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Management of Respiratory Failure in Peripartum Cardiomyopathy Patient: Case Report

Dewi Yuliana Fithri[1], Budiana Rismawan[2]

[1,2] Anesthesiology and Intensive Care Department, Medical Faculty of Padjadjaran University/ Hasan Sadikin General Hospital, Indonesia

Correspondence: Dewi Yuliana Fithri, Anesthesiology and Intensive Care Department, Medical Faculty of Padjadjaran University/ Hasan Sadikin General Hospital, Indonesia. Email: dewiyulianafithri@gmail.com Phone: +62811680232

Abstract
Introduction: Respiratory failure in peripartum cardiomyopathy is a lethal risk. The onset of respiratory failure in peripartum cardiomyopathy is due to left-to-right heart failure, which lowers PaO2. Unfortunately, symptoms of peripartum cardiomyopathy are often non-specific and progressive, so diagnosis and treatment are frequently delayed. Case: A woman, 41 years old, 35-36 weeks pregnant, complains of shortness of breath for four days, worsening one day before hospitalization. Cough (-), The patient has no history of heart disease; this is her 5th pregnancy, and she has never complained of the same thing in previous pregnancies. physical examination of consciousness, compost mentis,BP 90/60 mmHg, HR 122 x/i, RR 32-36 x/I, T 36.5 °C,, Spo2 is 85% without O2. On examination, blood gas analysis showed a respiratory alkalosis with type 1 respiratory failure. The patient underwent an emergency cesarean section on the indication of type 1 respiratory failure—postoperative care in the ICU with the installation of a ventilator. During the five days of treatment, the patient's condition improved when transferred to the ward. Discussion: Respiratory failure in peripartum cardiomyopathy results from a low PaO2, usually accompanied by an accumulation of fluid in the interstitial lung tissue (pulmonary edema), which exacerbates hypoxaemic conditions due to decreased cardiac output. Airway protection is significant, such as performing mechanical ventilation with sedation to optimize oxygen delivery; giving inotropic (digoxin) and vasoactive drugs (dopamine or dobutamine) to increase contractility and maintain mean arterial pressure (MAP) for organ perfusion; reducing preload and afterload; and maintaining negative fluid balance while paying attention to adequate organ perfusion. Conclusion: Airway patency is the primary key in the management of respiratory failure in patients with peripartum cardiomyopathy

Keywords: Respiratory Failure in Pregnancy, Peripartum Cardiomyopathy, Intensive Care Unit

1. Introduction

Peripartum cardiomyopathy, also known as peripartum cardiomyopathy (PPCM), is a rare idiopathic cardiomyopathy that develops without a known cause in the last month of pregnancy or the first six months after delivery. PPCM can only be diagnosed by exclusion, as there is no agreement on diagnostic standards.t(Ng et al., 2022). Complications of cardiovascular disease accompany approximately 0.2 - 4% of pregnancies in developed

countries. (Regitz-Zagrosek et al., 2018). PPCM is a lethal condition with a reported average incidence of between 1/3000 and 1/4000, accounting for about 4% of maternal deaths (Ng et al., 2022; Regitz-Zagrosek et al., 2018). On echocardiographic examination, there will be a decrease in the left ventricular systolic function below 45%, accompanied by symptoms of cardiac decompensation that arise during labor without any evidence of a previous history of heart disease.(Kawamoto et al., 2021; Minami et al., 2018).

Clinical manifestations of PPCM consist of dyspnea, cough, and hypertension. They can be followed by severe heart failure within hours to days. Therefore, early diagnosis concerning the airway is essential for PPCM patients with respiratory failure due to effective treatment with ventilation reducing morbidity and mortality. For this reason, this case report is peripartum cardiomyopathy with respiratory failure (Dahiya et al., 2020; Johnson-Coyle et al., 2012; Minami et al., 2018).

