

Afterload Reduction Techniques for Management of Peripartum Cardiomyopathy

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Abstract

Two women presented for delivery, each with peripartum cardiomyopathy and left ventricular ejection fraction <10 percent. Aggressive afterload reduction prior to delivery resulted in successful parturition with recovery of cardiac function. For the first patient, titrated epidural anesthesia allowed positioning for placement of hemodynamic monitors, additional regional anesthesia dosing, and Cesarean section. After failing a trial of pharmacologic afterload reduction, the second patient received intra-aortic balloon counterpulsation for afterload reduction and successful general anesthesia for Cesarean section.

Keywords: Afterload reduction; Peripartum cardiomyopathy

Introduction

Peripartum cardiomyopathy (PPCM), a rare and serious complication occurring during pregnancy or within 5 months thereafter, involves cardiac systolic dysfunction and low left ventricular ejection fraction [1-3]. We present two cases of women with PPCM undergoing cesarean section that benefitted from unique management techniques (Table 1).

	Case 1	Case 2
Age (years)	30	31
Gestational age at presentation	32 weeks	38 weeks
Presenting symptom	Dyspnea on exertion	Dyspnea, palpitations
Obstetric diagnosis	HELLP syndrome	Pre-eclampsia
Initial blood pressure	139/114	140/105
Initial heart rate/rhythm	147/min Sinus tachycardia	162/min Atrial flutter
Initial ejection fraction	<10%	5-10%
Method of afterload reduction	Epidural anesthesia	Intra-aortic balloon counter-pulsation
Ejection fraction after delivery	55-60% (3 weeks later)	41% (2 weeks later)

Table 1: Clinical features of 2 cases of peripartum cardiomyopathy.

Case 1: Management with epidural anesthesia for afterload reduction

A 30 year old G₂P₁₀₀₁ woman had an uncomplicated pregnancy until 32 weeks gestation, when dyspnea on exertion developed, progressing the next day to dyspnea at rest, without chest pain or syncope. Mitral valve prolapse had been diagnosed 9 years prior. Her first pregnancy terminated at 41 weeks with Cesarean section. Examination revealed blood pressure 139/114 mmHg, heart rate 147/min, oxygen saturation 99% breathing room air, and category I fetal heart tones. Laboratory results of aspartate transaminase 58 U/mL, alanine transaminase 28 U/mL, platelet count 111 G/L, and urine protein to creatinine ratio of 4134 mg/g suggested preeclampsia with HELLP syndrome. A serum BNP>4200 with troponin 0.81 led to an echocardiogram, revealing <10% ejection fraction (Figure 1A), consistent with stress-induced cardiomyopathy. She received 40 mg furosemide IV but could not tolerate a recumbent position.

Caregivers planned repeat Cesarean section under regional anesthesia. With pre-anesthetic heart rate 158/min and systemic blood pressure (BP) 88/50 mmHg, she received radial arterial and epidural catheters. Incremental epidural administration of 2% lidocaine achieved a T10 analgesic level without changes in fetal heart rate. Only at that time did she tolerate Trendelenburg's position for pulmonary arterial catheter placement via the right internal jugular vein. BP was 150/105 mmHg, cardiac output 4.3 liters/min, and pulmonary artery pressure (PAP) 49/21 mmHg. Cesarean section began after additional epidural lidocaine over 30 min decreased PAP to 25/17 mmHg, with BP 130/90 and 3.2 l/min cardiac output. The delivered neonate had Apgar scores of 6 and 9 at 1 and 5 minutes respectively. At ICU arrival, the parturient had BP 97/63 mmHg, heart rate 105/min, and 99% oxygen saturation on room air. She received oral carvedilol 6.25 mg and lisinopril 20 mg, each twice daily. Echocardiogram 2 days later revealed 30% ejection fraction; discharge occurred on postoperative day 4. Echocardiogram 3 weeks postpartum showed 55-60% ejection fraction (Figure 1B).

