CASE REPORT

An Unusual Case of Peripartum Cardiomyopathy in a Parturient With Preeclampsia

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Here we report an unusual development of peripartum cardiomyopathy (PPCM) in a parturient woman with preeclampsia. A 36-year-old nulliparous parturient woman underwent elective cesarean section for delivery of twins under spinal anesthesia. Both preoperative workup and past history were unremarkable except for proteinuria and hypertension for 1 week. Approximately 4 hours after cesarean section, progressive orthopnea developed. Chest plain film showed acute pulmonary edema, bilateral pulmonary infiltration with interstitial patches, and cardiomegaly. Postpartum cardiomyopathy was diagnosed afterward by echocardiography. This showed general hypokinesia and severe dysfunction of the left ventricle with ejection fraction of 15−20%. She was admitted to the intensive care unit for further management. Fortunately, the patient recovered after treatment and was discharged 15 days later. This case illustrates that we should bear in mind the possibility of PPCM if orthopnea develops while delivery is approaching in a parturient with preeclampsia. Echocardiography is helpful for early diagnosis of PPCM.

1. Introduction

Many complicated obstetric conditions, including amniotic fluid embolism, postpartum hemorrhage, preeclampsia with peripartum cardiomyopathy (PPCM) and HELLP syndrome (hemolysis, elevated liver enzymes and low platelets) have been reported, of which preeclampsia is responsible for up to 8% of complicated pregnancies. In contrast, PPCM is rarely observed and has an unknown etiology. PPCM is characterized by an acute onset of heart failure within 1 month before delivery or 5 months postpartum.¹,²

A number of factors are thought to increase the risk of developing PPCM. Here, we report an unusual case of PPCM that developed in combination with preeclampsia. We also review the risk factors, new treatment options and the outcomes of subsequent pregnancies in women with PPCM.

2. Case Report

A 36-year-old woman, gravida 0, para 0, and bearing twins, presented at our obstetric clinic to investigate...
irregular labor contraction at 37 4/7 weeks of gestation. The patient had an unremarkable medical history. At presentation, she had been experiencing slight shortness of breath for 1 week. Bilateral lower leg edema and hypertension were found. Consequently, a cesarean section delivery was suggested under the impression of preeclampsia. Preoperative evaluations, including laboratory studies, were unremarkable. In addition, her vital signs were relatively stable after management in the ordinary ward.

On the day of surgery, she was slightly nervous and her vital signs on arrival at the operating room were as follows: blood pressure was 170/110 mmHg, heart rate was 130 beats/minute and respiratory rate was 20–25 breaths/minute. Spinal anesthesia was performed with 11 mg of bupivacaine. Surprisingly, her tachypnea subsided after spinal anesthesia and the operation and delivery proceeded uneventfully. The total intraoperative fluid given was approximately 800 mL of normal saline. She was sent to the postanesthesia room for close observation, where she received 25 mg of pethidine (25 mg) to control her shivering. She was then returned to the ordinary ward with stable vital signs.

However, 4 hours after the cesarean section, she exhibited progressive orthopnea (respiratory rate: 25 breaths/minute). A chest X-ray (Figure 1) was taken and revealed bilateral upward pulmonary infiltration with opaque interstitial patches, pleural effusion and cardiomegaly. Electrocardiography (Figure 2) showed normal sinus rhythm, left atrial enlargement and poor R wave progression in leads V1–V4. Moreover, arterial blood gases analysis revealed that her PaO2 was 165 mmHg while breathing oxygen at a flow rate of 10 L/minute via a non-rebreathing mask. As her condition continued to worsen, she was transferred to an intensive care unit (ICU) for further management. Echocardiography was done immediately on arrival at the ICU and showed general hypokinesia with severe left ventricular dysfunction (ejection fraction: 15–20%). Therefore, PPCM was diagnosed by the cardiologist. Consequently, fluid restriction was started, and diuretics, β-blockers and inotropic agents were administered to treat heart failure. Right cardiac catheterization was also performed but revealed no significant findings. As her condition showed improvements after 5 days in the ICU, she was returned to a general ward and was discharged 15 days later with limited ambulation. Six months later, when she came for follow up, her cardiac function status was defined as congestive heart failure, New York Heart Association Functional Class II, and her daily activity was steadily improving.

Figure 1 Chest X-ray shows cardiomegaly, interstitial infiltration and patch opacities over both lung fields, and bilateral pleural effusion.

Figure 2 Electrocardiography shows normal sinus rhythm, left anterior fascicular block, left atrial enlargement, and poor R wave progression.
3. Discussion

Here, we report a parturient who developed PPCM 4 hours after cesarean section delivery of twins. One week before delivery, she was diagnosed with preeclampsia because of hypertension, lower limb edema, shortness of breath and irregular uterine contraction. PPCM is a rare parturient disorder of unknown cause that is characterized by acute onset of heart failure within 1 month before delivery to 5 months postpartum.1,2 PPCM is defined based on four criteria: (1) development of cardiac failure in the last month of pregnancy or within 5 months postpartum; (2) the absence of an identifiable cause for the cardiac failure; (3) absence of recognizable heart disease before the last month of pregnancy; and (4) left ventricular systolic dysfunction based on classic echocardiographic criteria.3 The incidence of PPCM is currently estimated to be approximately 1 in 3000 to 1 in 4000 live births. However, it must be noted that population-based estimates are not currently available and the diagnosis of this rare disease is not always straightforward.4

