ANESTHETIC MANAGEMENT OF A PREGNANT PATIENT WITH ACUTE PULMONARY EDEMA AND PERIPARTUM CARDIOMYOPATHY: A CASE REPORT

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Peripartum cardiomyopathy is a reason for pregnancy-associated pulmonary edema. Peripartum cardiomyopathy typically develops during the last month of, and up to 6 months after pregnancy in women without known cardiovascular disease. It is a rare and very severe type of idiopathic cardiomyopathy, associated with very high maternal mortality rate which can reach 28% despite adequate therapy[1]. We present the anesthetic management for a pregnant patient with preemplasia, acute pulmonary edema and peripartum cardiomyopathy.

Case Report

A 26-year-old woman, gravida 3 para 0 with symptoms of palpitation and shortness of breath, who could not lie down for 2 days was admitted to our hospital at 39 weeks and 3 days gestation. The patient's body weight was 75 kg and height was 165 cm. She was healthy before pregnancy without significant medical history and didn't undergo any prenatal examinations. A written consent was achieved from the patient for publishing this case report was after her discharge.

Physical examination revealed axillary temperature of 36.8℃, high blood pressure (142/106 mmHg) with a regular heart rate of 152 beat/min and respiratory rate of 42 breath/min. On admission, cyanosis, dyspnea, orthopnea and jugular venous distention were observed. Mild pitting edema was found over both lower extremities. The liver was enlarged with its lower edge felt 1cm subcostal. Chest auscultation revealed moist rales over bilateral lungs. No heart murmurs were detected over each auscultatory valve area.

The patient's electrocardiogram indicated sinus tachycardia with T waves changes. The chest X-ray demonstrated features of pulmonary edema and cardiomegaly. An echocardiogram revealed an ejection fraction of 36% with an enlargement in the left ventricle and left atrium without left ventricular hypertrophy [Fig. 1].

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The fetus had a fast heart rate which was regular as indicated by Doppler. The patient was transferred to the operating room with oxygen received through a mask for an emergent cesarean section 1 hour after admission. She was positioned supine with left side tilt, and then electrocardiography, pulse oximetry and blood pressure were monitored. Blood pressure was 127/93 mmHg with a heart rate of 134 beats/min. Respiratory rate was 31 breaths/min and pulse oxygen saturation was 93%. Radial artery was cannulated and invasive blood pressure was measured. Fluid infusion was restricted.

After disinfection of the surgical area and sterile drapes placed, 100 μg remifentanil and 60 mg propofol were injected slowly. When the patient lost consciousness, the operation began. At the same time, rapid induction was performed with an injection of 100 mg succinylcholine to facilitate intubation using a 7.0-cuffed tracheal tube. Mechanical ventilation with respiratory rate of 12 breaths/min and positive end expiratory pressure of 2 cmH_2O was set. Anesthesia was maintained with continuous infusions of propofol at a rate of 400 mg/h and remifentanil at a rate of 0.7 mg/h. At 5 min after the beginning of surgery, a baby was delivered and treated by the neonatal team. The baby's APGAR scores at 1 minute and 5 minutes were 6 and 8, respectively. Intravenous 0.4 mg cedilanid and 10 mg furosemide were administered to the mother to prevent progression of heart failure. Fentanyl and cisatracurium were administered intermittently as needed after the umbilical cord was clamped. Internal jugular vein catheterized for central venous pressure measurement. About 15 min after delivery of the baby, blood pressure 127/74 mmHg with heart rate 156 beats/min, SPO_2 of 91% and central venous pressure of 26 cmH_2O were observed. Pink frothy sputum started to flow out from the endotracheal tube. Secretions were suctioned. At the same time intravenous furosemide 20 mg, dexamethasone 10 mg and doxofylline 100 mg were given. Milrinone was infused at the rate of 0.25 μg/kg·min. Nitroglycerine infusion continued and were titrated to maintain the systolic blood pressure less than 120 mmHg and more than 90 mmHg. At the end of surgery, moist rales were reduced significantly, SPO_2 was 96% with a FiO_2 of 100% and central venous pressure was 17 cmH_2O. The duration of surgery was 40 min, estimated blood loss was 200 ml and urine output was 400 ml. A total of 100 ml of 5% glucose solution was given during the entire period in the operative room.

The patient was shifted to the intensive care unit on mechanical ventilation with a FiO_2 of 100%. The patient was weaned off from mechanical ventilation and extubated 10 hours after entering the intensive care unit. The patient received inotropic agents, diuresis and vasodilators postoperatively. Her ejection fraction increased to 47% on the next echocardiogram examination performed 48 hours later. She was discharged after 14 days of hospital stay.

