

Women with Persistent Left Ventricular Systolic Dysfunction in Peripartum Cardiomyopathy - A Case Report

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Abstract: *Background:* Peripartum Cardiomyopathy is left ventricular systolic dysfunction that occurs during late pregnancy and five months after postpartum without other causes of heart failure and disease beforehand¹⁰. The exact pathophysiological mechanism that leads to PPCM is unknown⁷. It is important to identify predictors for persistent LVSD. Early recognition can lead to decrease in morbidity and mortality. *Case Report:* A 24 years old woman came to emergency unit with complaints of shortness of breath, leg edema and cough without fever. The complaints have become increasingly in three days. The patient said there had been history of same symptoms in six months ago, and that came up after history of gave birth in five month. History of chest pain, dyspneu, hypetention and diabetes mellitus before pregnancy is denied. On examination, patient had a blood pressure of 130/70 mmHg with heart rate 78 bpm, respiratory rate was 24 times per minutes and body temperature 36.7°C. Electrocardiography showed sinus rhytm. Chest X-ray at admission had cardiomegaly. The latest echocardiography finding cardiomiopathy with EF 44.66% and Fractional Shortening 22.56%. From all of the examination result the conclusion was lead to acute heart failure et causa peripartum cardiomyopathy with chronic kidney disease. *Conclusion:* PPCM occurs in women at the end of pregnancy or a few months after giving birth, which previously had no other heart disease. Early diagnosis and serial examination for heart failure are expected to play an important role in reducing PPCM mortality and morbidity

Keywords: Peripartum Cardiomyopathy, Acute Heart Failure, Pregnancy

1. Introduction

Peripartum Cardiomyopathy is a rare, which affects women in the last month of pregnancy or in the first 5 months after give birth. In some women, clinical and echocardiography can improve until returning to normal conditions, but in some other women can develop into heart failure and sudden cardiac death. Early diagnosis and the therapies for heart failure have an important role in reducing PPCM mortality and morbidity¹⁰. It is hoped that there will be no delay in recognizing the disease and causing an increase in morbidity and mortality

2. Case Report

A 24 years old woman came to emergency unit with complaints of shortness of breath, leg edema and cough without fever. The complaints have become increasingly in three days. The symptoms getting worse when she do some activity and sometimes waking up during sleep due to tightness. The patient said there had been history of same symptoms in six months ago, that came up in five month after history of gave birth and the echocardiography show EF 36.17% and FS 17.68%. During pregnancy until last trimester there are no pre-eklamsia but sometimes there is swelling on leg. History of chest pain, dyspneu, hypertension and diabetes mellitus before pregnancy is denied. On examination, patient had a blood pressure of 130/70 mmHg with heart rate 78 bpm, respiratory rate was 24 times per minutes and body temperature 36.7°C. Peripheral oxygen saturation of 98% with 3 lpm nasal oxygenation. There are ronki in basal. Electrocardiography showed sinus rhytm. Chest X-ray at admission had cardiomegaly. Result of

laboratory test, Leukocytes 7500/uL, Hemoglobin 7.8 g/dl, Thrombocytes 273.000/uL, Hematocrit 24.3 %. Electrolyte serum result, Kalium 6.18 mmol/L, Natrium 135.8 mmol/L, Chlorida 109.1 mmol/L. Creatinin 16.1 mg/dl, Ureum 242 mg/dl. Nasofaring swab test are negative two times. The latest echocardiography finding cardiomiopathy with EF 44.66% and Fractional Shortening 22.56%. From all of the examination result the conclusion was lead to acute heart failure et causa peripartum cardiomyopathy with chronic kidney disease. Patient had acute heart failure therapy with 3 lpm O₂ nasal oxygenation, 0.9% NaCl IVFD 500cc/24 hours, and a urine catheter. Pharmacological therapy is given Furosemid 40 mg twice daily, Bisoprolol 5 mg daily, Valsartan 80 mg daily, Trimetazidine 35 mg twice a day, and hemodialysis from internist.

