

A 34-year-old female patient, gravida 3, para 2, with a history of two prior cesarean sections, presented at 32-34 weeks gestation with a complex medical history. Her diagnoses included mild mitral regurgitation, mild tricuspid regurgitation, mild pulmonary regurgitation, intermediate probability pulmonary hypertension, hypertensive heart failure with a biplane left ventricular ejection fraction (LVEF) of 27%, chronic hypertension, class II obesity, and a fetal diagnosis of ventricular septal defect. She also reported a history of uncontrolled hypertension for the past nine years, bronchial asthma, and heart disease for the past three years. The patient's presenting complaints included a cough, cold, and shortness of breath that worsened when lying supine and with mild physical activity such as bathing. She reported improvement in her symptoms with rest, furosemide medication, and sleeping with two pillows. She denied fever but reported fatigue. Her current medications included furosemide 1x20mg, adalat oros 1x30mg, methyldopa 3x500mg, and bisoprolol 1.25mg.

On physical examination, the patient was alert and oriented with a Glasgow Coma Scale (GCS) score of E4M6V5. Her vital signs were as follows: blood pressure 139/76 mmHg, pulse 105 beats per minute, respiratory rate 26 breaths per minute, weight 120 kg, height 160 cm, and oxygen saturation (SpO₂) 97% on room air. Her airway was patent. Examination of her eyes revealed no anemia or icterus. Chest auscultation revealed rhonchi in all lung fields. Abdominal examination was unremarkable with no distension, fundal height of 27 cm, cephalic presentation, estimated fetal weight of 2170 grams, fetal heart rate of 156 beats per minute, and no uterine contractions. Examination of her extremities revealed no edema. The STOP-BANG assessment for obstructive sleep apnea (OSA) revealed a score of 5, indicating a high risk.

Laboratory investigations revealed the following: hemoglobin 9.5 mg/dL, leukocytes 8,910/mm³, platelets 350,000/mm³, activated partial thromboplastin time (APTT) 27.30 seconds, prothrombin time (PT) 9.50 seconds, sodium 139 mmol/L, potassium 3.71 mmol/L, chloride 106 mmol/L, urea 11.8 mg/dL, creatinine 0.82 mg/dL,

aspartate aminotransferase (SGOT) 17 U/L, alanine aminotransferase (SGPT) 12 U/L, albumin 3.49 mg/dL, and blood glucose 119 mg/dL. Electrocardiography (ECG) showed sinus rhythm at 106 beats per minute and left axis deviation (LAD). Echocardiography revealed decreased left ventricular (LV) systolic function (EF Simpson Biplane 27%), LV diastolic dysfunction, normal right ventricular (RV) systolic function, global hypokinesis, mild mitral regurgitation, mild tricuspid regurgitation, intermediate probability of pulmonary hypertension, and mild pulmonary regurgitation.

The patient was managed with a two-way intravenous (IV) line with lactated Ringer's (LR) solution at 60 cc/hour. She was kept nil per os (NPO) for at least six hours for solids and two hours for clear liquids prior to surgery. The target systolic blood pressure was maintained below 180 mmHg, and the target diastolic blood pressure was maintained below 110 mmHg. The planned surgical procedure was a cesarean section for fetal delivery and a concurrent female operative method (MOW) for sterilization. Premedication included metoclopramide 10 mg and ranitidine 50 mg IV administered one hour preoperatively. Additionally, she received methylprednisolone 62.5 mg and nebulized salbutamol the night before surgery and one hour preoperatively.

Intraoperatively, anesthesia was conducted using a combined low-dose epidural and spinal technique. The epidural was performed at the L3-L4 interspace with a target of reaching the T6 level, using 10 cc of ropivacaine 0.75%. The epidural catheter tip was ultimately positioned at the T9-T10 level. The spinal anesthetic was performed at the L4-L5 interspace with a target of reaching the T6 level, using 10 mg of bupivacaine heavy 0.5%. Preoperative analgesia was provided with ketorolac 30 mg, and postoperative analgesia was maintained with an epidural infusion of ropivacaine 0.25% and morphine 1 mg at a rate of 10 cc every 12 hours. The estimated blood loss during the surgery was 300 cc. Postoperatively, the patient was transferred to the intensive care unit (ICU) for monitoring.

On postoperative day one, the patient's vital signs were as follows: blood pressure 145/89 mmHg, pulse 96 beats per minute, respiratory rate 20 breaths per

minute, and SpO₂ 99% with a nasal cannula at 3 liters per minute. Laboratory investigations revealed hemoglobin 9.5 mg/dL, leukocytes 11,850/mm³, hematocrit 30.4%, platelets 322,000/mm³, sodium 138 mmol/L, potassium 3.69 mmol/L, chloride 103 mmol/L, and albumin 3.56 mg/dL. Antihypertensive therapy and epidural analgesia were continued, with a mean arterial pressure (MAP) target of 70-90 mmHg and a numeric rating scale (NRS) pain score target of 0-1.

On postoperative day two, the patient's vital signs were as follows: blood pressure 122/92 mmHg, pulse 91 beats per minute, respiratory rate 20 breaths per

minute, and SpO₂ 99% with a nasal cannula at 3 liters per minute. Antihypertensive therapy and epidural analgesia were continued, with a MAP target of 70-90 mmHg and an NRS pain score target of 0-1. This case highlights the complexity of anesthetic management in a patient with PPCM and multiple comorbidities. The combined low-dose epidural and spinal technique provided effective anesthesia and analgesia with minimal hemodynamic changes. Close monitoring and individualized management were essential in ensuring a successful outcome for this high-risk patient.

