Case Report

Cardiac arrest during emergency cesarean section for severe pre-eclampsia and peripartum cardiomyopathy

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ABSTRACT

Objective: To present the correlation between severe pre-eclampsia and peripartum cardiomyopathy in pregnancy.

Case report: A 41-year-old parturient, gravida 3, para 1, at 34 4/7 weeks' gestation, was diagnosed with severe pre-eclampsia. At the time of admission, a plain chest film disclosed bilateral pleural effusions. An emergent cesarean section was planned because of decreased fetal movement, heavy daily protein loss, and bilateral pleural effusions. During the cesarean section, she developed shock with acute respiratory failure. She underwent advanced cardiac life support and intubation with mechanical ventilator support. Peripartum cardiomyopathy was subsequently diagnosed by echocardiography.

Conclusion: The presented case demonstrates that routine echocardiography is highly recommended for suspected peripartum cardiomyopathy in gravidas with severe pre-eclampsia and symptoms or signs of heart failure.

Introduction

Peripartum cardiomyopathy, which is characterized by systolic cardiac dysfunction, presents in the last month of pregnancy or within 5 months of delivery in women without pre-existing cardiac disease [1]. The diagnosis of peripartum cardiomyopathy is confirmed by the exclusion of other underlying disorders and strict echocardiographic indications of left ventricular dysfunction [2]. The reported incidence of peripartum cardiomyopathy varies globally and ranges from 1 in 1421 to 1 in 9861 deliveries [3]. The etiology remains unknown.

Peripartum cardiomyopathy, which is an infrequent complication of severe pre-eclampsia [4], is a potentially fatal disease [1]. Severe pre-eclampsia rarely causes heart failure alone, although it is a significant risk factor for peripartum cardiomyopathy [5]. Bello et al [6] report that patients with peripartum cardiomyopathy have a prevalence of pre-eclampsia that is four times the average worldwide rate that is expected in the general population. A recent study suggests that peripartum cardiomyopathy and pre-eclampsia may share similar pathophyslogic features [7]. The levels of antiangiogenic factors, which are markedly elevated in pre-eclampsia [8], may cause an angiogenic imbalance in susceptible parturients, and thus trigger the development of peripartum cardiomyopathy [9].

We present a patient with orthopnea and severe pre-eclampsia who developed cardiogenic shock and acute respiratory failure during an emergent cesarean section. After undergoing acute cardiac life support, spontaneous circulation returned. She was subsequently diagnosed as having peripartum cardiomyopathy and congestive heart failure, based on echocardiography.

Case Report

A 41-year-old woman, gravida 3, para 1 at 34 4/7 weeks of gestation, presented to our obstetric emergency department on referral from a local clinic. She had progressive dyspnea and chest tightness. The patient had a benign medical history before pregnancy. Two months earlier, a prenatal examination at a local clinic disclosed hypertension, which she ignored without treatment. At presentation, she had been experiencing shortness of breath for 1 week. Bilateral lower leg edema and hypertension were observed.
She reported having headaches. The initial vital signs were as follows: blood pressure, 165/110 mmHg; heart rate, 128 beats/min; respiratory rate, 23 breaths/min; temperature, 36.1°C; and oxygen saturation, 96–97% on 3–4 L/min by oxygen cannula. She underwent laboratory tests to investigate pre-eclampsia (i.e., complete blood cell count, complete metabolic profile, liver function tests, coagulation tests, and urinalysis). A plain chest film showed bilateral pleural effusions. The v-dimer level was 3540 ng/mL. Electrocardiography demonstrated sinus tachycardia. Cardiac enzymes were within normal limits. Dipstick urinalysis revealed 4+ protein. Based on these findings, the patient was admitted with a diagnosis of severe pre-eclampsia. A pulmonary embolism could not be excluded.