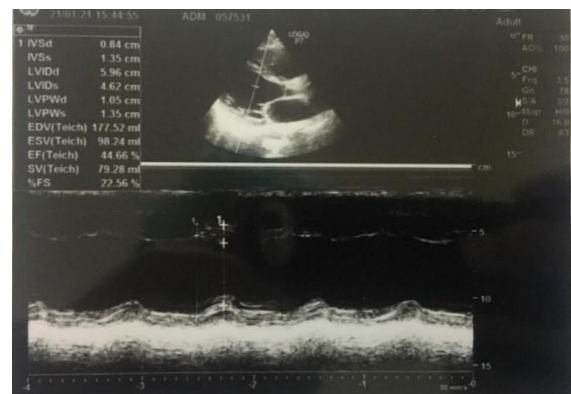


Figure 1: Parasternal long axis view. This picture shows decrease in left ventricular ejection fraction (EF 44.66%) and fractional shortening (FS 22.56%)

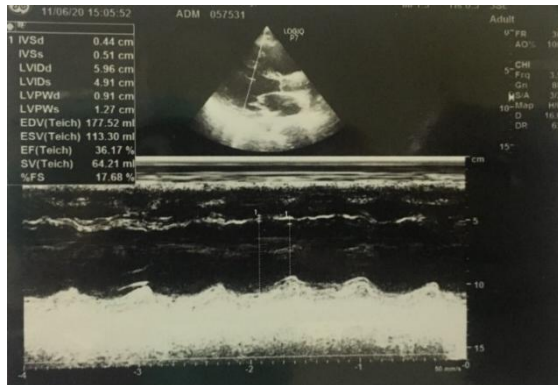


Figure 2: Parasternal long axis view. This picture six months ago shows decrease in left ventricular ejection fraction (EF 36.17%) and fractional shortening (FS 17.68%)

3. Discussion

Definition

The commonly used definition of PPCM from the National Heart and Blood Institute and the Office of Rare Diseases of the National Institute of Health are heart failure caused by left ventricular systolic dysfunction that occurs during late pregnancy and 5 months after postpartum without any other cause of heart failure and other previous illnesses^{1,5}. According Heart Failure Association of European Society of Cardiology is simpler by removing time constraints. Peripartum cardiomyopathy is an idiopathic cardiomyopathy which shows the presence of heart failure caused by left ventricular systolic dysfunction at the end of pregnancy or several months after delivery, where no other cause of heart failure is found. The left ventricle cannot be dilated but the ejection fraction almost always falls below 45%¹³.

Etiology and Pathophysiology

The exact pathophysiological mechanism that leads to PPCM is unknown, but increased oxidative stress and inflammation have been proposed. Recently, it was postulated that an oxidative stress-cathepsin D-Cleaved 16-kDa prolactin cascade is related to the pathophysiological mechanism of PPCM. During peri/postpartum period, enhanced oxidative stress that triggers the proteolytic cleavage of the prolactin into a potent anti-angiogenic, pro-apoptotic and proinflammatory 16-kDa prolactin fragment seems to play a central role in decreasing cardiomyocyte metabolism⁷.

Increased plasma concentrations of inflammatory cytokines including tumor necrosis factor α (TNF- α); C-reactive protein (CRP); and Fas/Apo-1, plasma markers of apoptosis, have been identified is higher in women with PPCM⁸. Sarojini et al. found that the baseline IL-6, CRP, and TNF-alpha were relevant to the mortality in PPCM patients¹¹.

Recently, monocyte-to-high density lipoprotein (HDL) cholesterol ratio (MHR) has emerged as a novel and widely available inflammation and oxidative stress-based marker. In several studies, MHR has been reported as a significant prognostic marker in various cardiovascular diseases^{2,4,9}. Higher MHR levels were significantly associated with persistent LV systolic dysfunction in PPCM. These results suggest that higher MHR levels may represent a pro-oxidant

and pro-inflammatory effect on the myocardium. As low-cost, simple, reproducible parameters of the CBC and lipid panel, the MHR can be widely used in clinical practice for prediction of LV recovery³.

