Journal of Society Medicine
Research & Review Articles on Diseases
Journal of Society Medicine. 2025; 4 (7)

Management of a P3A0 Postpartum Patient with Peripartum Cardiomyopathy (PPCM), Acute Decompensated Heart Failure (ADHF), Respiratory Failure Due to Acute Pulmonary Edema, and Community-Acquired Pneumonia (CAP) in the ICU

Paulus Sulistiono [1*], Dhany Budipratama [2]

[1] *Intensive Care Trainee, Faculty of Medicine Padjadjaran University / Hasan Sadikin General Hospital Bandung, Indonesia*
[2] *Intensive Care Consultant, Faculty of Medicine Padjadjaran University / Hasan Sadikin General Hospital Bandung, Indonesia*

***Corresponding Author**: Paulus Sulistiono, Email: paulusmukri@gmail.com

ARTICLE INFO

Article history:
Received
5 May 2025

Revised
10 June 2025

Accepted
31 July 2025

Manuscript ID:
JSOCMED-050525-47-3

Checked for Plagiarism: Yes

Language Editor: Rebecca

Editor-Chief:
Prof. AznanLelo, PhD

ABSTRACT

Introduction: Acute dyspnea following pregnancy is a rare condition often accompanied by significant comorbidities. Potential causes include pulmonary embolism, amniotic fluid embolism, pneumonia, aspiration, pulmonary edema, and other critical conditions. Pulmonary edema, in particular, may occur during pregnancy or the postpartum period, associated with preeclampsia, peripartum cardiomyopathy (PPCM), pre-existing cardiac disease, tocolytic therapy, or fluid overload. This case report highlights a complex clinical scenario involving these factors.

Case Description: We present the case of a 36-year-old woman, P3A0, who developed progressive acute dyspnea six days postpartum following a vaginal delivery. Her condition rapidly progressed to respiratory failure, necessitating admission to the intensive care unit (ICU) and mechanical ventilation. Physical examination and diagnostic workup revealed acute pulmonary edema secondary to peripartum cardiomyopathy, complicated by acute decompensated heart failure (ADHF) and community-acquired pneumonia (CAP). Following tailored medical therapy, the patient's condition improved, and she was discharged from the ICU on the fifth day in a stable condition.

Conclusion: This case underscores the importance of early recognition and multidisciplinary management of acute dyspnea in the postpartum period, particularly when linked to PPCM, ADHF, and CAP. Timely intervention with mechanical ventilation and targeted therapy can lead to favorable outcomes, emphasizing the need for heightened awareness among clinicians managing postpartum patients.

Keywords

Postpartum, Acute Pulmonary Edema, Peripartum Cardiomyopathy, Acute Heart Failure, Community-Acquired Pneumonia

How to cite: Sulistiono P, Budipratama D. Management of a P3A0 Postpartum Patient with Peripartum Cardiomyopathy (PPCM), Acute Decompensated Heart Failure (ADHF), Respiratory Failure Due to Acute Pulmonary Edema, and Community-Acquired Pneumonia (CAP) in the ICU. *Journal of Society Medicine*. 2025; 4 (7): 222-226. **DOI: https://doi.org/10.71197/jsocmed.v4i7.224**

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a rare but serious condition that affects women worldwide and is characterized by the onset of heart failure during the peripartum period of pregnancy. It is defined as idiopathic cardiomyopathy with left ventricular dysfunction (left ventricular ejection fraction [LVEF] <45%, with or without ventricular dilatation) occurring in the last month of pregnancy or within six months postpartum in the absence of pre-existing cardiac disease [1]. The diagnostic challenge arises from the overlap of PPCM symptoms, such as dyspnea, fatigue, and edema, with normal physiological changes in late pregnancy and the postpartum period, often leading to a delayed diagnosis [2]. While some patients experience mild disease with the potential for full recovery, others face significant morbidity and mortality due to severe cardiac impairment.

