Abstract
Peripartum cardiomyopathy (PPCM) is a relatively rare form of heart disease of unknown etiology with an incidence between 1:1300 and 1:4000 live births (1). PPCM is characterized by the onset of acute heart failure in the final trimester of pregnancy or within the first six months postpartum (2). Identified risk factors for PPCM include advanced maternal age, multiparity, obesity, preeclampsia, chronic hypertension, multiple gestation, and African descent. The diagnosis of PPCM presents a challenge as many otherwise healthy, pregnant women present with dyspnea and pedal edema that may mimic congestive cardiac failure in the last trimester. A high index of clinical suspicion is required for the diagnosis of PPCM, and the diagnosis relies on echocardiographic identification of systolic dysfunction after the exclusion of all other causes of cardiomyopathy. Treatment may include sodium restriction, loop diuretics, ace inhibitors, vasodilators, digoxin, anticoagulants, and inotropic support. The majority of patients recover partially or completely, while some require a cardiac transplant.

CASE REPORT
A 28 year old female, 115 kg, G3/P2, and nurse by profession, was admitted to our hospital for elective Caesarean section (CS) in view of suspected cephalopelvic disproportion. The estimated size of the baby by ultrasound was more than 4 kg with breech presentation. The patient was non-diabetic and was being treated for mild pregnancy-induced hypertension (PIH) with oral Aldomet (α-methyldopa) 250mg once daily. She had no history to indicate any cardiac or respiratory disease and no history of breathlessness or fatigue beyond what can be expected during a normal pregnancy. Upon examination, the patient was comfortable at rest. Her pulse rate was 98/min, and her blood pressure (BP) was 128/74 mm Hg with no pitting type of edema. Auscultation of chest revealed bilateral, normal vesicular breathing with no additional sound. The patient received a dose of Aldomet before surgery.

Two large bore IV cannulas were established, and the patient received 2000ml of Ringer Lactate as preload before administration of the subarachnoid block (SAB). A lumbar SAB was administered between the L3-L4 interspace by injection of 0.5% hyperbaric bupivacaine (2.5ml) and fentanyl (25mcg). After confirmation of the level of anesthesia at T8, the surgeon proceeded with the CS. For the first ten minutes, the patient's vital signs including heart rate, BP, and SPO2 were monitored at one minute intervals. Approximately five minutes after administration of spinal anesthesia, her BP dropped to systolic pressure of 90mm Hg and diastolic of 47mm Hg. Incremental bolus injections of ephedrine were given, without any marked improvement in
the blood pressure. Within two minutes of delivery, the patient complained of drowsiness, experienced a rapid fall in blood pressure with decreasing heart rate, and underwent cardiac arrest. Closed chest compressions were started immediately with the patient in the left lateral tilt position. The patient was intubated with a 7mm ID cuffed endotracheal tube, while cricoid pressure was maintained.

The patient reverted to a normal sinus rhythm after approximately 90 seconds of CPR. During this period of resuscitation, the patient received 2mg of adrenaline and 2 mg of atropine. The surgical procedure was completed within 15 minutes of cardiac arrest. Due to persistent hypotension, inotropic support with a dobutamine infusion of 5mcg/kg was started to maintain her blood pressure in an acceptable range. At the end of the procedure, auscultation of the chest revealed bilateral crepitations. An electrocardiogram showed non-specific ST-T wave changes, and a chest radiograph revealed cardiomegaly and pulmonary congestion. The patient's arterial blood gas analysis (ABG) showed gross hypoxia and hypocarbia. In the postoperative period, the patient was transferred to a coronary care unit for elective ventilation. Echocardiography showed a dilated left ventricle with an ejection fraction of 40 percent and no other abnormality, and therefore, a diagnosis of peripartum cardiomyopathy was made. The patient was maintained on dobutamine, digoxin, furosemide, heparin, and losartan. Over a period of four days, the patient's serial ABG measurements showed improvement, and treatment with inotropes was diminished slowly. Weaning from the ventilator began, and finally, the patient was extubated on the seventh postoperative day. Heparin was replaced with warfarin and was continued after discharge.

Echocardiography repeated two weeks following the CS revealed an ejection fraction of 60%.

**DISCUSSION**

PPCM is defined as the onset of cardiac failure with no identifiable cause during the last trimester of pregnancy or within six months following delivery. Echocardiography remains an important tool for evaluation and follow-up in cases of PPCM. A decrease in myocardial systolic dysfunction as manifested by a decrease in the left ventricular ejection fraction or fractional shortening is essential to the diagnosis. The existence of normal systolic function excludes the diagnosis of PPCM.

Hypertension during pregnancy occurs at a higher frequency in patients presenting with PPCM. As our patient was not diagnosed prior to the surgical procedure, various factors contributed to her hemodynamic instability and presentation after administration of the SAB for CS. Because this patient was to undergo a SAB, she received 2000ml of crystalloids as preload, without knowing that she had PPCM. A more judicious and conservative approach to preoperative intravascular expansion is advised for patients with cardiopulmonary dysfunction (,), and therefore, this preload may have contributed to her hemodynamic instability.

Another contributing factor in the development of cardiac arrest was the type of anesthesia selected for surgery (SAB), which can lead to a sudden sympathetic block, further leading to a fall in systemic vascular resistance (SVR) and hemodynamic instability, particularly in patients with underlying cardiac dysfunction.

Various methods of anesthesia have been advised for patients with PPCM undergoing a CS. There is disagreement on the optimal technique, as the uses of both general anesthesia (,) and regional anesthesia (,,,) have been described. General anesthesia is limited by the association with cardiac depressant drugs or high dose opioids, which may necessitate the need for postoperative elective ventilation for the newborn and/or the mother. Regional anesthesia using an epidural technique by administering small incremental doses of local anesthetics along with opioids seems a better option than administration of a SAB, which may be associated with a more precipitous and catastrophic fall in blood pressure and SVR (,). Shnaider et al. (,) reported the use of combined spinal epidural (CSE) in a patient with PPCM. They injected 6mg of hyperbaric bupivacaine together with 15 mcg fentanyl into the subarachnoid space and supplemented with epidural bupivacaine 5ml of 0.5% and 0.25% at 60 and 105 minutes, respectively. They did not report any hemodynamic instability during this period.

In conclusion, a high degree of clinical suspicion for diagnosis, prompt initiation of appropriate medical therapy in the perioperative period, and the selection of proper anesthetic technique are all required for successful outcome of PPCM patients undergoing a CS.

**CORRESPONDENCE TO**

Ghulam Mohammed Mir, MD  
Anesthesiology Specialist  
Department of Anesthesiology  
Madina Maternity and Children's Hospital  
Madina Munawara, KSA
References
Author Information

Ghulam Mohammed Mir, MD
Department of Anaesthesiology, Madina Maternity and Children's Hospital

Asaf Shora, MD
Department of Anaesthesiology, Madina Maternity and Children's Hospital