Diagnosa

Post partum cardiomyopathy (PPCM) can be diagnosed through the criteria created by the National Heart Lung and Blood Institute and the Office of Rare Diseases (NHLBI). In this criteria, PPCM occur if (1) heart failure appear in the last month of pregnancy or at 5 months postpartum, (2) no exact cause of heart failure (3) no cardiovascular disease found before pregnancy (4) systolic dysfunction can be ascertained by echocardiography with the left ventricular ejection fraction criterion $< 45\%$ or there is a fractional shortening, with or without the left ventricular diastolic end dimension $> 2.7\text{cm}/\text{m}^2$ body surface area^{5,14}.

Treatment and Outcomes

Management of patients with PPCM is almost similar to management in other congestive heart disease. Non-medical treatment can be done such as patient education, limiting salt intake, preventing excess fluid intake, and vaccination against infectious⁶.

The prognosis of PPCM is positively associated with improving ventricular function within 6 months after delivery. Recovery of LV function was defined as the presence of LVEF $\geq 50\%$ ¹⁵. PPCM might lead to serious heart failure, malignant arrhythmias, thromboembolism, and death. The risk of major adverse events was more common in women with lower LVEF ($\leq 25\%$) in non Caucasian patients. Chapa et al. reported that FS $< 20\%$ and LVEDD ≥ 60 mm at the time of diagnosis were associated with a more than threefold greater risk for persistent LVSD¹⁵. Predictors of persistent left ventricular systolic dysfunction (LVSD) are inconsistently defined and include lower baseline LV ejection fraction (LV EF), late diagnosis, older age, black race and elevated plasma markers of inflammation¹⁵. Brain natriuretic peptide (BNP) is a useful clinical predictor of heart failure and has good diagnostic and prognostic value. The study from Weiping Li et al (2015), LV function was normalized within 1 year of onset in 56% of the patients. Decreased LVEF $< 34\%$ and BNP > 1860 pg/mL at baseline were associated with an approximate threefold increased risk of persistent LVSD¹⁴.

The patient in this case report was treated with diuretic and an angiotensin receptor blocker (ARB). A low-dose beta-blocker. At the six-month since diagnosed with PPCM, the patient had come back to emergency unit with recurrent acute heart failure, an echocardiogram that documented an improvement in LV systolic function from the original EF of 36.17% to 44.66%. Six months after diagnosed, EF of this patient cannot reach $\geq 50\%$. According the study from chapa et al, this patient have greater risk for persistent Left Ventricular Systolic Dysfunction because FS $< 20\%$ at the time of diagnosis. But need more data and long time follow up to determine persistent Left Ventricular Systolic Dysfunction at this case such as the baseline IL-6, CRP, BNP and TNF-alpha. or recently, (MHR) can be a novel marker inflammation and oxidative stress. In several studies, can be

widely used in clinical practice for prediction of LV recovery³.

4. Conclusion

PPCM occurs in women at the end of pregnancy or a few months after giving birth, which previously had no other heart disease. The patient can ignore the initial complaint until the complaint becomes more severe. As this patient before pregnancy there is no cardiac complaint but in five months after give birth EF was 36.17% and patient come already in state of acute heart failure. Women with persistent ventricular dysfunction are more difficult to survive and return to normal heart function compared to women with increased left ventricular function. It is important to identify predictors for persistent LVSD. Early diagnosis, serial examination, and the latest therapy for heart failure are expected to play an important role in reducing PPCM mortality and morbidity

5. Conflict of Interest

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7. Author Contribution

IPAP contributing in the making of the case report, and follow-up examinations of the patient. IGAS contributing in the pharmacotherapy management of the patient.

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