Case Report

Peripartum Cardiomyopathy with Respiratory Failure and Cardiac Arrest

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Keywords
Peripartum cardiomyopathy · Heart failure · Acute pulmonary edema

Abstract
We describe the case of a 33-year-old female who went into cardiac arrest outside the hospital 7 days postpartum. We diagnosed her with peripartum cardiomyopathy (PPCM). After the return of spontaneous circulation, she suffered from acute pulmonary edema and hypoxia. The patient received intensive care after gaining return of spontaneous circulation. We also present an effective use of venovenous extracorporeal membrane oxygenation (VV-ECMO), which led to a rather short stay in the intensive care unit (ICU). An echocardiogram showed global hypokinesis with an ejection fraction of 28% and a left ventricular dilation with a diastolic dimension. The patient’s lungs recovered steadily during her stay in the ICU. VV-ECMO was disconnected on the seventh day of hospitalization, and intubation was withdrawn on the tenth day. On the thirteenth day, she was released from the ICU and transferred to another hospital.

If a pregnant or postpartum woman presents with cardiopulmonary arrest, heart diseases such as PPCM should be considered.

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Introduction

Peripartum cardiomyopathy (PPCM) is an idiopathic cardiomyopathy, a type of acute heart failure due to left ventricular (LV), systolic dysfunction within 1 month prior to or 6 months after childbirth. Patients with this condition have typically no previous history of heart disease. In addition, about 50% of the pregnant women with severe ventricular dysfunction recovered within 5 months after delivery [1].

Common symptoms of PPCM consist of dyspnea, cough, and hypertension [1], and may are followed by severe heart failure within a few hours to a few days [2–4]. Most of the fatal cases of PPCM occurred during pregnancy or developed during in-hospital delivery, but not often postpartum [2]. The treatment of PPCM can be similar to that use in other types of heart failure. Medical therapy would be the first choice for women with PPCM. In case that medical therapy went unsuccessful, a use of extracorporeal membrane oxygenation (ECMO) has been of benefit in women with severe heart failure [5], but so far, the efficacy of this treatment is unknown.

We describe the case of severe PPCM with cardiac arrest (CA) after dyspnea without prodromes and hypoxemia by acute pulmonary edema. We also utilized venovenous-ECMO (VV-ECMO).

Case Presentation

On her seventh day postpartum, a 33-year-old Black female, gravidity 4 and parity 4, was transferred by an ambulance to our institute as a tertiary emergency medical center in the area because of acute respiratory failure after CA and a need for advanced and intensive care.

Initially, she complained of dyspnea and lost consciousness outside the hospital on the same day as she was transferred. When she first arrived at another medical institute, she was in CA with ventricular fibrillation. After tracheal intubation and two defibrillation attempts in the hospital, she gained return of spontaneous circulation (ROSC). However, due to acute respiratory failure, she was transferred to our hospital and admitted to the intensive care unit (ICU). She had been diagnosed with pregnancy-induced hypertension during her third gravidity but not during the last one and was discharged immediately after the last parturition.

On the arrival at our hospital, the patient’s initial vital signs represented as following: blood pressure 125/89 mm Hg, heart rate 159 beats/min, and 48% oxygen saturation on a ventilator, P/F ratio of 40. There were no physical findings. Arterial blood gas tests revealed a PH of 7.13, PaCO₂ 83.2 mm Hg, PaO₂ 33.8 mm Hg, FiO₂ 1.0, and bicarbonate 26.7 mEq/L. Furthermore, her troponin level was 0.44 ng/mL, plasma NT-proBNP level 96.7 pg/mL, creatinine kinase level 306 U/L, and creatinine kinase MB level 53 U/L. Electrocardiography showed sinus tachycardia of 135 beats/min and a left-bundle branch block. We performed a chest radiograph and computed tomography (CT), revealing severe pulmonary edema in both lungs (Fig. 1a, b). In addition, echocardiography showed a dilated left ventricle with systolic dysfunction. At that time, we suspected acute coronary syndrome, but cardiac catheterization did not reveal myocardial infarction and occlusions.

On day 1, we started her on dobutamine, noradrenaline, vasopressin, and an intra-aortic balloon pump, which supported the patient’s circulation. We used a mechanical ventilator setting with a high positive end-expiratory pressure of 10. We utilized VV-ECMO, which also improved her respiratory function. We evaluated the following scores: an Acute Physiology and
Chronic Health Evaluation (APACHE) II score of 37, a Sequential Organ Failure Assessment (SOFA) score of 12, and a Murray score of 4 upon admission to the ICU.

The patient’s clinical course during hospitalization is shown in Figure 2. On day 3 in our ICU, echocardiography indicated LV diastolic failure, ventricular systolic dysfunction, mitral valve regurgitation, an E/A of 0.49, and an ejection fraction of 28%. Her plasma NT-proBNP level was 980 pg/mL, which was higher than upon admission. We reduced her medications gradually, while her blood pressure level was kept. Consequently, we removed the intra-aortic balloon pump on day 6 after ICU admission. Her respiratory function was improved, and her P/F ratio was increased above 300 after VV-ECMO insertion. On day 7, we removed the VV-ECMO and withdrew tracheal intubation on day 10. Because of her rather rapid improvement, we then began to plan her discharge from the ICU. Before the discharge, a comprehensive echocardiography revealed an ejection fraction of 44%. A chest radiograph indicated that her lung permeability was improved, compared to the date of admission (Fig. 1c). Additionally, she showed no disturbance of consciousness during the clinical course, so we presumed that there had not been cerebral hypoxia due to CA. She was discharged from our ICU and institute and was transferred to the hospital she was first admitted on the day 13.