Table 1. Timeline of disease in case 1.

Timeline	Events	Symptoms	Medications	Ejection
				fraction (EF)
9 years prior	Diagnosis of	-	-	-
	uncontrolled			
	hypertension			
4 years prior	Second cesarean	Likely related to	-	-
	section for severe	preeclampsia		
	preeclampsia	(e.g.,		
		hypertension,		
		proteinuria)		
3 years prior	Diagnosis of heart	-	Furosemide,	-
	disease (mild MR,		Adalat oros,	
	mild TR, mild PR,		Methyldopa,	
	PH, HF)		Bisoprolol	
Weeks leading up to	Worsening heart	Cough, shortness	-	-
the presentation	failure	of breath (worse		
		with exertion and		
		supine position),		
		fatigue		
On presentation	Hospital	Cough, shortness	Furosemide,	27%
	admission for	of breath (worse	Adalat oros,	
	cesarean section	with exertion and	Methyldopa,	
		supine position),	Bisoprolol	
		fatigue		
Post-cesarean	Recovery in ICU	-	Antihypertensive	-
section			therapy, epidural	
			analgesia	
			<u> </u>	

MR = Mitral Regurgitation, TR = Tricuspid Regurgitation, PR = Pulmonary Regurgitation, PH = Pulmonary Hypertension, HF = Heart Failure.

A 26-year-old female patient, gravida 1, para 0, presented at 34-36 weeks gestation with a primary diagnosis of peripartum cardiomyopathy (PPCM) with a left ventricular ejection fraction (LVEF) of 26%. She also had mild mitral regurgitation. The patient's PPCM diagnosis was relatively recent, and her LVEF had shown some improvement since the initial diagnosis. She was experiencing heart failure with reduced ejection fraction (HFrEF) classified as New York Heart Association (NYHA) functional class II. Her primary complaint was chest palpitations. She denied any shortness of breath, cough, or fever. The patient had no significant past medical history, denying any history of hypertension, diabetes mellitus, asthma, heart disease, kidney disease, or HIV. She had not undergone any prior surgeries. Her current medications included furosemide 2x20mg, propranolol 2x10mg, folic acid 2x1 tablet, and iron supplements 2x1 tablet.

On physical examination, the patient was alert and oriented with a GCS score of E4M6V5. Her vital signs were as follows: blood pressure 100/51 mmHg, pulse 100-102 beats per minute, respiratory rate 18 breaths per minute, weight 66 kg, height 155 cm, and SpO₂ 97% on 3 liters per minute of oxygen via nasal cannula. Her airway was patent. Examination of her eyes revealed no anemia or icterus. Chest auscultation revealed rhonchi in all lung fields. Abdominal examination was unremarkable with no distension, fundal height of 33 cm, fetal heart rate of 156 beats per minute, and no uterine contractions. An examination of her extremities revealed no edema.

Laboratory investigations revealed the following: hemoglobin 13 mg/dL, leukocytes 16,440/mm³, platelets 433,000/mm³, APTT 28.10 seconds, PT 10.20 seconds, sodium 134 mmol/L, potassium 4.36 mmol/L, chloride 102 mmol/L, urea 34.2 mg/dL, creatinine 0.58 mg/dL, brain natriuretic peptide (BNP) 602 pg/mL, SGOT 15 U/L, SGPT 12 U/L, albumin 3.54 mg/dL, and blood glucose 101 mg/dL. ECG showed atrial fibrillation with a rapid ventricular response (AF with RVR) at 106 beats per minute. Chest x-ray revealed cardiomegaly and pulmonary edema (Figure 2). Echocardiography confirmed the diagnosis of PPCM

with HFrEF, an apical left ventricular thrombus, and right ventricular failure.

The patient was managed with a single intravenous (IV) line with lactated Ringer's (LR) solution at 70 cc/hour. She was kept NPO for at least six hours for solids and two hours for clear liquids prior to surgery. The planned surgical procedure was a cesarean section for fetal delivery with concurrent intrauterine device (IUD) placement for contraception. Premedication included metoclopramide 10 mg and ranitidine 50 mg IV administered one hour preoperatively.

Intraoperatively, anesthesia was conducted using a combined spinal-epidural (CSE) technique. The epidural was performed using 8 cc of ropivacaine 0.375%. The spinal anesthetic was performed at the L4-L5 interspace with a target of reaching the T6 level, using 7.5 mg of bupivacaine heavy 0.5%. Postoperative analgesia was maintained with an epidural infusion of ropivacaine 0.25% and morphine 1 mg at a rate of 8 cc every 12 hours. She also received scheduled ketorolac 3x30mg for breakthrough pain. The estimated blood loss during the surgery was 200 cc. Postoperatively, the patient was transferred to the ICU for monitoring.

