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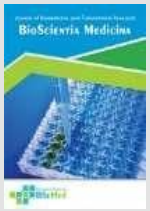

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Peripartum Cardiomyopathy: A Case Report

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A B S T R A C T

Background: Peripartum cardiomyopathy (PPCM) is a type of dilated cardiomyopathy of unknown origin. Predisposing factors for PPCM are multiparity, family history, ethnicity, smoking, diabetes, hypertension, preeclampsia, malnutrition, and advanced age in pregnant women or teenage pregnancy. **Case presentation:** A 29-year-old female patient was treated in the internal medicine department of Dr. M. Djamil General Hospital Padang on March 18th, 2023, with the main complaint of shortness of breath. On physical examination, signs of congestion were found. ECG shows sinus tachycardia, QT prolongation, and T inversion in V3-V6, II, III, and aVF. The patient is established with peripartum cardiomyopathy, community-acquired pneumonia, nonsevere low-risk MDR, hypochromic microcytic mild anemia et causa chronic disease, high-risk VTE, h hypokalemia et causa diuretic. **Conclusion:** Education regarding recurrent recurrence of PPCM in subsequent pregnancies is 30-50%. The prognosis depends on the recovery of left ventricular function; 30% of patients can return to baseline ventricular function within 6 months, and 50% of patients have an improvement in symptoms and ventricular function. Contraception is recommended that does not pose a risk of thromboembolism, such as a uterine device (IUD) and subcutaneous progesterone.

1. Introduction

Peripartum cardiomyopathy (PPCM) is a rare disorder but can cause heart failure in women in late pregnancy or the postpartum period. Peripartum cardiomyopathy (PPCM) is a type of dilated cardiomyopathy of unknown origin. This situation occurs in women in the last month of pregnancy and up to 5 months after giving birth who previously had no complaints.[1] Although PPCM occurs worldwide, most epidemiological data come from the United States, South Africa, Nigeria, and Haiti. In the US, the incidence is estimated to be between one in 900 and one in 4000 live births. A recent study using the US Nationwide Inpatient Sample found that the incidence increased from 1 in 1181 live births in 2004 to 1 in 849 live births in 2011.[5] Reasons for this increase include increasing rates of advanced maternal age, pre-eclampsia, and multiple pregnancies (driven in part by the use of reproductive technologies), which are risk factors for PPCM. Increased prevalence of cardiovascular risk factors such as hypertension, diabetes, and obesity among women of reproductive age.

The etiology of PPCM remains unknown, and many potential causes have been proposed but have not been proven. These include abnormal immune responses to pregnancy, abnormal responses to the increased hemodynamic burden of pregnancy, hormonal abnormalities, malnutrition, inflammation, and apoptosis. Recently, experimental studies have suggested a new and specific pathogenic mechanism by demonstrating the development of PPCM in female mice with deletion of the protein transcription factor signal transducer and activator of transcription 3

(STAT3). The absence of STAT3 cardiomyocytes in the postpartum heart results in increased oxidative stress secondary to impaired induction of the antioxidant enzyme manganese superoxide dismutase, which leads to increased expression and proteolytic activity of cardiac cathepsin D and results in the cleavage of the hormone prolactin to antiangiogenic and proapoptotic 16-kDa forms with detrimental effects on the microvasculature myocardium resulting in hypoxemia, myocardial apoptosis and development of PPCM. Preliminary data in humans showing the beneficial effects of bromocriptine, a prolactin inhibitor in a small number of PPCM patients, may support this mechanism of PPCM.[8]

The clinical presentation resembles dilated cardiomyopathy with systolic heart failure, including fatigue, dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, leg edema, swollen neck veins, pulmonary crackles, hepatic congestion, and third heart sound. Early symptoms are often confused with normal physiological phenomena of pregnancy. In most cases, symptoms develop within 4 months after delivery. Antepartum presentation occurs in less than 10% of cases. The severity of symptoms varies greatly from New York Heart Association functional class I to IV. The diagnosis of peripartum cardiomyopathy is considered in any peripartum patient with symptoms of unexplained heart failure. The greatest dilemma is the lack of specific clinical criteria that allow differentiation between PPCM and other types of heart failure. Therefore, all other possible causes of cardiac dilatation with heart failure must be completely excluded before establishing a clinical diagnosis of PPCM.

The prognosis of PPCM is positively associated with recovery of ventricular function. Failure to return to normal heart size is associated with increased mortality and morbidity. Women with persistent left ventricular dysfunction have a lower likelihood of survival and recovery than women with improved left ventricular function. Fractional shortening of less than 20% and a left ventricular diastolic dimension of 6 cm or greater at diagnosis were associated with a more than 3 times higher risk of persistent cardiac dysfunction