The acute clinical presentation of PPCM typically includes signs of congestion and poor organ perfusion, necessitating urgent therapeutic interventions [3]. Acute decompensated heart failure (ADHF) secondary to PPCM can range from moderate volume overload to cardiogenic shock. Acute pulmonary edema, a critical complication, frequently results from left heart failure and is a primary reason for intensive care unit (ICU) admission, often requiring mechanical ventilation in critical cases [4]. The management of heart failure in this context demands tailored strategies, considering foetal safety during pregnancy or breastfeeding postpartum, with a focus on controlling volume status, mitigating maladaptive neurohormonal responses, and preventing thromboembolism and arrhythmias [5].

Diagnostic complexity increases with concurrent conditions such as community-acquired pneumonia (CAP), which is diagnosed by the presence of new pulmonary infiltrates with clinical evidence of infection [6]. CAP can precipitate acute decompensation of underlying chronic diseases, including congestive heart failure, potentially obscuring the initial diagnostic clarity and delaying treatment. Therefore, maintaining vigilance for alternative diagnoses throughout the clinical course of the patient is essential. This case report aimed to illustrate the intricate management of a postpartum patient with PPCM complicated by ADHF, acute pulmonary edema, and CAP, emphasizing the need for a multidisciplinary approach in the ICU setting.

CASE DESCRIPTION

A 36-year-old woman (gravida 3, para 0, weight 60 kg) presented to the emergency department with a 1-day history of progressive dyspnea that was unrelieved by rest, accompanied by intermittent fever and productive cough for 2 days. She had undergone an uncomplicated vaginal delivery 6 days prior, assisted by a midwife. On admission, the patient was somnolent, with vital signs indicating a blood pressure of 140/90 mmHg, heart rate of 170 beats/min, respiratory rate of 45 breaths/min, oxygen saturation of 90-92% on a 15 L/min non-rebreather mask, and temperature of 37.4°C. Physical examination revealed a jugular venous pressure of 5+4 cmH_2O, bilateral coarse wet rhonchi, regular heart sounds without murmurs, and no peripheral edema. Her medical history was unremarkable for hypertension, cardiac disease, and diabetes, although she reported a childhood history of asthma that had been quiescent for years without the use of regular medication. Initial management in the emergency department included ceftriaxone 1 g IV every 12 h, levofloxacin 750 mg IV daily, paracetamol 1 g orally three times daily, N-acetylcysteine 400 mg orally three times daily, nebulized combivent plus fluticasone every 8 h, furosemide 40 mg IV twice daily, calcium gluconate 2 g IV, and 40% dextrose in two flasks. Owing to the deteriorating respiratory status, she was intubated and transferred to the intensive care unit (ICU) for advanced care.

Table 1: Laboratory Results Before ICU Admission

Parameter	Result
Hematology	Hb 13.6 g/dL, Ht 42.1%, Leukocytes 19,250/µL, Platelets 466,000/µL
Renal Function	Urea 41.3 mg/dL, Creatinine 0.64 mg/dL
Electrolytes	Na 141 mmol/L, K 3.5 mmol/L, Ca 3.94 mmol/L
Glucose	48 → 112 mg/dL (post-correction)
Arterial Blood Gas	pH 7.36, pCO_2 24.4 mmHg, pO_2 78.1 mmHg, HCO_3 14.1 mmol/L, BE -8.6 mmol/L, SaO_2 95%
Procalcitonin	0.43 ng/mL
CRP (Quantitative)	2.88 mg/L
NL Ratio	8.85