On postoperative day one, the patient's vital signs were as follows: blood pressure 97/66 mmHg, pulse 104 beats per minute, respiratory rate 22 breaths per minute, and SpO₂ 100% with a nasal cannula at 3 liters per minute. Laboratory investigations revealed sodium 134 mmol/L, potassium 4.51 mmol/L, chloride 104 mmol/L, and albumin 3.05 mg/dL. Her postoperative medications included cefazolin 2x1 gram, tranexamic acid 3x500mg, ketorolac 3x30mg, paracetamol 3x1 gram, ranitidine 2x50mg, ondansetron 2x4mg, fentanyl infusion at 10 mcg/hour, amiodarone 540 mg every 18 hours, and propranolol 2x10mg. The target MAP was maintained above 65 mmHg. This case demonstrates the successful use of CSE anesthesia in a patient with PPCM and significant cardiac dysfunction. The patient's hemodynamic stability was maintained throughout the perioperative period with careful monitoring and appropriate pharmacological support. The use of a multimodal analgesic regimen provided effective pain relief and minimized opioid requirements.

Table 2. Timeline of disease in case 2.

Timeline	Events	Symptoms	Medications	Ejection fraction (EF)
Prior medical history	No history of hypertension, diabetes mellitus, asthma, heart disease, kidney disease, HIV, or previous surgery	-	-	- -
Weeks leading up to the presentation	Development of PPCM; severity and progression unclear	Chest palpitations	Furosemide, propranolol, folic acid, SF (sulfate ferrous)	-
On presentation	Hospital admission for cesarean section	Chest palpitations	Furosemide, propranolol, folic acid, SF	26%
Post-cesarean section	Recovery in ICU	-	Cefazolin, tranexamic acid, ketorolac, paracetamol, ranitidine, ondansetron, fentanyl, amiodarone, propranolol	-

A 26-year-old female patient, gravida 1, para 0, presented with a complex constellation of diagnoses, including heart failure with reduced ejection fraction (HFrEF) classified as NYHA functional class II, suspected peripartum cardiomyopathy (PPCM), trivial mitral regurgitation, trivial aortic regurgitation, trivial pulmonary regurgitation, mild pericardial effusion, thrombocytosis, hypoalbuminemia, and abdominal pain. Her primary complaint was a persistent cough. She denied any fever, shortness of breath, nausea, vomiting, or runny nose. The patient had no significant past medical history, denying any history of hypertension, diabetes mellitus, asthma, heart disease, or kidney disease. Her current medications included bisoprolol 2.5mg, lisinopril 10mg, furosemide 40mg, spironolactone 25mg, and folic acid 1x1 tablet.

On physical examination, the patient was alert and oriented with a GCS score of E4M6V5. Her vital signs were as follows: blood pressure 91/55 mmHg, pulse 79 beats per minute, respiratory rate 19 breaths per minute, weight 47 kg, height 160 cm, and SpO₂ 99% on

room air. Her airway was patent. Examination of her eyes revealed no anemia or icterus. Chest auscultation revealed rhonchi in all lung fields. Abdominal examination was unremarkable with no distension. An examination of her extremities revealed no edema.

Laboratory investigations revealed the following: hemoglobin 10.8 mg/dL, leukocytes 9,960/mm³, platelets 642,000/mm³, APTT 25.60 seconds, PT 11.30 seconds, sodium 142 mmol/L, potassium 3.46 mmol/L, chloride 92 mmol/L, urea 25 mg/dL, creatinine 0.74 mg/dL, SGOT 24 U/L, SGPT 9 U/L, albumin 3.76 mg/dL, and blood glucose 82 mg/dL. ECG showed normal sinus rhythm at 82 beats per minute. Chest x-rav was unremarkable. Echocardiography revealed decreased LV systolic function (Simpson Biplane EF 34%), LV diastolic dysfunction, normal RV systolic function, and global hypokinesis.

A computed tomography (CT) scan of the abdomen revealed an abscess on the right abdominal wall communicating with the uterine cavity, a subcutaneous tissue defect in the suprapubic region, an air-density lesion in the uterine cervix with a slightly hyperdense lesion on the posterior wall of the cervix, suspected fistulation with the anterior wall of the rectum, minimal ascites, and hepatomegaly. Magnetic resonance imaging (MRI) of the abdomen confirmed the presence of a multiloculated intraperitoneal abscess extending from the pelvic cavity to the left side of the abdomen, with fistulation to the anterior wall of the uterus and involvement of the subcutaneous tissue through a defect in the rectus abdominis muscle. The MRI also revealed multiple Tarlov cysts at the S2 level bilaterally and hepatomegaly.

The patient was managed with a single intravenous (IV) line with lactated Ringer's (LR) solution at 90 cc/hour. She was kept NPO for at least six hours for solids and two hours for clear liquids prior to surgery. The planned surgical procedure was re-hecting and debridement of the abdominal and pelvic abscesses. Premedication included metoclopramide 10 mg and ranitidine 50 mg IV administered one hour preoperatively.

Intraoperatively, anesthesia was conducted using an epidural technique. The epidural was performed at the L4-L5 interspace with a target of reaching the T6-L2 level, using 10 cc of a mixture of ropivacaine 0.75% and fentanyl 50 mcg. The epidural catheter tip was positioned at the T10-T11 level. Postoperative analgesia was maintained with an epidural infusion of ropivacaine 0.25% at a rate of 10 cc every 8 hours. She also received scheduled metamizole 3x1 gram for breakthrough pain. The patient was transferred to the ICU for postoperative monitoring. This case illustrates the successful use of epidural anesthesia in a patient with suspected PPCM and multiple comorbidities, including thrombocytosis, hypoalbuminemia, and a complex intra-abdominal and pelvic infection. The epidural technique provided effective anesthesia and analgesia, facilitating the surgical procedure and postoperative pain management. The patient's hemodynamic stability was maintained throughout the perioperative period with careful monitoring and individualized management.

