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Peripartum Cardiomyopathy: Challenges in Diagnostic and Management in Limited Resources Hospital in Kalabahi, Indonesia

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Abstract

Introduction: Peripartum cardiomyopathy is a rare condition of heart failure between the last month of pregnancy and the first five months post-delivery.
Case report: A 22 years old female G1P0A0 was experiencing dyspnea during her labor phase. Patient was assessed with PPCM and pulmonary edema based on her signs and symptoms of heart failure with no history of cardiac abnormalities, rales on both lungs, vascular congestion bilaterally, and left ventricle ejection fraction (LVEF) of 43%. Patient was managed with heart failure management including inotropes, furosemide, ACE inhibition, and on a mechanical ventilator. Patient was extubated, and inotropic and furosemide free on the sixth day of care, but redevelop pulmonary edema. Patient then reintubated, and inotrope and furosemide were recontinued. Patient was stable and extubated on the ninth day of care and discharged on the fifteenth day of care.
Conclusion: The diagnosis and management of PPCM can be challenging especially in limited resources hospitals. Evaluation of the clinical characteristics of the patient are the keys of this management.
Keyword: peripartum cardiomyopathy, PPCM, pulmonary edema, limited resources hospital

Introduction

These Peripartum cardiomyopathy is also known as postpartum cardiomyopathy (PPCM), is a new onset of heart failure that present in late pregnancy or early post-partum period with no other determinable causes [1,9,10]. The cases of PPCM have been reported in various countries and areas. The incidence was higher in the black race and developed countries [1,2]. The definite etiology and mechanism of disease are unknown. Some hypotheses such as viral myocarditis, nutritional deficiencies, autoimmune response, hemodynamic stresses due to pregnancy (increased fluid load), vascular dysfunction, and underlying genetic have been described [4,9,10].

The diagnostic criteria for PPCM include cardiac failure in a previously healthy woman in the last month of pregnancy or within the first five months post-delivery; the absence of determinable etiology for the cardiac failure; the absence of cardiac disease prior to the last month of pregnancy; evaluation using echocardiography shows left ventricular dysfunction

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includes ejection fraction less than 45% [1,9]. The symptoms of PPCM are suggestive of congestive heart failure such as dyspnea on effort, orthopnea, paroxysmal nocturnal dyspnea, palpitation, and lower extremities edema [1,9]. ECG usually shows non-specific changes [1,9]. Plain chest x-ray usually shows cardiac enlargement, pulmonary venous congestion [1,9]. Treatment of PPCM is similar to the management of heart failure, including angiotensin converting enzyme (ACE) inhibitions, angiotensin receptor blockers (ARBs), beta-blockers, spironolactone, digoxin, diuretics, vasodilators, and inotropes if needed[4]. The symptoms are usually found in normal pregnancy, which makes PPCM is challenging and often too late to be diagnosed. This leads to mortality as high as 20% to 50% [1,4].

Case Report

A 22 years old female G1P0A0, was experiencing dyspnea and desaturation during the delivery process. There were no symptoms of coughing, dyspnea, edema of lower extremities, and proteinuria, no history of fatigue, shortness of breath, palpitation, or inability to lay flat before or during pregnancy, hypertension, allergy, and diabetes. Patient denied ever having any heart problem and never been diagnosed with any heart abnormalities. Patient had total of 5 times antenatal care (ANC) in the Primary Health Care Facility with no significant problems.

During the labor phase, she suddenly experiencing severe dyspnea, and desaturation. Patient was given 15 l/min oxygen using non-rebreathing mask. Vital signs were follows: BP of 140/90 mmHg, HR of 123 beats/minute, RR of 35 rates/minute, and SpO_2 of maximum at 70%. Upon physical examination, she had a slight distended of jugular vein. Breathing was symmetric, vesicular breathing and bilateral rales on both lungs was detected. Cardiac auscultation showed normal first and second heart sound, no cardiac murmur was detected. Patient delivered a living male infant via vacuum-assisted vaginal delivery. Patient was then intubated and connected to mechanical ventilator with initial setting as follows, mode Pressure controlled ventilation (PCV), pressure control (PC) of 12, PEEP of 12 cmH2O, FiO_2 of 50%, frequency of 18 rates/minute resulting TV of 360 mL, respiratory rate of 18 rates/min and minute volume (MV) of 9.6 L.

Evaluated laboratory results as follows: hemoglobin= 7.3 g/dL, hematocrit= 24.7%, white blood cell= 23.8 x 10^3/μL, platelet= 218 10^3/μL, albumin= 2.5 mg/dL, another laboratory test within normal. Arterial blood gas cannot be analyzed because there was no testing tool available. Chest X-ray showed an increase of vascular congestion bilaterally resembles bat-wing appearance (Figure 1A). Electrocardiogram (ECG) showed sinus tachycardia of 119 beats/minute. Echocardiography showed EF of 43% (on dopamine 3 mcg/kg/minute) and severe septal hypokinetic. There were no abnormalities of valves and heart structure.

