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Management of Peripartum Cardiomyopathy in Severe Preeclampsia Patients in ICU

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ABSTRACT

Introduction: Peripartum cardiomyopathy (PPCM) is a life-threatening cardiomyopathy with left ventricular systolic dysfunction, in late pregnancy, during delivery, or in the first months postpartum, in women who have no previously identifiable cause of heart failure.

Case: A 25-year-old primigravida woman with a pregnancy of 35 weeks came to the Emergency Department (ER) with symptom of severe pre eclampsia, and pulmonary edema and later underwent a cesarean section. After surgery, the patient is admitted to the ICU , and then became worsening. Echocardiography was then performed and found a decrease in left ventricular systolic function, EF 35%, posterior anterior acinetics, mild MR. The diagnosis of PPCM was established. The therapy given is drugs and a negative fluid balance is created for this patient. On day 10, the patient showed improvement in left ventricular function with EF 48%. The patient is extubated from a mechanical ventilator and moved to the internal medicine inpatient room.

Conclusion: I Early diagnosis with supporting echocardiography as well as appropriate management in PPCM cases greatly determines the success of therapy in patients in the ICU.

Keywords

Peripartum cardiomyopathy, Severe Preeclampsia, PPCM

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INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a life-threatening cardiomyopathy characterized by the development of acute or slow left ventricular systolic dysfunction, with an ejection fraction of <45% in late pregnancy, during delivery, or in the first months postpartum, in women who have no previously identifiable cause of heart failure.[1-3] Delay in diagnosis may occur because symptoms and signs of PPCM may resemble normal findings in late pregnancy and peripartum periods. Some women can experience relatively mild conditions and recover completely, while others experience significant morbidity and mortality. The majority of women with PPCM develop severe symptoms (NYHA class III or IV).[1]

CASE REPORT

A 25-year-old primigravida woman with a pregnancy of 35 weeks came to the Emergency Department (ER) complaining of shortness of breath for 1 day and progressive orthopnea for 2 days. Complaints of hypertension are felt since 6 months of gestation, patients also complain of blurred vision, heartburn and frequent dizziness. Never had a seizure. Before pregnancy, the patient had no history of hypertension, heart disease or lung infection. In auscultation, bilateral ronki with gallop heart sounds are heard. On a random midstream urine dipstick test, the urine protein is 3+ (indicating proteinuria). His medical records showed no previous history of heart disease, and his antenatal visits were mediocre. Complaints and examinations experienced by patients

indicate a problem of the cardiovascular system with severe preeclampsia. The patient was diagnosed with severe preeclampsia and pulmonary edema and later underwent a cesarean section.

After surgery, the patient is admitted to the ICU using a PSIMV mode mechanical ventilator with PEEP 8. On day 5 of treatment, HR increased to 120-130x/min, SaO2 98-99%, with additional pansystolic and gallop noise. Echocardiography was then performed and found a decrease in left ventricular systolic function, EF 35%, posterior anterior acinetics, mild MR. The diagnosis of PPCM was established. The therapy given is furosemide, dobutamine, digoxin, captopril. A negative fluid balance is created for this patient (- 7800 ml) during the 10-day treatment. On day 10, the patient improved and echocardiography evaluation showed improvement in left ventricular function with EF 48%. The patient is extubated from a mechanical ventilator and moved to the internal medicine inpatient room.