Table 3. Timeline of disease in case 3.

Timeline	Events	Symptoms	Medications	Ejection
				fraction (EF)
Prior medical	No history of	-	-	-
history	hypertension, diabetes			
	mellitus, asthma, heart			
	disease, or kidney			
	disease			
Weeks leading up	Development of PPCM,	Cough,	Bisoprolol,	-
to presentation	thrombocytosis, and	abdominal	lisinopril,	
	hypoalbuminemia;	pain	furosemide,	
	severity and		spironolactone,	
	progression unclear		folic acid	
On presentation	Hospital admission for	Cough,	Bisoprolol,	34%
	cesarean section	abdominal	lisinopril,	
		pain	furosemide,	
			spironolactone,	
			folic acid	
Post-cesarean	Recovery in ICU	-	Metamizole	-
section				

A 30-year-old female patient, gravida 1, para 0, presented at 32-34 weeks gestation with a history of post-repair tetralogy of Fallot (TOF) at 24 years of age. Her current diagnoses included mild tricuspid regurgitation, intermediate probability pulmonary hypertension, stage II hypertension with NYHA functional class II heart failure attributed to suspected PPCM, marginal placenta previa, uterine myoma, and severe myopia. Her primary complaint was shortness of breath when lying down for prolonged periods. She reported a history of hypertension and TOF since childhood but denied any history of diabetes mellitus, asthma, or prior surgeries. Her current medications included candesartan 8mg once daily and bisoprolol 2.5mg once daily.

On physical examination, the patient was alert and oriented with a GCS score of E4M6V5. Her vital signs were as follows: blood pressure 116/87 mmHg, pulse 123 beats per minute, respiratory rate 20-24 breaths per minute, weight 70 kg, height 158 cm, and SpO₂ 99% on room air. Her airway was patent. Examination of her eyes revealed no anemia or icterus. Chest auscultation revealed rhonchi in all lung fields. Abdominal examination revealed a fundal height of 31 cm, longitudinal lie with cephalic presentation, estimated fetal weight of 2100 grams, fetal heart rate of 150 beats per minute, and no uterine contractions. An examination of her extremities revealed no edema.

Laboratory investigations revealed the following: hemoglobin 11.5 mg/dL, leukocytes 6,910/mm3, platelets 224,000/mm3, APTT 24.6 seconds, PT 56 seconds, sodium 135 mmol/L, potassium 4.62 mmol/L, chloride 105 mmol/L, urea 31.9 mg/dL, creatinine 0.75 mg/dL, SGOT 58 U/L, SGPT 56 U/L, albumin 3.06 mg/dL, and blood glucose 83 mg/dL. ECG showed atrial fibrillation with a rapid ventricular response (AF with RVR) at 106 beats per minute. Echocardiography revealed dilatation of all cardiac chambers, decreased LV systolic function suggestive of

PPCM, adequate RV contractility, intact interatrial septum, intact interventricular septum, no patent ductus arteriosus, no residual ventricular septal defect, no residual pulmonary stenosis, mild to moderate tricuspid regurgitation with a high probability of pulmonary hypertension, mild mitral regurgitation, and mild pulmonary regurgitation. Cardiac MRI confirmed the echocardiographic findings, showing an LV ejection fraction of 40.8% (reduced), RV ejection fraction of 37% (reduced), enlargement of the right ventricle, right atrium, and left ventricle, and regional hypokinesis in the right coronary artery (RCA) and left circumflex artery (LCx) territories.

The patient was managed with a single intravenous (IV) line with lactated Ringer's (LR) solution at 60 cc/hour. She was kept NPO for at least six hours for solids and two hours for clear liquids prior to surgery. The planned surgical procedure was a cesarean section for fetal delivery with concurrent IUD placement for contraception. Premedication included metoclopramide 10 mg and ranitidine 50 mg IV administered one hour preoperatively.

Intraoperatively, anesthesia was conducted using an epidural technique. The epidural was performed at the L3-L4 interspace with a target of reaching the T6-L1 level, using 10 cc of a mixture of ropivacaine 0.75% and fentanyl 50 mcg. Postoperative analgesia was maintained with an epidural infusion of ropivacaine 0.1875% and morphine 1 mg at a rate of 10 cc every 12 hours. The patient was transferred to the ICU for postoperative monitoring. This case demonstrates the successful use of epidural anesthesia in a patient with PPCM and a complex cardiac history, including repaired TOF and pulmonary hypertension. The epidural technique provided effective anesthesia and analgesia while maintaining hemodynamic stability. The patient's complex medical history and the presence of placenta previa necessitated careful monitoring and individualized management throughout the perioperative period.

Table 4. Timeline of disease in case 4.