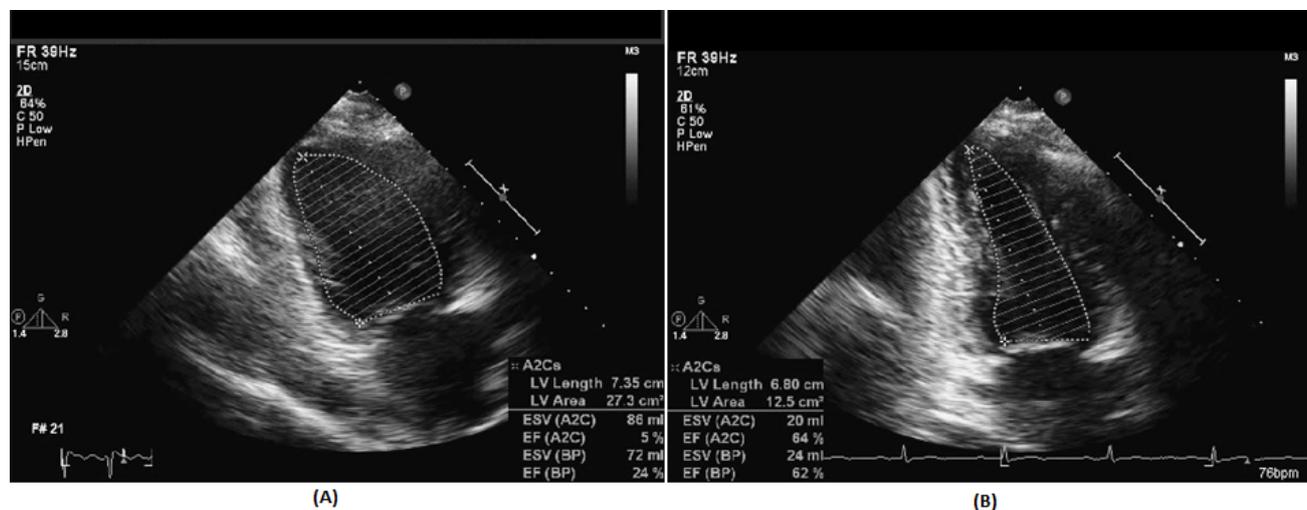


Figure 1: Case 1- Transesophageal echocardiogram mid-papillary level transverse view at end-systole. (A) Prior to epidural lidocaine titration, with ejection fraction 5-10%; (B) Three weeks after delivery, with ejection fraction 55-60%.

Case 2: Management with intra-aortic balloon for afterload reduction

A G₆P₂₀₃₂, 31 year old woman, 61 inches tall and weighing 120 kg, presented at 24 weeks gestation with spotting and cramping for 1 week. Her obstetric history featured a left ectopic pregnancy at age 14, spontaneous abortions at ages 15 and 18, one live vaginal birth, and one live birth by cesarean section. Medical history included type 2 diabetes for 5 years, sickle-cell trait, depression with attempted suicide, uterine fibroids, and hepatitis C. She denied smoking and drug allergies. Medications included a complicated insulin schedule, with injections in early morning, following every meal, and at bedtime, oral sustained release bupropion 100 mg daily, and alpha methyl dopa 250 mg twice daily.

At 34 weeks and 6 days gestation the patient presented with headache, 1+ deep tendon reflexes, blood pressure 140/105 mmHg, and platelet count 139 G/L. She received fluids, alpha methyl dopa 250 mg, and was discharged. At a routine 36-week gestation checkup, she claimed to sense a lack of fetal motion. Serum creatinine concentration was 1.4 mg/dL. She was diagnosed with pre-eclampsia and scheduled for delivery via repeat cesarean section.

At 38 weeks and 4 days gestation, the day prior to scheduled cesarean delivery, the patient presented with abdominal pain, palpitations, and dyspnea. Maternal heart rate was 162/min, blood pressure 163/129 mmHg, and SpO₂ 98%. Fetal heart rate was 140/min. Vaginal examination showed 1cm cervical dilation with 80% effacement. CT scan revealed pulmonary vascular congestion, dependent edema, and cardiomegaly. An electrocardiogram revealed atrial flutter. An echocardiogram demonstrated a left ventricular

ejection fraction of 5-10% (Figure 2A); it had been 20% six months prior. The caregivers planned first to control the patient's hypertension and tachycardia prior to the elective cesarean section. A labetalol infusion, initiated to treat the hypertension, was halted when her blood pressure decreased to 70/30 mmHg and the fetal heart rate displayed variable decelerations. Four doses of intravenous digoxin 0.125 mg over 12 hours converted her atrial flutter to sinus rhythm at a rate of 115/min. Fetal heart rate, however, showed frequent episodes of bradycardia. Cesarean section, initially planned for the following day, became urgent. The consulting cardiologist suggested regional anesthesia.