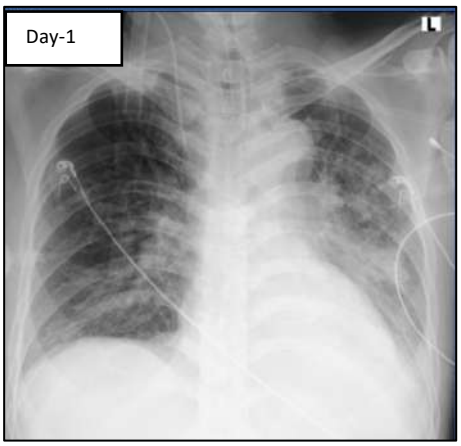

Fig. 1 Imaging of the Thorax

DISCUSSION

Symptoms and clinical signs of peripartum cardiomyopathy (PPCM) in the form of dyspneu, othopneu, paroxysmal nocturnal dyspneu, cough, chest pain, anorexia, fatigue and lower extremity udema. On physical examination found jugular venous dystention, tachycardia, tachypneu, hepatomegaly, ascites, peripheral edema, changes in mental status and thromboembolism. On examination of thoracic photos, mild to moderate heart enlargement is obtained with the left ventricular boundary rounded.[3] Echocardiography examination is an important diagnostic tool for the diagnosis of cardiomyopathy. LV dimensions, geometry, and systolic and diastolic functions can be assessed by echocardiography. PPCM is diagnosed using LV systolic dysfunction. The echocardiography criteria for PPCM are strict: the final dimensions of the diastolic LV > 2.7 cm/m2 and fractional shortening of M-mode <30% or LV EF <45%. In the latest guideline note, LV dilation is not mandatory for the diagnosis of PPCM. In addition, echocardiography is useful to rule out differential diagnosis.[3]

This patients are treated with an initial diagnosis post SC with severe preeclampsia and pulmonary edema. The diagnosis of PPCM is established based on a history of no previous heart disease or a history of family members who have similar diseases.1 Echocardiography examination showed decreased left ventricular function with EF 35%, anterior posterior akinetic heart wall with mild mitral valve regurgitation, and pericardium effusion was found.

To reduce excess fluid in pulmonary edema, mechanical ventilation with high PEEP and diuretics is given.[3] Pharmacological management using a combination of angiotensin converting inhibitory enzymes, namely captopril and digoxin through NGT, and dobutamine adjusted to hemodynamic changes. The use of ACE inhibitor class oral drugs because the ACE inhibitor class is one of the optimal oral drugs as pharmacological therapy in patients with peripartum cardiomyopathy in reducing after load and for pregnant women it is better to use angiotensin receptor blockers (ARBs).[3] Intravenous administration of dobutamine

in these patients due to the presence of unstable hemodynamics. Dobutamine is an adrenergic β1 agonist and has a weak α1 zero. This property causes dobutamine not to greatly decrease peripheral resistance so that it does not cause tachycardia reflexes. Dobutamine increases cardiac output because it increases cardiac contractility, lowers pulmonary artery pressure.[4] Dobutamine is discontinued after normal blood pressure. The main purpose of administering the above drugs is to reduce preload, reduce afterload and increase the strength of heart contractions.[4]

Nonpharmacological management of PPCM in these patients by limiting excess fluid, restriction of low-salt enteral diit. In peripartum cardiomyopathy (PPCM) poor prognosis is influenced by several factors, including: age at the time of old pregnancy, multiple pregnancies, multipara, late onset (>2 weeks after delivery), and complications of thromboembolism. However, 30-50% of patients can recover without complications, with left ventricular systolic function can return to normal. The risk of recurrence of PPCM is high, especially if left ventricular systolic function has not fully recovered. However, for women who have normal left ventricular systolic function as shown in the stress dobutamine examination by echocardiography, the risk of severe cardiomyopathy including death is relatively low in subsequent pregnancies.[4] This patients receive air way, breathing, circulating and PPCM management in accordance with the procedure. After treatment in the ICU the patient was in good condition and was transferred for ward care.

CONCLUSION

Early diagnosis with anamnesis, physical examination and supporting echocardiography as well as appropriate management, such as the use of mechanical ventilation devices for breathing support, pharmacological and non-pharmacological medicamentose in PPCM cases greatly determines the success of therapy in patients in the ICU.

DECLARATIONS

The research has received approval from Faculty of Medicine, Padjadjaran University / Hasan Sadikin Hospital Research and Ethics Committee. Participants were informed about this report.

CONSENT FOR PUBLICATION

The Authors agree to publication in Journal of Society Medicine.

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COMPETING INTERESTS

The authors declare that there is no conflict of interest in this report.

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