Timeline	Events	Symptoms	Medications	Ejection
				Fraction (EF)
Since childhood	Tetralogy of Fallot	-	-	-
	repair			
Prior medical	Hypertension	-	Candesartan,	-
history			Concor	
Weeks leading up	Development of	Shortness of	Candesartan,	-
to	PPCM; severity	breath when lying	Concor	
the presentation	and progression	down		
	unclear			
On presentation	Hospital	Shortness of	Candesartan,	35% (estimated
	admission for	breath when lying	Concor	based on
	cesarean section	down		the description of
				"decreased LV
				systolic function")
Post-cesarean	Recovery in ICU	-	-	-
section				

These cases illustrate the variety of presentations and anesthetic management strategies for women with PPCM undergoing cesarean section. Regional anesthesia techniques, including epidural and CSE anesthesia, were successfully used in all cases, providing effective anesthesia and analgesia while maintaining hemodynamic stability (Table 5).

Table 5. Anesthetic management for 4 cases.

Case	Anesthesia type	Medications	
Case 1	Combined epidural and low-	Epidural: Ropivacaine 0.75%	
	dose spinal	Spinal: Bupivacaine heavy 0.5%	
Case 2	Combined epidural and low-	Epidural: Ropivacaine 0.375%	
	dose spinal	Spinal: Bupivacaine heavy 0.5%	
Case 3	Epidural	Ropivacaine 0.75%, fentanyl	
Case 4	Epidural	Ropivacaine 0.75%, fentanyl	

3. Discussion

Peripartum cardiomyopathy (PPCM) is a rare disorder characterized by the development of heart failure in the final month of pregnancy or up to 5 months after delivery. The clinical presentation of PPCM can vary from mild to severe, with common symptoms including fatigue, shortness of breath, edema (swelling), and palpitations. In severe cases, patients may present with signs of heart failure, such as pulmonary edema (fluid in the lungs) and cardiogenic shock (the heart's inability to pump

enough blood). The diagnosis of PPCM is typically made through a combination of clinical evaluation and imaging studies, particularly echocardiography. Echocardiography is a type of ultrasound that allows visualization of the heart's structure and function. In PPCM, echocardiography typically reveals an enlarged left ventricle with reduced contractility, resulting in a decreased ejection fraction (EF). EF is a measure of the percentage of blood pumped out of the left ventricle with each heartbeat. An EF of less than 45% is suggestive of PPCM. Studies have identified specific

gene mutations that may increase susceptibility to PPCM, suggesting a genetic component in some cases. The presence of autoantibodies against cardiac proteins in PPCM patients suggests a potential role for immune system dysfunction in the development of the disease. The dramatic hormonal fluctuations that occur during pregnancy and the postpartum period may contribute to the development of PPCM in susceptible individuals. Increased oxidative stress has been implicated in the pathogenesis of PPCM, potentially leading to myocardial damage and dysfunction. Additional factors that may play a role in PPCM development include nutritional deficiencies, infections, pre-eclampsia, and advanced maternal age. The clinical presentation of PPCM can vary widely, depending on the severity of the condition. Some women may experience only mild symptoms, while others may develop severe heart failure. Fatigue is often one of the earliest symptoms of PPCM and may be attributed to the heart's reduced ability to pump blood effectively. Shortness of breath may occur at rest or with exertion and is due to fluid buildup in the lungs (pulmonary edema). Edema, swelling in the legs, ankles, and feet is common due to fluid retention. Palpitations are sensations of a rapid or irregular heartbeat. Chest pain may experience chest pain or discomfort. A persistent cough may be present, especially when Dizziness lying down. lightheadedness symptoms may occur due to decreased blood flow to the brain. In severe cases, patients may present with signs of heart failure. Severe shortness of breath may be accompanied by wheezing or gasping for air. The heart beats faster to try to compensate for its reduced pumping ability. The heart's inability to pump enough blood can lead to low blood pressure. Cyanosis is a bluish discoloration of the skin and mucous membranes due to lack of oxygen. Cardiogenic shock is a life-threatening condition in which the heart is unable to pump enough blood to meet the body's needs. The diagnosis of PPCM is typically made based on a combination of clinical findings and imaging studies. Echocardiography is the most important diagnostic test for PPCM. It allows visualization of the heart's structure and function, revealing characteristic features of PPCM such as left

EF. ventricular enlargement and reduced Electrocardiogram (ECG) test records the electrical activity of the heart and may show abnormalities in heart rhythm or signs of left ventricular hypertrophy (enlargement). Chest X-ray may reveal an enlarged heart and fluid in the lungs. Blood tests may be used to rule out other causes of heart failure and to assess kidney function and electrolyte levels. Cardiac catheterization invasive procedure may be performed in some cases to measure pressures within the heart chambers and assess coronary artery disease. The treatment of PPCM focuses on managing heart failure and improving cardiac function. Various medications may be used to treat PPCM, including diuretics to remove excess fluid, ACE inhibitors or ARBs to lower blood pressure and improve heart function, betablockers to slow the heart rate and reduce workload on the heart, and inotropic agents to improve heart contractility. Lifestyle changes such as sodium restriction, fluid restriction, and regular exercise may be recommended. In some cases, devices such as pacemakers or implantable cardioverter-defibrillators (ICDs) may be necessary to regulate heart rhythm and prevent sudden cardiac death. In severe cases of PPCM that do not respond to other treatments, a heart transplant may be considered. The prognosis for women with PPCM varies depending on the severity of the condition and the response to treatment. Some women experience a full recovery of heart function, while others may have persistent heart failure. The risk of complications and mortality is higher in women with severe PPCM or those who do not respond well to treatment.11-13

