

## Witnessed Sudden Cardiac Arrest and Peripartum Cardiomyopathy

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**Received:** September 15, 2020; **Published:** February 27, 2021

### Abstract

Witnessed sudden cardiac arrest, is one of the most serious cardiac emergencies which could improve dramatically if treated properly. If a woman present to the hospital with cardiac arrest in the late stage of pregnancy or five months after her delivery, it is very likely that she has peripartum cardiomyopathy (PPCM). Since the symptoms of PPCM are similar to the discomforts experienced during pregnancy and lack specificity, the early diagnosis and prevention of PPCM is very difficult. As a result, patients may easily overlook their heart defects during her peripartum period and thus have a poorer prognosis. Therefore, early detection of PPCM in order to avoid life-threatening situations is the biggest challenge in peripartum care. The patient was a 30-year-old pregnant woman without any discomfort symptoms of cardiac during pregnancy and who did not receive any examine about heart function. Unfortunately, the patient happened suddenly witnessed sudden cardiac arrest after 2 months of postpartum. Hope via describes the care experience, it provides first-line health care professionals and nurse practitioners to dig out the impact and risk of the disease for pregnant woman.

**Keywords:** Peripartum Cardiomyopathy; Pregnancy; Cardiac Arrest; Early Detection; Life-Threatening

### Introduction

Peripartum cardiomyopathy (PPCM) is an idiopathic cardiomyopathy, which tends to occur in the last month of pregnancy or within five months following delivery. PPCM is a life-threatening disease and one of the main causes of maternal death during pregnancy [1,2]. PPCM mostly occurs in low- and middle-income countries and Nigeria has the highest incidence rate of 1 case out of 102 births. It is rare in developed countries. Approximately 1 out of 15,533 births in Japan and 1 out of 3,790 births in Taiwan [3]. A number of risk factors of PPCM have been identified, including diet, lifestyle, medication history, genetics, preeclampsia, hypertension, obesity, low family income, twin delivery [4]. Pregnant women suffering from PPCM may have one or more of following symptoms: breathlessness on exertion or rest, limb edema, palpitation, chest tightness, etc., that are easily confused with the symptoms of pregnancy discomfort and so early diagnosis is extremely difficult. Unless there is a cardiac echography examination during pregnancy, it is very easy to ignore the cardiac dysfunction during the perinatal period and this leads to poor prognosis or sudden cardiac death in 2.2% of pregnant women [5].

## Case presentation

A 30-year-old G1P1 woman. The patient is an office worker at an insurance company. She indicated that she does not have the habits of smoking, drinking, and chewing betel nuts. She does not have a family history of heart disease, stroke, hypertension, and diabetes. Her dietary and activity statuses during her pregnancy were normal, and she never experienced wheezing, dyspnea, or edema. However, her weight had dropped from 66 kg to 58 kg after her pregnancy. In 2011, she experienced temporary heart palpitations and arrhythmia. Her 24-hour electrocardiogram (EKG) indicated that she had premature ventricular contraction, premature atrial contraction, and left bundle branch block (LBBB). Echocardiography results showed that her left ventricular ejection fraction (LVEF) was 81% and she had mitral valve prolapse as well as premature ventricular contraction (1.1%). She has previously used  $\beta$ -blockers for treatment for two months, but withdrew of her own accord after experiencing whole body discomfort. In 2012, coronary computed tomography (CT) angiography results revealed myocardial bridging in the distal segment of her left anterior descending artery, but it was left untreated and she did not undergo regular follow-ups. By the end of 2016, she underwent echocardiography at a hospital in preparation for her pregnancy. The results showed that her LVEF was 56%, her left ventricular end diastolic diameter was 51 mm, and her LV end-systolic dimension was 31 mm.

In June 2018, she delivered via Cesarean section (C-section) at a general clinic following a full-term pregnancy. During the operation, she experienced tachycardia and hypertension, which subsided after the surgery. She did not report any signs of discomfort, so the doctor did not administer any treatment for her. The infant had no abnormalities as had an Apgar score that changed from 8 to 9. The patient was discharged five days following the surgery and took a leave of absence. One afternoon in October 2018 (four months after her delivery) while watching television at home with her family, she suddenly lost consciousness and her breathing and pulse stopped. Therefore, he was sent to the emergency room (ER) of the hospital for further treatment.