On admission, management of the patient included a magnesium infusion for seizure prophylaxis, betamethason to achieve fetal lung maturity, and enoxaparin to prevent pulmonary embolism. On the following day, an emergent cesarean section was performed because of decreased fetal movement, heavy daily protein loss (i.e., the urine protein collected was 10.542 g/24 h), and bilateral pleural effusions. During the cesarean section under spinal anesthesia, she developed loss of consciousness with shock and acute respiratory failure. She received acute cardiac life support and intubation with mechanical ventilation. For shock and severe hypovolemia, spontaneous circulation returned within 1 minute after initiating cardiopulmonary resuscitation and she regained consciousness. The intubated patient was transferred to the intensive care unit. A computed tomography scan of the brain disclosed no hemorrhage and no hypodense lesions. A computed tomography scan of the chest revealed no evidence of pulmonary embolism, confirmed the presence of bilateral pleural effusions. An echocardiogram revealed moderate mitral regurgitation, dilation of the left ventricle, and general hypokinesia with severe left ventricular dysfunction (i.e., ejection fraction of 25–30%), but excluded pericardial effusion or cardiac tamponade. The patient was diagnosed as having peripartum cardiomyopathy in pregnancy, based on the echocardiogram and on clinical symptoms of congestive heart failure.

Diuretics, β-blockers, angiotensin-converting enzyme inhibitors, and spironolactone were administered to treat heart failure and fluid restriction, and a low sodium diet was initiated. A massive pleural effusion, which proved to be a transudate from thoracentesis, improved after the insertion of a chest tube and correction of the hyypoalbuminemia. Because her condition improved, the patient was extubated on Postoperative Day 10. She was returned to a general ward on Day 12, and subsequently discharged on Day 15. At follow up, 6 months after discharge, the ejection fraction had improved to 40–50%.

**Discussion**

The diagnosis of peripartum cardiomyopathy in the current patient was based on the following criteria: (1) development of heart failure in the last month of pregnancy or 5 months postpartum; (2) no history of pre-existing heart disease; (3) unknown etiology for heart failure; and (4) echocardiography findings include a left ventricular ejection fraction less than 0.45, or M-mode fractional shortening <30%, or both, and an end-diastolic dimension >2.7 cm/m² [1,5,10,11].

Prompt diagnosis of peripartum cardiomyopathy is essential. When approaching patients with symptoms or signs of heart failure and severe pre-eclampsia, clinicians should always list peripartum cardiomyopathy as a differential diagnosis. Because symptoms or signs of heart failure can be ambiguous in women with peripartum cardiomyopathy, compared to women with a normal pregnancy, echocardiography used to determine left ventricular systolic function can help detect patients with undiagnosed peripartum cardiomyopathy earlier, and thereby facilitate earlier intervention and improve the outcomes of these patients [10].

Symptoms of peripartum cardiomyopathy are similar to common presentations of heart failure and include shortness of breath, dyspnea on exertion, palpitations, and edema [12,13]. Patients may be unable to lie down because of shortness of breath. Signs include jugular vein engorgement, displaced apical impulse, right ventricular heave, murmurs of mitral and tricuspid regurgitation, third heart sound, rales, and peripheral edema [13]. Pleural effusions can be a sign of heart failure, which can also be attributed to capillary leak syndrome and hypoalbuminemia present in the pre-eclamptic puerperium [14,15].

In our patient, the sudden collapse with altered consciousness during the cesarean section was presumably related to peripartum cardiomyopathy due to left ventricular systolic dysfunction. The vasodilatory effect of spinal anesthesia, which may cause a rapid drop in blood pressure, could be devastating [16–18]. The principles of anesthetic management of patients with peripartum cardiomyopathy is not well-established and opinions differ regarding the optimal method [18,19]. However, it is essential to collaborate with anesthesiologists for adequate anesthesia to minimize undesirable hemodynamic changes in patients undergoing emergent cesarean delivery with a suspected or confirmed diagnosis of peripartum cardiomyopathy. Our patient highlights the importance of cooperation among obstetricians, cardiologists, intensivists, and anesthesiologists in the evaluation and management of this challenging clinical situation.

**Conflicts of interest**

The authors have no conflicts of interest relevant to this article.

**References**