Prepared equipment included a stand-by left ventricular assist device ready for emergency implantation and full cardiopulmonary bypass circuit. In the operating room, the patient had a heart rate of 116/min and blood pressure 89/57 mmHg. Following intravenous midazolam, 2 mg, and 100 mcg fentanyl, and insertion of a right radial arterial catheter, an intra-aortic balloon (IABP) was inserted via the right femoral artery and advanced to the thoracic aorta for cardiac assist. A successful rapid sequence anesthesia induction followed IABP placement, using 30 mg etomidate and 180 mg succinylcholine. Desflurane provided anesthesia maintenance. The delivered full-term child had Apgar scores of 1 and 7. Measurement of cord arterial blood gases revealed pH 7.17, PCO₂ 56 mmHg, and PO₂ 42 mmHg. Following cesarean section, the patient's blood pressure was 152/99 mmHg, heart rate of 112/min, and SpO₂ 100% while breathing supplemental oxygen. IABP remained in place for 24 hours. The patient was discharged 1 week following cesarean section. Echocardiogram 2 weeks after delivery disclosed ejection fraction of 41% (Figure 2B).

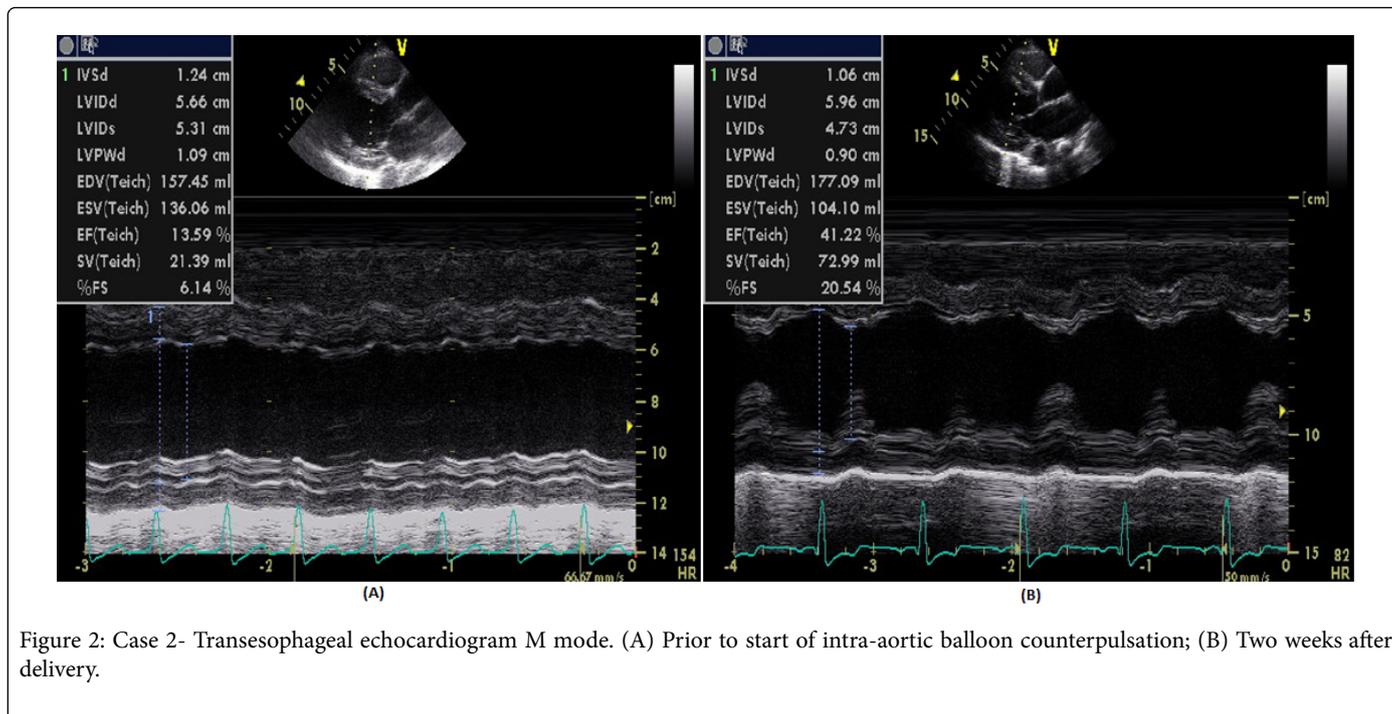


Figure 2: Case 2- Transesophageal echocardiogram M mode. (A) Prior to start of intra-aortic balloon counterpulsation; (B) Two weeks after delivery.