In ER, the patient's EKG showed that she had ventricular tachycardia. Following emergency treatment, her body temperature was 37.2°C, her pulse was 122 bpm, her respiratory rate was 25 breaths/min, and her blood pressure was 142/128 mmHg. A physical examination indicated that her Glasgow Coma Scale was E1M1VE due to her use of sedatives; both her pupils had a diameter of 2.0 mm and were responsive to light; and she was placed on combined endotracheal intubation and ventilation support. Rales were present in both of her lungs as well. Her heart rate was rapid but regular; the third heart sound was present but the fourth heart sound and abnormal murmurs were absent; the point of maximal impulse had extended 3 cm beyond the left sixth intercostal space medial to the midclavicular line; and she did not experience any heaves and thrills. She also did not have jugular vein distention, jugular vein bruits, or thyroid goiter. A visual inspection of her abdomen showed that she had a 15 cm C-section scar. The auscultation results showed that her bowel sounds were normal and vascular bruits were absent. Tympany was heard during percussion. The palpation results indicated neither hepatosplenomegaly nor liver masses. Edema and other abnormalities were absent in her appendicular skeleton.

With regard to emergency department laboratory tests, the patient's routine blood tests demonstrated an elevated white blood cell count of 14150/ $\mu$ L, in which neutrophils = 7%, lymphocytes = 82%, and atypical lymphocytes = 8%; her hemoglobin level and platelet count were 13.8 g/dL and 260 x 103/ $\mu$ L, respectively. An arterial blood gas (ABG) test showed that the patient's hydrogen ion concentration corresponded to a pH level of 7.376; arterial carbon dioxide partial pressure (PaCO<sub>2</sub>) = 42.5 mm Hg; arterial oxygen partial pressure (PaO<sub>2</sub>) = 86.9 mmHg; hydrogen carbonate ion (HCO<sub>3</sub>) level = 24.4 mmol/L; and base excess = -0.9 mmol/L. A biochemical analysis of the patient's blood showed that her potassium ion level = 3.2 mmol/L; calcium ion level = 7/7 mg/dL; an elevated phosphorus level of 4.87 mg/L; alanine transaminase level = 561 IU/L; creatinine phosphokinase (CPK) level = 1020 IU/L; creatine kinase-muscle/brain (CK-MB) level = 64 U/L; Troponin T (Tn-T) = 91.4 ng/L; and lactic acid level = 4.72 mmol/L. EKG results demonstrated atrial flutter with 2:1 conduction and left bundle branch block. Chest X-ray results indicated cardiomegaly and congestion in the left lobes. A CT scan of the patient's head revealed no obvious signs of intracranial hematoma or infarction. A CT scan of her chest indicated pneumonia in the left lung but no pulmonary embolism. A cardiac catheterization showed that the patient had an elevated left ventricular end-diastolic pressure (LVEDP)

of 34 mmHg, as well as an absence of coronary artery embolism and aortic regurgitation. However, ventricular fibrillation and torsade de pointes had occurred during the catheterization procedure.

Summarizing the aforementioned history taking, physical examination, laboratory tests, and imaging results, it is unlikely that the patient had a hemorrhagic stroke or a pulmonary embolism. Acute coronary syndrome was also excluded as a possibility since she did not experience chest tightness and pain during her pregnancy, hypoxic changes were absent in her EKG results, and coronary artery embolism was not detected through cardiac catheterization. Based on this clinical impression, the patient was likely to have concomitant PPCM and ventricular tachycardia. Other differential diagnoses included suspected myocarditis, concomitant pneumonia and sepsis, and metabolic acid-base disturbances. However, since the patient only developed her symptoms four months after childbirth (so there were no obvious reasons for heart failure) and she did not have any heart disease prior to her pregnancy [13], it is deduced that she had PPCM.