The diagnosis of PPCM relies on the echocardiographic identification of left ventricular systolic dysfunction, which happens before the narrow period of late pregnancy and postpartum. Differentiating subtle symptoms of heart failure (exercise dyspnea, fatigue, and pedal edema) from normal findings in late pregnancy is a challenge for cardiologists. Therefore, it is essential to consider the presence of PPCM if the symptoms are ambiguous. In our case, the tachypnea subsided after spinal anesthesia. This might result from the vasodilatory effect of spinal anesthesia, which decreased the systemic vascular resistance and improved the performance of the heart. However, progressive orthopnea developed 4 hours after cesarean section, which suggests that the improvements in systemic vascular resistance increased the work of the heart again. Thus, it is possible that the spinal anesthesia masked the signs of heart failure and prevented the clinician from diagnosing PPCM. Therefore, we should cautiously manage parturients who develop dyspnea during delivery.

The etiology and risk factors of PPCM are unknown. A number of articles have proposed various mechanisms and presented conflicting evidence for the pathogenesis of PPCM, including viral myocarditis, abnormal immune responses to pregnancy, abnormal responses to the hemodynamic stress of pregnancy, accelerated myocyte apoptosis, cytokine-induced inflammation, malnutrition, genetic factors, excessive prolactin production, abnormal hormonal function, increasing adrenergic tone, and myocardial ischemia.5 Of note, excessive prolactin production has been reported to play a marked role in the pathogenesis of PPCM in pregnant mice and women.6 Accordingly, it has been reported that bromocriptine, a dopamine 2 receptor antagonist, which inhibits prolactin secretion in combination with standard therapy, can improve the symptoms of PPCM. In addition, a number of factors are thought to increase the risk of PPCM.7 However, routine screening for PPCM in this high-risk population is very difficult to recommend, unless the risk factors can be confidently identified. Although PPCM is a rare complication of preeclampsia, anesthesiologists and intensivists should be aware of the likelihood of PPCM in parturients with preeclampsia.8

In the absence of systematic clinical studies to compare the therapeutic approaches to treat PPCM, the standard therapeutic modalities for heart failure, including salt restriction, diuretics, vasodilators and digoxin, should be initiated. However, the clinician should consider neonatal safety because maternal excretion of drugs or their metabolites may be harmful to the neonate through breast feeding after delivery. Angiotensin-converting enzyme inhibitors given during late pregnancy and parturition are contraindicated because they may cause teratogenicity, neonatal anuric failure and neonatal death, but they should be considered as the primary treatment for PPCM after delivery.9 β-adrenoceptor antagonists have been reported to improve the overall survival in parturients with dilated cardiomyopathy.10 Additionally, atrial arrhythmia should be treated with digoxin, which may exert positive inotropic effects on PPCM. Nevertheless, Class III (e.g. amiodarone) and Class IV (e.g. verapamil) antiarrhythmic agents should be avoided because of their severe side effects, which may include fetal hypothyroidism and premature delivery.11

When cardiomyopathy occurs during late pregnancy, early delivery of the fetus is recommended to reduce hemodynamic stress on the maternal heart. The mode of delivery for a parturient with PPCM is generally based on obstetric indications.12 After optimizing the mother’s condition by the cardiologist and obstetrician, the induction of vaginal delivery can be attempted in most cases, with close cooperation with the consulting anesthesiologist. The advantages of vaginal delivery are little blood loss, greater hemodynamic stability, low risk of postoperative infection, and low incidence of pulmonary complications. Cesarean section delivery should be reserved for events in which it is indicated, such as fetal distress or failure of parturition to progress. For cesarean section delivery, regional or general anesthesia may be used, depending on the patient’s concurrent anticoagulation medications. In patients given general anesthesia, the anesthetic management should maintain a normal or acceptable heart
rate to decrease oxygen demand and prevent large fluctuations in blood pressure.12

The prognosis for PPCM largely depends on the recovery of left ventricular size and function within 6 months after delivery.13 Demakis et al reported that about half of the 27 women in their study with PPCM had persistent left ventricular dysfunction and the mortality rate was 85% in 5 years.14 Similar results were reported in a more recent study.15 In another study, it was suggested that patients with PPCM recovering from left ventricular systolic dysfunction should be followed-up for 6–12 months after establishing diagnosis.16 It is very important to provide continuous treatment with follow-up for an adequate period of time to avoid further decline in heart function.

Finally, the risks associated with subsequent pregnancies in women with sustained PPCM should be understood by clinicians and communicated to patients. It has been reported that, among women with normal left ventricular function after PPCM, 23% develop cardiac dysfunction and 2% die during subsequent pregnancies.17 Among women with persistent left ventricular dysfunction after PPCM, 54% develop cardiac dysfunction and 9% die in subsequent pregnancies. Thus, a woman with a history of PPCM should be aware of the risks involved if she wishes to conceive again.17

In conclusion, our case illustrates that PPCM should be suspected and an echocardiography is essential if orthopnea develops in a parturient with preeclampsia in the late stage of pregnancy, in parturition or postpartum.

References