2. Case report

A multigravida woman with a gestational age of 35-36 weeks complained of shortness of breath for four days and worsened one day of SMRS. Shortness of breath initially occurs when the patient walks to the bathroom, but the shortness of breath goes away with rest. The patient admits that she began to feel shortness of breath when lying down at six months of gestation, which reduced if the patient used 3-4 pillows. However, since one day of SMRS, the tightness has worsened and doesn't go away with rest. The patient claimed to have no complaints such as cough, fever, or complaints like this in previous pregnancies. This is the 5th pregnancy for the patient, history of abortion (-). The patient had no previous history of heart disease or lung disease. On examination, the consciousness was compos mentis, BP 90/60 mmHg, HR 122 x/i, RR 32 – 36 x/i, T : 36.5°C, SpO2 : 85% without O2. Physical examination showed TVJ increased 5 ± 4 cmH20. On auscultation of the lungs, wet basal crackles were seen accompanied by gallop sounds (+). The patient also underwent a chest X-ray examination for cardiomegaly with pulmonary edema. On echocardiographic examination, an EF of 35% and left ventricular dilatation were seen (Figure 2). Laboratory examination found no abnormalities, and blood gas analysis showed respiratory alkalosis with type 1 respiratory failure (pH: 7.51 mmHg, PO2: 44 mmHg, PCO2: 25 mmHg, HCO3: 22, BE: -1.7, SaO2: 91.8 %). The patient was given 100 mg of furosemide followed by 60 mg with monitoring of urine output (UOP), drip dobutamine five mcg/kilogram body weight/minutes, and injection of enoxaparin 1 x 0.6 cc. An emergency cesarean section (SC) was performed under general anesthesia. The operation lasted 15 minutes with a urine output of 1000 ml and bleeding of 400 ml. Furthermore, the patient was treated in the ICU with the installation of a ventilator with RR 20, PEEP 8, I; E = 1:2, FiO2 50%, SpO2: 97-98%. On day four, the patient's condition improved with CM awareness, BP: 120/80 mmHg, HR: 88 x/minutes RR: 24 x/minutes, temperature: 36.7 C, SpO2: 98% with O2 liters per minute nasal cannula. On day 5, the patient was transferred to the ward.

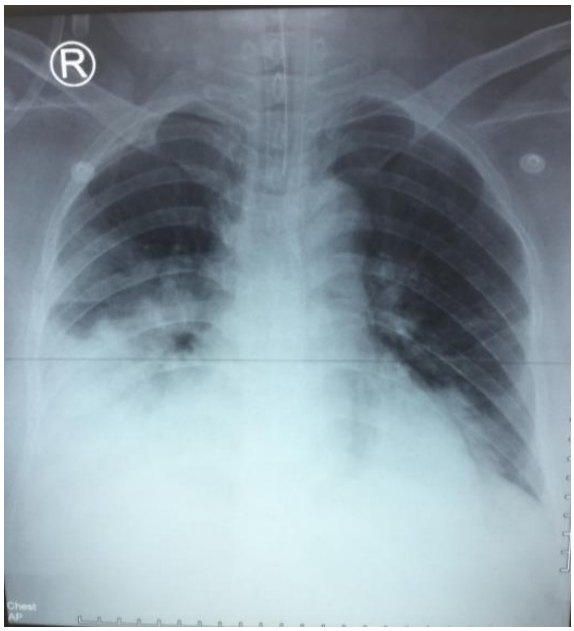

Figure 1: Photo of the PPCM Chest

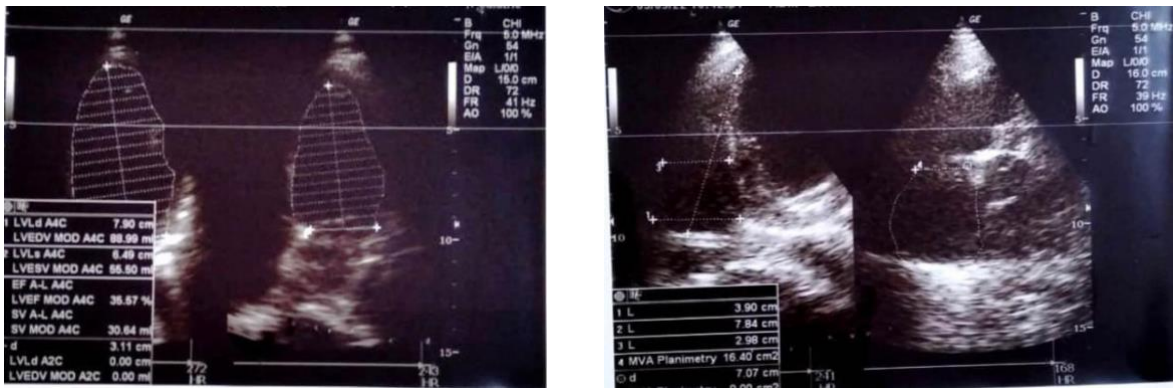

Figure 2: PPCM echocardiography

3. Discussion

During a typical pregnancy, 60% to 70% of pregnant women report having difficulty breathing. Physicians should be aware of PPCM because it can easily be misdiagnosed due to vague symptoms. The true prevalence of PPCM is unknown. The incidence ranges from 1 in 1300 to 1 in 15000 pregnancies Morbidity and mortality ranges from 25% to 90% due to geographic and diagnostic variation (Çimen et al., 2013). Usually, the PPCM condition is often accompanied by pulmonary edema on chest X-ray examination, which is also found in this patient. PPCM deaths generally result from heart failure, pulmonary embolism, and respiratory failure. Therefore, early management is crucial to prevent mortality in patients with PPCM.