### Imaging and laboratory diagnosis

After being admitted, therapeutic hypothermia was given to the patient in order to prevent neurologic injuries from emergency treatment. She was given 40 mEq of 15% potassium chloride, 250 ml of sodium bicarbonate (Rolikan), and 20ml of 10% magnesium sulfate in order to prevent hypokalemia, hypomagnesemia, and metabolic acidosis. She was also administered the empirical antibiotic Tazocin (4450 mg, IV, Q6H) to control her pneumonia, as well as Ivabradine HCl (Coralan, 5 mg, PO, BID) and Cordarone (200 mg, PO, BID) to control her heart rate. In order to conduct further differential diagnoses, blood tests for toxins, cultures, thyroid function, adrenal function, and viral hepatitis were performed. The results showed that her thyroid-stimulating hormone level = 1.510 uIU /mL, free T4 level = 1.12 ng/ dL, adrenocorticotrophic hormone level = 465.90 pg/mL, cortisone level = 31.130, and also indicated normal thyroid and adrenal cortex functioning. CPK 642 IU/L, CK/MB 24 U/L, Tn-T 79.3ng/L, no findings were yielded from the blood and sputum culture and toxin tests. Blood routine tests showed that her white blood cell count =  $9.46 \times 10^3$ /uL, blood potassium level = 3.7 mmol/L; her ABG test results showed that her pH = 7.328; PaCO<sub>2</sub> = 46.7 mmHg, PaO<sub>2</sub> = 204.3 mmHg; HCO<sub>3</sub> level = 24.0 mmol/L; and base excess = -2.3 mmol/L. A follow-up chest X-ray exam on the second day showed no signs of pneumonia, and her temperature and vital signs had returned to normal ranges. Based on the aforementioned findings and the patient's status, concomitant pneumonia and sepsis, metabolic acid-base disturbances, and myocarditis were excluded. On the sixth day, results from a follow-up echocardiography showed that her LVEF = 20%, LVEDD = 63 mm, and LVESD = 57 mmHg. Based on the continuous decline in her heart function, as well as a Growth Stimulation Expressed Gene 2 (ST2) level of 33.8 ng/m, the patient was diagnosed with PPCM and left ventricular systolic dysfunction.

### Treatment

The goals of treatment for PPCM are to alleviate the patients' symptoms, reduce hospitalizations, decrease mortality, and improve their prognosis. The focus is to reduce the patients' pre/post-treatment cardiac load and improve coronary circulation. Hence, the drugs used in pharmacotherapy must be able to alleviate heart failure and reduced ejection fraction and shorten the course of disease. The symptoms of the patient in this study were classified as Class IV under the New York Heart Association Functional Classification system. Various drugs, such as a beta-blocker (Concor), a diuretic (Spironolactone), and an angiotensin II receptor antagonist (Valsartan) were administered to her. In addition, her in-hospital 24-hour EKG results indicated that she had continuous sinus rhythm with LBBB, a QRS complex >120ms, as well as ventricular tachycardia and ventricular fibrillation, which reduced her left ventricular systolic function and ejection fraction and elevated her risk of death and sudden death. Hence, she received cardiac resynchronization therapy (CRT-D), so as to synchronize the systolic function of her left and right ventricles, prevent sudden and unexpected ventricular arrhythmia, and reduce the risk of sudden death.

### Discussion

PPCM is a form of idiopathic cardiomyopathy and is one of the main causes of death among pregnant women. It usually occurs during the last month of pregnancy or five months postpartum. Therefore, during the diagnosis of PPCM, health care professionals have to take

note of the time of occurrence in order to verify if a patient's compensatory systolic/diastolic heart failure, valvular heart disease, or myocardial infarction were a result of PPCM. In addition, they must also take note that poor blood pressure control during pregnancy could be associated with preeclampsia (PE), gestational hypertension, and pulmonary edema, all of which endanger a fetus's life [6].

The patient did not display any symptoms of PPCM during her pregnancy and did not disclose that she had received any cardiovascular treatment, hence her heart functions were not closely monitored during her pregnancy. The symptoms of PPCM lack specificity, and about 70 - 80% of pregnant women exhibit symptoms similar to those of PPCM during the final stages of their pregnancy. Therefore, it is extremely difficult to distinguish between PPCM and normal physiological changes during pregnancy. PPCM must be taken into consideration when pregnant women display its classic symptoms such as weight loss, pedal edema, and dyspnea; or symptoms of heart failure such as right upper quadrant pain, dizziness, chest pain and palpitations, anemia, and orthostatic hypotension [7], as well as fatigue, cough, and other flu-like symptoms [6,8].

Pregnant women must undergo regular blood pressure and blood glucose or urine glucose tests during their pregnancy. Furthermore, laboratory biochemical markers with better sensitivity and specificity to PPCM must be checked as they are crucial for the early diagnosis of PPCM as well as for preventing sudden events. At present, the primary diagnostic indicators are the same for pregnant women with suspected PPCM and patients with heart failure, that is, serum BNP and NT-proBNP levels. PPCM patients usually experience a two-fold increase in their BNP level, which is helpful for the early detection of LVEF deterioration and diagnosis. However, the test could be influenced by anemia, renal disease, and bacterial sepsis, which increases the patient's BNP level [9]. Even though NT-proBNP is a non-specific biomarker of PPCM, it has a good sensitivity for heart failure and a longer half-life than BNP. Compared to their healthy counterparts, pregnant women with PPCM have elevated postpartum NT-proBNP levels [10]. In addition, the cardiac function of postpartum women is irreversible. Their baseline NT-proBNP, baseline total cholesterol, serum apoptosis antigen 1 (Fas/Apo1), serum cardiac troponin I (cTnT), and sFLT1 (soluble fms-like tyrosine kinase-1) levels also play an important role in the management of the disease as they contribute to the early prediction of LVEF improvements and survival [11].