Peripartum cardiomyopathy (PPCM) presents unique challenges for anesthesiologists due to the complex interplay of cardiovascular, respiratory, and hematological changes that occur during pregnancy and the postpartum period. The presence of PPCM further complicates this delicate balance, increasing the risk of perioperative complications. Therefore, meticulous planning and execution of anesthetic care are crucial for optimizing maternal and fetal outcomes in women with PPCM undergoing cesarean section. A comprehensive preoperative assessment is essential to evaluate the severity of PPCM and identify any

potential risk factors that may influence anesthetic management. This assessment should include a thorough review of the patient's medical history, including the onset and progression of PPCM medications, symptoms, current comorbidities. Physical examination should focus on signs of heart failure, such as jugular venous distension, pulmonary rales, and peripheral edema. Echocardiography plays a pivotal role in assessing the severity of left ventricular dysfunction and guiding anesthetic management. Key echocardiographic parameters to consider include left ventricular ejection fraction (LVEF), left ventricular end-diastolic dimension (LVEDD), and the presence of valvular abnormalities or intracardiac thrombi. Maintaining stable hemodynamics is paramount to ensure adequate uteroplacental perfusion and prevent maternal cardiovascular complications. This involves optimizing preload, afterload, and contractility to ensure adequate cardiac output and tissue perfusion. Ensuring adequate oxygenation is crucial to prevent hypoxemia, which can exacerbate cardiac dysfunction and lead to complications such as pulmonary edema and arrhythmias. Providing effective analgesia is essential to minimize pain and stress, which can trigger sympathetic nervous system activation and worsen hemodynamic instability. Vigilant monitoring and prompt intervention are necessary to prevent and manage potential complications such as pulmonary edema, arrhythmias, thromboembolism, and cardiac arrest. The choice of anesthetic technique for cesarean section in women with PPCM is a critical decision that should be individualized based on the patient's clinical condition, preferences, and the anesthesiologist's expertise. Regional anesthesia, including spinal, epidural, and combined spinal-epidural (CSE) techniques, is generally preferred over general anesthesia in this population. By avoiding the myocardial depressant effects of general anesthetics and reducing afterload, regional anesthesia can help maintain hemodynamic stability. Regional anesthesia allows for a faster recovery and earlier ambulation, which can reduce the risk of thromboembolic complications. In patients with the potential for pulmonary edema, regional anesthesia avoids the risks associated with airway manipulation and intubation. However, regional anesthesia is not without its considerations in PPCM patients. Hypotension is a common side effect, and careful fluid management and vasopressor support may be required. Additionally, the presence of coagulopathy or thrombocytopenia may increase the risk of bleeding complications with regional techniques. General anesthesia may be considered in certain situations, such as when regional anesthesia is contraindicated or when the patient's hemodynamic instability precludes its use. However, general anesthesia requires careful titration of anesthetic agents to avoid myocardial depression and hemodynamic fluctuations. Regardless of the anesthetic technique chosen, meticulous intraoperative management is crucial to ensure hemodynamic stability and prevent complications. Continuous ECG monitoring, invasive blood pressure monitoring (arterial line), and pulse oximetry are essential. In patients with severe cardiac dysfunction, additional monitoring modalities such as central venous pressure (CVP) monitoring or pulmonary artery catheterization may be considered. Fluid management is a critical aspect of intraoperative care. Excessive fluid administration can worsen pulmonary edema, while inadequate fluid replacement can lead to hypotension and decreased cardiac output. A balanced approach is necessary, with careful titration of fluids based on the patient's hemodynamic status and urine output. Vasopressors may be required to treat hypotension, while inotropic agents may be used to improve cardiac contractility. Antiarrhythmic medications should be readily available to manage any arrhythmias that may occur. Supplemental oxygen should be administered to maintain adequate oxygen saturation and prevent hypoxemia. Maintaining normothermia is important to prevent shivering, which can increase myocardial oxygen demand. Effective communication teamwork between the anesthesiologist, obstetrician, and other healthcare providers are essential to ensure coordinated care and prompt management of any complications. Postoperative care of PPCM patients undergoing cesarean section requires close monitoring in an intensive care unit (ICU) or a high-dependency

unit (HDU). This allows for early detection and management of potential complications such as pulmonary edema, arrhythmias, and thromboembolism. Multidisciplinary collaboration between anesthesiologists, obstetricians, cardiologists, and intensivists is essential to optimize patient outcomes.¹⁴⁻¹⁶