Pulmonary edema occurs in PPCM patients due to heart failure and causes respiratory failure. The process of pulmonary edema in PPCM patients due to heart failure in the systolic and diastolic phases. Systolic heart failure is characterized by weakened left ventricular contractions that occur, while diastolic heart failure is characterized by the inability to dilate at the time of filling of the left ventricle resulting in decreased cardiac output. Compensatory mechanisms for decreased cardiac output are characterized by vasoconstriction and fluid retention. Vasoconstriction arises as a result of the resulting hypoxia, which stimulates the release of noradrenaline. In addition, decreased blood supply to the kidneys stimulates the renin-angiotensin system, which causes systemic vasoconstriction. In the short term, all of these mechanisms increase cardiac output. Compensatory vasoconstriction can lead to increased cardiac output but can weaken cardiac function. Furthermore, the increased pressure and volume in the left ventricle will eventually increase the pressure and importance in the pulmonary

circulation, which causes the movement of intravascular fluid into the alveolar cells and interstitial space. g(Andre et al., 2022; Shebl et al, 2022)

The mechanism of hypoxia arises in PPCM patients due to decreased oxygen delivery (DO2). Oxygen delivery levels depend on cardiac output (CO) and oxygen content of the arterial blood (CaO2). In patients with PPCM cardiac output has decreased so that compensation occurs with vasoconstriction and fluid retention, which increases afterload. The increased afterload on the left ventricle causes pulmonary congestion, which reduces the diffusing capacity of the lung, causing an increase in intrapulmonary shunt resulting in arterial hypoxemia. Another mechanism is pump failure, which causes decreased cardiac output and reduced tissue perfusion leading to circulatory hypoxia (Ramachandran et al., 2022).

Management of peripartum cardiomyopathy is mainly supportive. Where medical therapy for peripartum cardiomyopathy is the same as for heart failure, the management must involve a medical team consisting of cardiologists, obstetricians, and anesthesiologists to achieve optimal results during pregnancy and childbirth. In administering therapy, besides attaining optimal results for the mother, the effects of the drug on the fetus and baby who are breastfeeding must also be considered. The goal of therapy in patients with peripartum cardiomyopathy is hemodynamic optimization, optimizing preload, reducing afterload, and increasing contractility. Standard treatment in peripartum cardiomyopathy is: bed rest, and oxygenation, to maintain tissue oxygenation to help prevent end-organ dysfunction and multiple-organ failure (Lata et al., 2009; Shaikh, 2010).

Treatment of PPCM is based on improving the patient's hemodynamic status by reducing preload and afterload and increasing the heart's contractile strength, as in the treatment of heart failure. Management of acute decompensated heart failure still pays attention to the airway, breathing, and circulation. The airway must be secured as early as possible because the accumulation of interstitial fluid can result in airway edema and exacerbate hypoxemia due to decreased cardiac output. Patients with pulmonary edema often require assisted ventilation. In addition, the target for arterial oxygen saturation ≥ 95% should be achieved. Interventions can be performed, such as performing mechanical ventilation with sedation to optimize oxygen delivery, giving inotropic and vasoactive drugs to increase contractility and maintain mean arterial pressure (MAP) for organ perfusion, reducing preload and afterload and maintaining negative fluid balance while paying attention to the adequacy of organ perfusion (Dash et al., 2022; *Fragneto R*, 2006; Shaikh, 2010).

Safe inotropic drugs for pregnant and lactating women, namely digoxin, increase the ejection fraction in combination with a vasodilator. Usually, administration for 6-12 months reduces the risk of PPCM recurrence. In addition to inotropes, the administration of vasoactive drugs such as dopamine or dobutamine can be considered. For this reason, it is essential to know PPCM early and immediately administer therapy to reduce the morbidity and mortality of PPCM sufferers (Dash et al., 2022).

4. Conclusion

PPCM is a condition rarely found in pregnant women and is usually characterized by shortness of breath that appears at the last gestational age up to 6 months after giving birth. PPCM conditions often need to be explained. Therefore it is essential to consider PPCM in pregnant women who complain of severe shortness of breath in late pregnancy or up to 6 months postpartum. PPCM mortality is very high if it is late in diagnosis and therapy. Therefore airway patency is the primary key to managing respiratory failure in patients with cardiomyopathy.

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