IV pump of furosemide 10 mg/hr dan isosorbide dinitrate were administered. Patient had angiotensin converting enzyme inhibition (ACEi) captopril start from 6.25 mg every 4 hours up to 50 mg/TID. Patient also had supportive treatment such as IV Ceftriaxone 2 gram/24hr, albumin correction, PRC transfusion, IV Paracetamol 1 gram/8hr, applying warmth and breast pump to avoid mastitis, nutrition via nasogastric tube and fluid balanced has to be deficit around 500-1000 mL/ 24hr.

On the fifth day, patient was stable and alert. The ventilator mode was weaned to spontaneous mode. On the sixth day, patient was extubated and given 3 l/min oxygen using nasal canula, dopamine and furosemide was tapered off and stopped. Dyspnea was reoccurred, hours after extubation. Vital sign as follows: BP of 130/82 mmHg, HR of 120 beats/min, RR of 25 rates/min, and SpO_2 of 87% using 5 l/min of simple mask. On physical examination, breathing was symmetric, basal bilateral rales, inspiratory and expiratory wheezing was detected. Chest x-ray was conducted and showed an increase of vascular congestion bilaterally (Figure 1B). Patient was breathing inadequately so reintubation was performed, IV furosemide 5 mg/hr and IV dopamine 5 mcg/kg/min were recontinued.

On the eighth day, patient was stable, thus the ventilator mode was weaned to spontaneous mode. On the ninth day, patient was extubated and given 3 l/min of oxygen using nasal canula. Patient was stable and the IV Furosemide and IV Dopamine were tapered off and stopped. On the fifteenth day patient was discharged. Three days after discharged, patient was followed up as an outpatient. Patient had no complaints, and echo showed LVEF of 49.2% and there was still hypokinetic on anteroseptal.

Discussion

In this case report, we diagnosed our patient with PPCM on the basis there are clinical presentations of acute heart failure such as sudden dyspnea, desaturation, slight jugular venous distension and diffuse rales upon chest auscultation in a woman with no history of sign of symptoms of cardiac abnormalities. Echocardiogram (ECG) showed no abnormalities of the heart. Chest X-ray showed vascular congestion bilaterally suggested pulmonary edema. Echocardiography showed left ventricle ejection fraction (LVEF) of 43% and septal hypokinetic.[1,2]

During pregnancy there are some physiological changes in the cardiovascular system such as increased blood volume, increased cardiac output, anemia, increased metabolic demand and changes in vascular resistance [4,5,7]. During labor and immediately after delivery, cardiac output increased about 60-80% above levels before the onset of labor [5]. This is related to some factors such as increasing heart rate and preload associated with the pain of uterine

contraction, increases in circulating catecholamines, and the autotransfusion of 300-500 mL blood from the uterus into the systemic circulation [5]. This will explain the mechanism how pulmonary edema develop during labor phase in our patient. Low left ventricle ejection fraction (LVEF 43%) and septal hypokinetic in this patient can cause left ventricle fail to eject the blood (systolic dysfunction), which can cause low cardiac output and excessive end diastolic volume. Due to volume overload in the left ventricle, the pressure in pulmonary vein also increased and cause fluid extravasation to the pulmonary interstitial space. Based on that reason, patient was given inotropic to enhance myocardial contractility to increase cardiac output and maintain mean arterial perfusion (MAP) for organ perfusion [6], furosemide (loop diuretic) to reduce preload and nitrate to dilatate arterial and reduce pump workload [6]. Fluid balanced are kept deficit or negative to reduce preload. Volume overload causes cardiac remodeling, that's why patient was given angiotensin converting enzyme (ACE) inhibition to alter the process of cardiac remodeling in order to reduce morbidity and mortality [8].

Fluid in the pulmonary interstitial space can widens the alveolar epithelium and capillary epithelium, thus gas exchange becomes interrupted [6]. Patient was intubated and given high PEEP to open and kept the alveoli distended to increase the surface area for gas exchange [6].

Patient was response to ventilator, inotropic and furosemide. We don't have blood gas analysis to evaluate exact oxygenation in the blood in order to wean the ventilator. We evaluate the patient based on adequate perfusion signs such as stable blood pressure, normal heart rate, Spo2 > 95%, warmth extremities, and patient were not agitated while on mechanical ventilator.[6]

Patient was extubated and inotropic was tapered off and stopped. Hours after extubating, dopamine and furosemide were stop, patient redeveloped dyspnea, increased of blood pressure and heart rate. On physical exam there were rales on both lungs. Our assessment was pulmonary edema. This may happen because the pump of the left ventricle may still inadequate enough to eject excessive blood. Reintubation was performed and connected to mechanical ventilator to prevent further hypoxemia. Dopamine and furosemide were recontinued to increase cardiac contractility and decrease preload [6].

Patient was discharged with no complaints. Patient went back to the hospital as an outpatient clinic three days later, patient had no complaints and echocardiography evaluation showed LVEF of 49,2%. Studies showed recovered of EF to > 50% can be around at 6 months to 1 year [1]. Further follow up is planned to see the response of the treatment and recovery of ejection fraction (EF).[6]

Conclusion

Diagnosis and management of Peripartum Cardiomyopathy (PPCM) can be challenging because of the symptoms are similar to the normal pregnancy and limited resources of diagnostic tools, especially in peripheral hospital. Holistic management and using clinical characteristics of the patient to evaluate and to treat the patient are the keys of this management.

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