Hemodynamic monitoring is the cornerstone of safe and effective anesthetic management in patients with peripartum cardiomyopathy (PPCM) undergoing cesarean section. The dynamic nature of this condition, coupled with the physiological changes of pregnancy and the potential for rapid deterioration, necessitates continuous and vigilant monitoring to ensure maternal and fetal well-being. PPCM patients are at increased risk of hemodynamic instability, including hypotension, hypertension, arrhythmias. Continuous monitoring allows for early detection of these changes, enabling prompt intervention and preventing adverse outcomes. Monitoring parameters such as heart rate, blood pressure, and oxygen saturation provide valuable information about the patient's cardiac function and response to treatment. Hemodynamic monitoring helps guide fluid and drug therapy, ensuring adequate tissue perfusion and preventing fluid overload or excessive vasoconstriction. Monitoring the patient's response to anesthetic interventions, such as regional anesthesia or general anesthesia, is crucial to ensure hemodynamic stability and prevent complications. Continuous ECG monitoring is essential to detect arrhythmias, myocardial ischemia, and electrolyte imbalances. PPCM patients are at increased risk of atrial fibrillation, ventricular tachycardia, and other arrhythmias that can compromise cardiac output and maternal stability. An arterial line provides continuous and accurate blood pressure measurement, which is crucial for detecting and managing hypotension or hypertension. In PPCM patients, maintaining adequate blood pressure is essential to ensure uteroplacental perfusion and prevent maternal endorgan damage. Pulse oximetry measures the oxygen saturation of arterial blood, providing a continuous assessment of oxygenation. Hypoxemia can exacerbate cardiac dysfunction and lead to complications such as pulmonary edema and arrhythmias. CVP monitoring provides information about right ventricular preload and can help guide fluid management. In PPCM patients, CVP monitoring can help prevent fluid overload, which can worsen pulmonary edema and compromise respiratory function. Pulmonary artery catheterization (PAC) is an invasive monitoring technique that provides detailed information about cardiac output, pulmonary artery pressures, and mixed venous oxygen saturation. PAC is typically reserved for patients with severe PPCM or those who are hemodynamically unstable despite standard monitoring and treatment. Esophageal Doppler monitoring provides a non-invasive estimate of cardiac output and can be used to guide fluid and drug therapy. Pulse contour analysis uses arterial waveform analysis to estimate cardiac output and other hemodynamic parameters. Bioreactance is a non-invasive technique that measures the electrical properties of the thorax to estimate cardiac output and fluid status. The interpretation of hemodynamic data in PPCM patients requires a thorough understanding of the pathophysiology of the disease and the potential impact of anesthetic interventions. It is essential to consider the patient's clinical context, including the severity of cardiac dysfunction, the presence of comorbidities, and the type of anesthetic technique used.17,18

Fluid management is a critical aspect of anesthetic care in patients with peripartum cardiomyopathy (PPCM) undergoing cesarean section. It requires a delicate balance to ensure adequate intravascular volume for uteroplacental perfusion and maternal stability while avoiding fluid overload that can exacerbate pulmonary edema and compromise cardiac function. Pregnancy induces significant physiological changes in the cardiovascular system, including increased blood volume, cardiac output, and venous capacitance. These changes, coupled with the left ventricular dysfunction characteristic of PPCM, create a delicate balance that can be easily disrupted by fluid mismanagement. The failing left ventricle in PPCM patients is less able to handle increased preload. Excessive fluid administration can lead to pulmonary congestion and edema, compromising oxygenation

and further stressing the heart. Fluid overload can increase systemic vascular resistance, increasing afterload on the left ventricle and further impairing cardiac output. Excessive crystalloid administration can dilute clotting factors and platelets, increasing the risk of bleeding. Hypovolemia can lead to hypotension, compromising uteroplacental perfusion and fetal oxygenation. In PPCM patients, even mild hypovolemia can significantly reduce cardiac output, leading to maternal and fetal distress. Inadequate renal perfusion can lead to acute kidney injury. Maintaining normovolemia (normal blood volume) is crucial to ensure adequate uteroplacental perfusion and maternal stability. Avoiding fluid overload is essential to prevent pulmonary edema and its associated complications. Fluid management should aim to optimize cardiac output by balancing preload and afterload. A thorough preoperative assessment is essential to evaluate the patient's fluid status and identify any potential risk factors for fluid mismanagement. This includes reviewing the patient's medical history, performing a physical examination, and evaluating laboratory data such as hematocrit hemodynamic electrolytes. Continuous and monitoring is crucial to guide fluid management during surgery. This includes monitoring heart rate, blood pressure, urine output, and central venous pressure (CVP) if available. The choice of fluid depends on the patient's individual needs and the clinical situation. Crystalloids, such as lactated Ringer's solution or normal saline, are commonly used for initial volume replacement. Colloids, such as albumin or hydroxyethyl starch, may be considered in patients with hypoalbuminemia or significant fluid shifts. Goaldirected fluid therapy (GDFT) involves using hemodynamic monitoring parameters to guide fluid administration. This approach aims to optimize cardiac output and tissue perfusion by titrating fluids to achieve specific hemodynamic goals. Fluid management should be individualized based on the patient's specific needs and clinical condition. Factors to consider include the severity of PPCM, the presence of comorbidities, and the type of anesthetic technique used. 19,20

4. Conclusion

This case series highlights the successful implementation of regional anesthesia techniques in managing four pregnant women with peripartum (PPCM) undergoing cardiomyopathy cesarean sections. The use of epidural anesthesia, with or without the addition of a low-dose spinal component, facilitated hemodynamic stability throughout the perioperative period. This approach proved particularly valuable in mitigating the risk of pulmonary edema, a serious complication associated with general anesthesia in patients with PPCM. These cases underscore the importance of individualized anesthetic plans tailored to the unique needs and circumstances of each patient with PPCM. The careful selection of anesthetic techniques, along with vigilant monitoring and prompt management of potential complications, contributed to the favorable outcomes observed in this case series. The findings from this study support the growing body of evidence suggesting that regional anesthesia is a safe and effective option for cesarean delivery in women with PPCM. By sharing these experiences, we aim to enhance awareness and promote the adoption of best practices in the anesthetic management of this challenging patient population.

