Cardiac Arrest in a Patient with History of Peripartum Cardiomyopathy

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Introduction: Peripartum cardiomyopathy (PPCM) is the development of cardiac failure in the last month of pregnancy or within five months after delivery, in the absence of both an identifiable cause of cardiac failure and of recognizable heart disease prior to last month of pregnancy. In the US, the incidence is predicted to be 1 per 3000 to 4000 births. We present an unusual case of sudden cardiovascular compromise, which developed prior to cesarean delivery in a patient with history of PPCM.

Case History: A 36 year-old G6P2 African-American female presented at 38-weeks gestation for possible preeclampsia. Past medical history was relevant for PPCM (diagnosed 9 years ago), along with chronic hypertension, asthma, and obstructive sleep apnea. Physical examination revealed a BMI 59.9, HR 107, BP 150’s/90’s, FHR 130’s, and 2+ pretibial pitting edema. A recent transthoracic echocardiogram showed normal LV systolic function (EF >55%) with moderate LV hypertrophy. Given her history, decision was made to proceed with urgent cesarean section under spinal anesthesia with invasive hemodynamic monitoring. During placement of central venous catheter, the patient felt claustrophobic and sat up; she quickly became dyspneic, and then unresponsive. Patient was immediately positioned supine with left uterine displacement; SBP was in the 60’s, with FHR in the 70’s. She was immediately intubated following rapid sequence induction with IV propofol and succinylcholine. With nonpalpable pulses, chest compressions were initiated and multiple boluses of IV epinephrine were administered. Fetus was delivered immediately by c-section. Resuscitation was continued, and femoral pulse became palpable following delivery of the fetus. Chest compressions were discontinued and dopamine and epinephrine infusions were initiated. An intraoperative transesophageal echocardiogram showed severe LV hypertrophy with septum measuring 2.5cm; EF was estimated to be >55% with significant reduction of LV cavity size. Aggressive fluid replacement with Voluven (1.5 L), PRBC (1 U), and LR (2 L) improved the patient’s hemodynamic status and inotropic infusions were discontinued. The patient was transported to the ICU in fair condition; she was extubated on POD#2 and discharged home on POD#5.

Discussion: Sudden cardiac arrest in this patient with a history of PPCM displayed an interesting picture. In spite of her history, poor left ventricular function, thromboembolism or myocardial infarction did not appear to be causative factors. The cardiac decompensation resulted from severe hypovolemia; probable contributory factors included intravascular volume depletion from chronic hypertension, inadequate fluid intake, and peripheral vasodilation. Her history of PPCM initially restrained us from aggressive fluid resuscitation; severe hypovolemia was not considered in the differential diagnosis until intraoperative echocardiogram provided the direction for medical management.