Acute peripartum cardiomyopathy after cesarean section – A case report –

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We experienced a case of acute postpartum cardiomyopathy after cesarean section. A twin-pregnant woman at 36 weeks gestation showed pregnancy-induced hypertension. After an emergency cesarean section, shortness of breath, paroxysmal dyspnea, hypertension, and tachycardia developed in the recovery room. She was diagnosed with acute peripartum cardiomyopathy.

Peripartum cardiomyopathy (PPCM) is a disorder of unknown cause in which initial left ventricular systolic dysfunction and symptoms of heart failure occur between the last month of pregnancy and the first 5 months postpartum.2) The clinical presentation of patients with PPCM is similar to that of patients with dilated cardiomyopathy. It can be diagnosed only by the exclusion of other causes of acute heart failure such as infections, metabolic disorder, and previously asymptomatic cardiac disease exacerbated by pregnancy. The incidence of PPCM varies with individual studies and has been reported to range from 1 in 1,300 to 15,000 pregnancies.3,4) Multiparity, twins, advanced maternal age, preeclampsia, gestational hypertension, and black race are known risk factors.3,5) Here, we present a case of acute postpartum cardiomyopathy after cesarean section.

CASE REPORT

A 28-year-old primigravida was diagnosed as twin pregnancy in a primary clinic. The patient visited our hospital at 14 weeks of gestation for further evaluation about high level of thyroid function test. She was diagnosed as hyperthyroidism and was given propylthiouracil (200 mg/day), she could keep the euthyroid state afterwards. Hypertension (160/80 mmHg) with proteinuria (+++; 100–300 mg/dL) was apparent at 35 weeks of gestation, diagnosed as severe preeclampsia. She gained body weight rapidly during pregnancy, and then pitting edema of the lower extremities was developed. She was recommended to enter the hospital and managed with magnesium, but she refused. At 36 weeks of gestation, she visited our hospital due to preterm labor pain, and her physician decided for emergency cesarean section. Preoperative electrocardiogram (ECG) was normal sinus rhythm, chest-X ray finding was within normal limits, and laboratory findings were in normal range except proteinuria (+++; 1,000 mg/dL↑). Initial vital sign in the operating room were: blood pressure, 160/110 mmHg; heart rate, 120 beats/min; respiration, 25 breaths/min; and O₂ saturation, 97%. The ECG showed sinus tachycardia. Following infusion of normal saline 400 mL, she was placed in the left lateral decubitus position, and combined spinal epidural (CSE) anesthesia was induced in the median approach via the L₃–₄ interspace using a CSE set (Espectan®). After tuohy needle was inserted into L₃–₄ epidural space by loss of resistance technique, spinal puncture was accomplished using a 27-gauge pencil point spinal needle. The spinal puncture was accomplished smoothly at the first attempt. After obtaining a free and clear flow of cerebrospinal fluid, 7.5 mg of hyper-
baric bupivacaine with 0.2 mg of epinephrine were injected intrathecally, and epidural catheter was inserted 3 cm into the L3−4 interspace. After she was turned in supine position, the blood pressure was 130/85 mmHg and slowly reduced. Five minutes after anesthesia, the blood pressure had fallen to 95/50 mmHg, we injected 10 mg ephedrine intravenously. The systolic blood pressure was maintained about 120−130 mmHg. Sensory blockade to the T4 level was confirmed by pinprick test. When the operation was started, the vital signs were: blood pressure, 120/70 mmHg; heart rate, 100 beats/min; respiration, 20 breaths/min; O2 saturation 98%. Cesarean section has been done without complications, and she delivered male twin. The first neonatal body weight was 2,740 g, one minute APGAR score was nine and five minutes APGAR score was ten. The other was 2,870 g, nine and ten. The total operation time was 55 minutes and total administered volume was 2,050 mL. Urine output and estimated blood loss during operation were 150 mL and 1,200 mL. Her vital signs were stable throughout the operation. She was then transferred to the recovery room for further observation.

One hour after arriving the recovery room, she became short of breath, and paroxysmal dyspnea appeared, her vital signs at this time were: blood pressure, 180/100 mmHg; heart rate, 140 beats/min; respiration, 40 breaths/min; O2 saturation 85%. The electrocardiogram showed sinus tachycardia (Fig. 1A). Since dyspnea became more severe, intubation was done, and she was treated for pulmonary edema with oxygen, furosemide, morphine sulfate, and water restriction. Esmolol was injected intravenously for high blood pressure and tachycardia management. After a postoperative chest X-ray checked in the recovery room, she was transferred to intensive care unit (ICU), and mechanical ventilator was applied. Arterial blood gas values at FiO2 1.0 were pH, 7.32; PCO2, 32.3 mmHg; PO2, 90.5 mmHg; HCO3− 16.8 mmHg; and O2 saturation, 96%. The postoperative chest X-ray showed pulmonary edema, pleural effusion, and cardiomegaly (Fig. 2A). She was treated with furosemide and labetalol. Six hours later, her blood pressure and heart rate were normalized, and the treatment for pulmonary edema were maintained. On the second postoperative dawn, she complained of chest tightness, at that time her vital signs were: blood pressure, 154/108 mmHg; heart rate, 173 beats/min; respiration, 16 breaths/min; O2 saturation 88%. ECG showed sinus tachycardia with nonspecific-ST change (Fig. 1B).