5. References

- Behary Paray N, Ramphul K, Picker SM, Akkaramani S, Memon RA, Ahmed M, et al. Age-related disparities in complications among women with peripartum cardiomyopathy. Curr Probl Cardiol. 2024; 49(8): 102647.
- Perea Rojas DM, Seni Hernandez CD, Rojas Torres IL, Olivares Olmos M, Garcia Jarava CM, Gaivao Arciniegas DJ, et al. Peripartum cardiomyopathy: a case report of mortality from a rare and potentially fatal condition. J Med Cases. 2024; 15(8): 171–9.
- 3. Nalla L, Ragam AS, Prakash V. Peripartum cardiomyopathy: case series from a tertiary care centre in India. Int J Reprod Contracept Obstet Gynecol. 2024; 13(9): 2487–92.

- 4. Huang H, Ye Q, Xu Y, Tao B, Liu J, Xie T, et al. Risk factors and clinical features of peripartum cardiomyopathy in a Chinese population. J Multidiscip Healthc. 2024; 17: 3763–72.
- 5. Sliwa K, Rakisheva A, Viljoen C, Pfeffer T, Simpson M, Jackson AM, et al. Living with peripartum cardiomyopathy: a statement from the Heart Failure Association and the Association of Cardiovascular Nursing and Allied Professions of the European Society of Cardiology. Eur J Heart Fail. 2024.
- Iannaccone G, Graziani F, Kacar P, Tamborrino PP, Lillo R, Montanaro C, et al. Diagnosis and management of peripartum cardiomyopathy and recurrence risk. Int J Cardiol Congenit Heart Dis. 2024; 17(100530): 100530.
- Ibeh C, Kulick ER, Boehme AK, Friedman AM, Miller EC, Bello NA. Incident stroke in individuals with peripartum cardiomyopathy. Am Heart J. 2024; 275: 138–40.
- 8. Prameswari HS, Kamarullah W, Pranata R, Putra ICS, Undarsa AC, Iqbal M, et al. Meta-analysis of cardiac magnetic resonance in prognosticating left ventricular function in peripartum cardiomyopathy. ESC Heart Fail. 2024.
- van der Meer P, van Essen B, Viljoen C, Böhm M, Jackson A, Hilfiker-Kleiner D, et al. Bromocriptine treatment and outcomes in peripartum cardiomyopathy: the EORP PPCM registry. Eur Heart J. 2024.
- Lasica R, Asanin M, Vukmirovic J, Maslac L, Savic L, Zdravkovic M, et al. What do we know about peripartum cardiomyopathy? Yesterday, today, tomorrow. Int J Mol Sci. 2024; 25(19): 10559.
- 11. Falola AO, Razvi N, Gada R, Thompson DR, Martin CR. Takotsubo syndrome or peripartum cardiomyopathy? Depends on who you are talking to. Behav Sci (Basel). 2024; 14(9): 777.
- 12. Du Plessis J, Gujrathi R, Hassanin M, McKee H, Hanneman K, Karur GR, et al. Peripartum

- cardiomyopathy is associated with abnormalities of myocardial deformation and late gadolinium enhancement. Can Assoc Radiol J. 2024; 8465371241268426.
- 13. Sliwa K, Hilfiker-Kleiner D, Petrie MC, Mebazaa A, Pieske B, Buchmann E, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. Eur J Heart Fail. 2010; 12(8): 767–78.
- 14. Schelbert EB, Elkayam U, Cooper LT, Givertz MM, Alexis JD, Briller J, et al. Myocardial damage detected by late gadolinium enhancement cardiac magnetic resonance is uncommon in peripartum cardiomyopathy. J Am Heart Assoc. 2017; 6(4).
- 15. Karaye KM, Ishaq NA, Sai'du H, Balarabe SA, Ahmed BG, Adamu UG, et al. Disparities in clinical features and outcomes of peripartum cardiomyopathy in high versus low prevalent regions in Nigeria. ESC Heart Fail. 2021; 8(4): 3257–67.
- 16. Jackson AM, Bauersachs J, Petrie MC, van der Meer P, Laroche C, Farhan HA, et al. Outcomes at one year in women with peripartum cardiomyopathy: Findings from the ESC EORP PPCM Registry. Eur J Heart Fail. 2024; 26(1): 34–42.
- 17. Sliwa K, van der Meer P, Viljoen C, Jackson AM, Petrie MC, Mebazaa A, et al. Socioeconomic factors determine maternal and neonatal outcomes in women with peripartum cardiomyopathy: a study of the ESC EORP PPCM registry. Int J Cardiol. 2024; 398(131596): 131596.
- 18. Mbakwem AC, Bauersachs J, Viljoen C, Hoevelmann J, van der Meer P, Petrie MC, et al. Electrocardiographic features and their echocardiographic correlates in peripartum cardiomyopathy: results from the ESC EORP PPCM registry. ESC Heart Fail. 2021; 8(2): 879–89.

- 19. Kodogo V, Viljoen C, Hoevelmann J, Chakafana G, Tromp J, Farhan HA, et al.
 Proteomic profiling in patients with peripartum cardiomyopathy: a biomarker study of the ESC EORP PPCM registry. JACC Heart Fail. 2023; 11(12): 1708–25.
- 20. Karaye KM, Sa'idu H, Ishaq NA, Balarabe SA, Ahmed BG, Mohammed IY, et al. Selenium deficiency as a risk factor for peripartum cardiomyopathy. West Afr J Med. 2024; 41(2): 209–14.