Discussion and Conclusion

We described a case of PPCM with CA, suffering from acute respiratory failure. The patient received ICU care at our hospital after gaining ROSC. We also presented an effective use of VV-ECMO, which led to a rather short stay at the ICU.

PPCM is rare in Japan. Several recent studies have assessed the incidence of PPCM in Japan at a rate of approximately 1 case in 20,000 deliveries, which is lower than in Europe and the United States [6]. However, our patient with multiple risk factors incidentally resided in Japan at the time of incidence. An epidemiological study reveals that risk factors consist of a maternal age of ≥30 years, Black race, multifetal pregnancy, preeclampsia, and gestational hypertension [5]. Some of these risk factors were present in our patient.

In addition, the patient presented some of the common symptoms such as dyspnea, chest pain, cough, neck vein distension, fatigue, and peripheral edema. Echocardiography of PPCM would indicate LV systolic dysfunction, and the pathophysiology could be similar to dilated cardiomyopathy. PPCM occurs between the last month of pregnancy and the first 5 months postpartum, and its onset is highest in the first week after childbirth [6]. In prior reports, sudden SpO2 decrease, stridor, and substantial dyspnea occurred a few days after the appearance of prodromes such as hypertension, mild dyspnea, and chest discomfort [2–4]. Those common symptoms of PPCM matched with those found in our patient.

The patient in this case report suffered from hypoxemia by acute pulmonary edema. Thus, we utilized VV-ECMO to improve her respiratory function. ECMO would be able to provide effective circulatory and respiratory support for a patient. Both venoarterial ECMO (VA-ECMO) and VV-ECMO could be considered devices for treating heart failure with hypoxia. The patient’s blood pressure was stabilized using medications, and we used VV-ECMO so that hypoxia would be decreased due to enhanced oxygenation. Then, the acute pulmonary edema was improved because hypoxemia was not perfused in the brain.

Instead of VV-ECMO, VA-ECMO might be a standard treatment for a patient such as the one described herein [7]. However, we chose to utilize VV-ECMO for the following reasons. First, upon arriving at our hospital, the patient had maintained circulatory functions for more than 90 min after ROSC, and we diagnosed her with PPCM on day 1. Given the patient’s
condition, using VA-ECMO might have induced differential hypoxia [8] or brain hypoxia due to the combination of the patient’s own circulation and ECMO. Veno-VA ECMO might have been another possibility for treating a patient the one described herein, but our hospital does not possess such equipment. Thus, for the aforementioned reasons, we chose to treat the patient with VV-ECMO instead of VA-ECMO.

We consider treating hypoxemia necessary for neurologic convalescence and improvement. An animal experiment involving rats found that a PaO\(_2\) level <50 mm Hg for 180 min influenced brain function; the normal value in rats is a PaO\(_2\) level of 90 mm Hg [9]. Of the sudden cardiac arrest patients, 29.2% returned to spontaneous circulation of Cerebral Performance Category 1–2 at the time of hospital discharge, in which hypoxemia could cause poor outcomes [10, 11]. Introducing VV-ECMO for severe respiratory failure after cardiac surgery [12], even if cardiac function decreases and circulatory dynamics remains, should be considered for respiratory function improvement.

The management of patients with PPCM could be similar to the standard treatment for other forms of heart failure. However, cardiac function may not improve, and CA could occur [3, 5]. A follow-up survey in Japan conducted from 2007 to 2009 found 4 deaths (4%) among 102 cases; 3 parturient cases were fatal after <1 week, and 1 postpartum case died after 6 months [6]. Dilated cardiomyopathy generally has a mortality rate of approximately 8% [13]. There have been reports of fatal cases of PPCM, but most occurred during pregnancy or developed during in-hospital delivery; there are no reports of cases that developed outside the hospital. Causes of death in pregnant and parturient women in acute crisis included massive bleeding and obstetrical embolism [14].

In conclusion, our patient with PPCM who was in CA and gained ROSC improved rather quickly after ICU care for respiratory failure. A VV-ECMO would be a choice of treatment for PPCM with acute respiratory failure. If a pregnant or postpartum woman presents with cardiopulmonary arrest, heart diseases such as PPCM should be considered.

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Statement of Ethics

The authors have no ethical conflicts to disclose.

Disclosure Statement

The authors have no conflicts of interest to declare.

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Author Contributions

S.M., H.T., and T.D. made substantial contributions to the study conception and design as well as the acquisition of data. S.M., T.A., and I.T. made substantial contributions to the analysis and interpretation of the data. All authors were involved in drafting the manuscript and critically revising it, and all gave approval to the final version.

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Fig. 1. Chest radiograph and CT on days 1 and 13. a Chest radiograph showing severe pulmonary edema in both lungs. b CT presenting severe pulmonary edema in both lungs. c Lung permeability was improved on day 13 compared to day 1, the day of admission.

| DOB(µg/kg/min) | 3 | 1 | 1.5 | 1 | 0.8 | 0.6 | 0.4 |
|---------------|---|---|-----|---|-----|-----|-----|
| AVP(U/hr)     | 1.8 | 0.7 |
| NAD(µg/kg/min)| 0.5 | 0.1 |
| hANP(µg/kg/min)| 0.2 |

Fig. 2. The patient’s clinical course during hospitalization showing a steady improvement and a rather rapid discharge. DOB, dobutamine; AVP, arginine vasopressin; NAD, noradrenaline; hANP, human atrial natriuretic peptide; IABP, intra-aortic balloon pump.