Discussion

Demakis et al. provide diagnostic criteria for peripartum cardiomyopathy: 1: the development of heart failure during the last month of pregnancy or within 5 months after delivery; 2: the absence of determinable etiology for heart failure and 3: the absence of heart disease before the last month of pregnancy[2]. In order to make the definition of peripartum cardiomyopathy more precise, echocardiographic result is adopted in the diagnostic creteria: 1: left ventricular ejection fraction <45%, 2: left ventricular fractional shortening <30% or both, and 3: left ventricular end-diastolic dimension >2.7 cm/m^2 body surface area[3]. The Heart Failure Association of the European Society of Cardiology Working Group recommend the following simplified definition: "Peripartum cardiomyopathy is an idiopathic cardiomyopathy presenting with heart failure secondary to left ventricular systolic dysfunction towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is found. It is a diagnosis of exclusion. The left ventricule may not be dilated but the ejection fraction is nearly always reduced below 45%"[1]. These criteria help to distinguish peripartum cardiomyopathy from other types of dilated cardiomyopathy.

In this case, the diagnosis of peripartum cardiomyopathy was based on the occurrence of heart failure in the last weeks of pregnancy, no other reasons for heart failure detected, except for pregnancy, and an ejection fraction of 36% revealed by an echocardiogram.

Based on the history, clinical presentation and

laboratory test results, the patient is in an extremely high-risk condition with New York Heart Association class III-IV[4]. The anesthetic management of pregnant patients with peripartum cardiomyopathy and pulmonary edema can be a major challenge, but it is not different from that of other forms of dilated cardiomyopathy. Basic principles of anesthetic management are needed to prevent dramatic changes in cardiac preload and afterload as well as suppression of cardiovascular system.

Remifentanil and propofol which both can cross the placenta appear to be rapidly metabolized by both the mother and the fetus[5,6]. The combination of remifentanil and propofol can reduce the incidence of maternal awareness, inadequate analgesia and hypertensive response resulting from endotracheal intubation and surgical incision. However, the use of remifentanil and propofol for anesthetic induction for caesarean delivery is still controversial. Therefore, we chose to shorten the time from administration of anesthetics to delivery of the baby. Fentanyl which is associated with a more need for neonatal resuscitation[7] was avoided during induction.

Even though actions were taken to prevent deterioration of heart failure after delivery, severe acute pulmonary edema still developed. More blood getting into circulatory system because of uterine contractions and fluid shifts after delivery can change the condition of patients with peripartum cardiomyopathy, increase the cardiac preload, and then lead to acute heart decomposition[8]. What's more, the patient has preeclampsia which is related with a high risk of pulmonary edema because of decreased colloid osmotic pressure, increased capillary permeability, increased hydrostatic pressure and cardiac diastolic dysfunction[9].

With the combination of preeclampsia and heart failure, it is very important to distinguish whether it is peripartum cardiomyopathy in preeclamptic patient or heart failure as a complication of preeclampsia. In the present case, echocardiographic results helped to diagnose peripartum cardiomyopathy based on heart failure with decreased ejection fraction and without left ventricular hypertrophy. Diastolic dysfunction, preserved ejection fraction, left ventricular hypertrophy, and non-dilated ventricles are specific for preeclampsia[10].

In this case, peripartum cardiomyopathy has been described as a cause of severe heart failure. Although peripartum cardiomyopathy occurrence is rare, it can vitally jeopardize both the mother and the fetus. The clinical course of peripartum cardiomyopathy can be short. Progression from the earliest presentation of symptom to heart failure can be fast, very often within few days[1]. Delayed diagnoses can lead to higher incidence of complications and poor prognoses. When the diagnosis is made before ejection fraction declines less than 35%, the mortality rate is near 0, and the chance of full recovery is much higher. The definitive diagnosis of peripartum cardiomyopathy is based on echocardiographic findings which enable prompt diagnosis and evaluation of heart function[1]. Therefore, in any case of suspicion of peripartum cardiomyopathy, bed-side echocardiography should be performed as soon as possible.

We have described a successful anesthetic management of a parturient with preeclampsia, severe acute pulmonary edema and peripartum cardiomyopathy undergoing cesarean section under general anestheisa. In this case, the patient had stable hemodynamics during surgery and an uneventful postoperative course.

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