2. Case Presentation

A 29-year-old female patient was treated in the Internal Medicine department of Dr. M. Djamil General Hospital Padang on March 18th, 2023, at 20.00, with the main complaint of shortness of breath increasing since 2 days ago. Shortness of breath since 1 month ago, since the patient was pregnant with her first child at 34 weeks of gestation, and the shortness of breath has been increasing since the past 2 days. Shortness of breath gets worse when the patient carries out daily activities. Shortness is not influenced by weather, food and changes in position. Shortness of breath is not accompanied by a whistling sound. There is a history of waking up at night due to shortness of breath and the patient prefers to sleep with the pillow elevated. Swelling in both legs since 1 month ago. Weak and tired since 1 month ago. Looks pale since 2 weeks ago. Decreased appetite since 3 months ago. Cough for 1 week. Post-operative patients cesarean section first child 14 days ago at 39 weeks gestation due to severe shortness of breath. The child immediately cried. Because it was felt that the complaints were not decreasing and felt more burdensome, the patient and family came to the emergency room of a private hospital, and then the patient was referred from the private hospital for further examination and management. On physical examination, a general condition of moderate pain was found, with compos mentis awareness, an increase in respiratory rate of 30 times per minute, and a pulse of 107 times per minute. blood pressure 130/60 mmHg, and temperature 37.4°C. On physical examination, anemic conjunctiva, light reflexes, and isochoric pupils were found. On physical examination of the lungs, symmetrical movement of the chest wall was seen, and auscultation revealed bronchovesicular breathing sounds and soft, non-loud, wet crackles at the base of both lungs. Physical examination of the heart revealed an enlarged heart, regular heart rhythm with S3 gallop. Physical examination of the abdomen revealed

that the liver was palpable 4 fingers below the arcus costarum, 3 fingers below the xiphoid process, flat surface, springy consistency, sharp edges, no tenderness, no palpable spleen, no epigastric tenderness, hepatojugular reflux, and shifting dullness. In the extremities, edema was found in both legs. A complete blood laboratory examination found mild anemia (10.0 gr/dL). ECG examination with sinus tachycardia, prolong QT, inverted T in V3-V6, II, III, aVF. Echocardiography results showed EF 42% and NT-Pro BNP results 4.016. So the patient was diagnosed with peripartum cardiomyopathy Community, community-acquired pneumonia, non severe, low-risk MDR, Mild microcytic hypochromic anemia, and chronic disease high risk VTE, Hypokalemia, and causa diuretic. The patient was given drip therapy of furosemide 5 mg/hour, bisoprolol 1x2.5, ramipril 1x5 mg po, and spironolactone 1x25 mg po and experienced clinical improvement.

3. Discussion

The patient is diagnosed with peripartum cardiomyopathy because there are clinical symptoms of heart failure in the form of dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. This complaint has been felt by the patient since 2 months ago when the patient was pregnant with her first child at 34 weeks of gestation, which caused the patient to give birth vaginally. caesarean section at 39 weeks gestation. There is no previous history of hypertension, heart disease, or congenital heart defects. When the patient arrived, the physical examination found that he had cardiomegaly and S3 gallop, increased JVP, hepatomegaly, and bilateral leg edema, which were signs of overload fluid. According to Salam et al., the early symptoms of PPCM are often confused with the physiological phenomena of normal pregnancy. In most cases, symptoms develop within 4 months after delivery. Antepartum presentation occurs in less than 10% of cases. In late pregnancy, symptoms such as orthopnea and paroxysmal nocturnal dyspnea often leads to diagnosis peripartum cardiomyopathy which is late.[10,11] Electrocardiography showed sinus tachycardia, prolong QT, inverted T in V3-V6, II, III, aVF. The patient underwent echocardiography. Echocardiography in this patient showed decreased global LV systolic function with an EF of 42%. Echocardiography is central to the diagnosis of PPCM.

The chest x-ray shows cardiomegaly with pulmonary congestion accompanied by pneumonia, in accordance with Patel et al. that the chest x-ray picture in PPCM can be a sign of congestion such as pleural effusion. The principles for managing acute heart failure due to PPCM do not differ from those applied to acute heart failure arising from other causes. In summary, prompt treatment is essential, especially when patients have pulmonary edema and/or hypoxemia. Oxygen should be administered to achieve arterial oxygen saturation ≥95%, using, if necessary, non-invasive ventilation with a positive end-expiratory pressure of 5–7.5 cm H_2O. Intravenous diuretics (i.e.) should be administered when there is congestion and volume overload, with an initial bolus of furosemide 20-40 mg i.v. recommended.[12,13]

Education for PPCM patients is very important. Breastfeeding in patients with heart failure remains controversial. According to the 2018 ESC guidelines for the management of cardiovascular disease during pregnancy,[2] in patients with severe heart failure preventing lactation may be considered due to the high metabolic demands of lactation and breastfeeding (class IIb recommendation). These guidelines state that stopping lactation allows safe treatment with all existing heart failure medications.[14,15]

4. Conclusion

Education regarding recurrent recurrence of PPCM in subsequent pregnancies is 30-50%. The prognosis depends on the recovery of left ventricular function, 30% of patients can return to baseline ventricular function within 6 months, and 50% of patients have an improvement in symptoms and ventricular function. Contraception that does not pose a risk of thromboembolism is recommended, such as intrauterine device (IUD) and subcutaneous progesterone.

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