When pregnant women display the symptoms or signs of heart failure during the late stage of their pregnancy or after giving birth, and the results of laboratory tests, baseline chest X-ray exams (cardiomegaly or pleural effusion), and EKG tests (QT interval prolongation, LV hypertrophy, or atrial fibrillation) show that they have a high risk of being diagnosed with PPCM, imaging tests should be performed for further verification. However, in light of the impacts of radiation on a fetus, echocardiography is still the primary imaging diagnostic tool with the most desirable cost-benefit balance and is suitable for differential diagnosis. If the echocardiography fail to differentiate between PPCM and chronic systolic heart failure, cardiovascular magnetic resonance imaging (MRI) could be performed for further verification as it is not harmful to the fetus and is safe during pregnancy [12].

Pharmacotherapy regimens for PPCM patients are the same as those for patients with heart failure. However, during the late stage of pregnancy, teratogenic angiotensin converting enzyme inhibitors (ACEI) and angiotensin receptor blockers (ARB) should not be used. Digoxin is safe for pregnant women, but when it is combined with diuretics, care must be taken to prevent hypokalemia, hypovolemia, and poor circulation within the placenta. Moreover, if a patient is diagnosed with PPCM during the late stage of pregnancy and her EKG indicates that she has concomitant atrial fibrillation and cardiac dysfunction, preventive anticoagulants should be given to her before childbirth in order to reduce the risk of thromboembolism [13].

After being admitted, the patient's follow-up echocardiography result showed that her LVEF was only 20%, which suggests that she has poor left ventricular systolic function. She also has a relatively higher risk of cardiomyopathy recurrence, which highlights the need for subsequent follow-ups on her cardiac function as well as regular medication use after being discharged. In general, a follow-up echocardiography should be performed every three to six months in order to evaluate a patient's recovery and cardiac function, as well as to

reduce the drug dosage or the need for lifelong medication [13]. If a patient plans to get pregnant again, the risks of doing so should be explained to her beforehand, and echocardiography should be performed regularly during her pregnancy [14]. However, holistic healthcare is not only achieved by physicians and nurses alone, as multidisciplinary teamwork is essential. As a nurse practitioner, the author was responsible for providing care to the patient during her hospitalization. After understanding her needs, the author arranged consultations with rehabilitation therapists, pharmacists, dieticians, case managers, and gynaecologists. Not only did the team take care of the patient and treated her PPCM symptoms, they also took into account her psychological feelings as a mother.

### Conclusion

PPCM is a form of non-ischemic dilated cardiomyopathy and its causes remain poorly understood. The risk factors of PPCM include one's diet, lifestyle, medication history, inheritance, preeclampsia, hypertension, obesity, low family income, and having twins [4]. Life-threatening ventricular arrhythmias could lead to sudden cardiac death among peripartum women, just as the patient reported here; although this is a rare presentation, it is catastrophic for the whole family. In our case, patient falsely attributed her cardiac discomfort to physiological changes during pregnancy, resulting in her neglecting the importance of undergoing further cardiac examinations. She also failed to disclose her history of cardiovascular disease during her prenatal examination, which increased her risk of PPCM and ultimately resulted in sudden cardiac arrest. From the perspective of preventive medicine, nurse practitioners must understand the means of preventing and reducing the risk of PPCM. Therefore, understanding a patient's blood pressure control before and during her pregnancy, as well as her history/family history of hypertension, routine medication use, body mass index, lifestyle (smoking and alcohol ingestion) are important steps in medical history taking. Moreover, performing differential diagnosis and noting the time in which a symptom had occurred during a woman's pregnancy are beneficial toward the early diagnosis of PPCM.

### Conflict of Interest

The authors declare no conflicts of interest.

### Academic Ethics

Ethical approval was obtained from the Institutional Review Board of the institution (No.: 109127-C).

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### Volume 3 Issue 3 March 2021

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