Discussion

PPCM has some distinct geographic features. The incidence among women of the Zorian tribe in Nigeria is one in every 100 births; among Haitian women, one in every 350 births [4]. PPCM in the United States occurs in approximately one in every 3000-4000 births [5]. PPCM is not distinguishable from other forms of primary dilated cardiomyopathy. A number of disparate proposed mechanisms lead to PPCM, each with some plausible evidence. These include myocarditis of non-infectious and infectious origin, Chlamydia pneumoniae in particular; auto-immune processes; dietary selenium deficiency; excessive salt intake; excess relaxing production; and unbalanced oxidative stress generating a cardiotoxic prolactin fragment. Management of women with PPCM presents considerable challenges for hemodynamic stability. The large changes in heart rate and cardiac preload and afterload that occur during labor lead to a recommendation that women with PPCM undergo Cesarean section.

During pregnancy, increases in blood volume, i.e. preload, and cardiac output induce remodeling of the left ventricle. Transient left ventricular hypertrophy in response to the physiologic changes of pregnancy can impair left ventricular systolic function in susceptible individuals. Afterload reduction, by mechanical or pharmacologic means, facilitates systolic function and restores normal left ventricular filling pressures, which improve cardiac output. Left ventricular systolic dysfunction in PPCM benefits from the decreased afterload with balloon deflation in systole. The intra-aortic balloon assists cardiac function also by increasing coronary blood flow during diastole [6].

Pre-eclampsia creates an additional afterload burden to a dysfunctional left ventricle leading to symptoms of heart failure. Afterload reduction, a main treatment modality for heart failure, becomes doubly important in pre-eclampsia. In each case described in this report, caregivers attempted pharmacologic afterload reduction, using epidural blockade in the first case, and labetalol infusion in the

second case. Titrated epidural block in the first case did not engender an adverse fetal response and successfully allowed instrumentation to measure pulmonary vascular pressures, which guided subsequent afterload reduction. The second patient developed fetal bradycardia with labetalol infusion, leading to abandonment of pharmacologic techniques in favor of a mechanical solution, the IABP. Counterpulsation simultaneously reduces cardiac afterload and augments forward cardiac output. The different fetal responses could have arisen from a host of factors other than the choice of afterload reduction strategy, including accumulated metabolic stress, differences in placental perfusion, presence or absence of maternal diabetes.

We avoided regional anesthesia in the second case, based on the adverse fetal response to afterload reduction using labetalol. Changes in sympathetic tone with regional anesthesia can be substantial and poorly reversed, despite epidural titration. Even modest hemodynamic alterations, without the mechanical support of an IABP, could have proven catastrophic for the first mother and her baby. This, together with confidence in the ability to secure the patient's airway despite her body habitus, led caregivers to choose general anesthesia for Cesarean section. IABP placement carries risks of post-operative hemorrhage, should anticoagulation be needed during balloon operation. Maintaining balloon motion minimizes the need for anticoagulation.

In contrast, the first patient had not failed a pharmacologic approach to afterload reduction, so gradual establishment of epidural block presented an opportunity to improve her cardiac output by decreasing peripheral resistance without the encumbrance of mechanical support [7]. Indeed, systemic blood pressure increased, rather than decreased, with increasing cephalad block, despite fluid restriction. Her pulmonary pressure decreased, most likely from pooling of blood in venous capacitance vessels as well as decreased impedance to left ventricular ejection. PPCM presents significant challenges to anesthesia caregivers: cardiac and respiratory distress limit management choices, as can concomitant pre-eclampsia. These cases underscore the need for early consideration of titrated epidural

blockade for afterload reduction and ample communication among caregivers to achieve successful results for both mother and neonate.

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