Diagnostic evaluation confirmed a complex clinical picture. Chest radiography demonstrated cardiomegaly with pulmonary edema (Figure 1), while electrocardiography showed sinus tachycardia (Figure 2). The laboratory results prior to ICU admission are presented in Table 1. Echocardiography revealed a dilated left ventricle, reduced left ventricular ejection fraction (estimated 30-35%, possibly underestimated due to tachycardia), left ventricular diastolic dysfunction, mild mitral and pulmonary regurgitation, trivial tricuspid regurgitation, low probability of pulmonary hypertension, and normal right ventricular systolic function (TAPSE, 18 mm). Hemodynamic assessment indicated a cardiac output of 4.82 L/min, cardiac index of 3.03 L/min/m^2, and lung ultrasound B profile, consistent with peripartum cardiomyopathy (PPCM) complicated by

acute decompensated heart failure (ADHF), respiratory failure due to acute pulmonary edema, and community-acquired pneumonia (CAP), confirmed by Streptococcus pneumoniae on sputum culture. In the ICU, the initial ventilation was set to pressure-support synchronized intermittent mandatory ventilation (P-SIMV) at a rate of 12 breaths/min, pressure control of 14 cmH_2O, pressure support of 12 cmH_2O, PEEP of 8 cmH_2O, and FiO_2 of 60%, achieving tidal volumes of 430-470 mL and SpO_2 of 97-98%. The therapy was escalated to include midazolam (5 mg/h) IV for sedation, ceftriaxone, levofloxacin, paracetamol (1 g IV every 6 h), omeprazole (40 mg IV every 12 h), digoxin (0.5 mg IV every 24 h), nebulised combivent (every 8 h), furosemide (20 mg/h IV), and fluid restriction. A central venous catheter was placed, and cardiology consultation with hemodynamic and structural echocardiography was performed.

Table 2: ICU Daily Management Overview

Day	Oxygen Therapy	Urine Output / Balance	Therapy and Interventions	Key Laboratory Results
1	P-SIMV: R 12, PC 14, PS 12, PEEP 8, FiO_2: 60%, TV: 430-470 mL	Urine: 2-5 mL/kg/h, -2333 mL/13h	Ceftriaxone, Levofloxacin, Paracetamol, Omeprazole, Digoxin, Furosemide, Nebulized Combivent, CVC, Echo consult	See Table 1 for lab results
2	P-SIMV: R 12, PC 12, PS 10, PEEP 6, FiO_2: 50%, TV: 380-450 mL	Urine: 1.5-3 mL/kg/h, -1300 mL/24h	Dexmedetomidine, Furosemide ↓, Ca gluconate, Digoxin, Nutrition 1000 kcal	Troponin I: 0.48 ng/mL, Glucose: 136 mg/dL
3	Spontaneous: PS 10, PEEP 5, FiO_2: 50%	Urine: 0.5-1.5 mL/kg/h, -147 mL/24h	Norepinephrine, Fluid loading, Enoxaparin, Nutrition 1500 kcal	Troponin I: 0.20 ng/mL, Urea: 120 mg/dL
4	Spontaneous: PS 8, PEEP 5, FiO_2: 40%, Extubation	Urine: 0.5-1.5 mL/kg/h, +545 mL/24h	Norepinephrine/Dexmedetomidine stopped, Bromocriptine, Repeat X-ray	Glucose: 157 mg/dL
5	Nasal cannula 3 L/min	Urine: 0.3-1.5 mL/kg/h, +585 mL/24h	Transfer to HCU	Glucose: 138 mg/dL

Over the subsequent days, the patient's condition improved with the tailored interventions. On day 2, persistent fever and tachycardia prompted a switch to dexmedetomidine (0.4 µg/kg/hour) for sedation, with ventilator settings adjusted to P-SIMV (rate 12, pressure control 12 cmH_2O, pressure support 10 cmH_2O, PEEP 6 cmH_2O, FiO_2 50%) and weaning initiated. Fluid balance was closely monitored, and enteral nutrition at 1000 kcal/24 h was initiated. By day 3, despite reduced rhonchi, hypotension necessitated norepinephrine 0.05 µg/kg/min to maintain mean arterial pressure >65 mmHg, with fluid loading of 4-6 mL/kg and enoxaparin 60 mg SC daily.

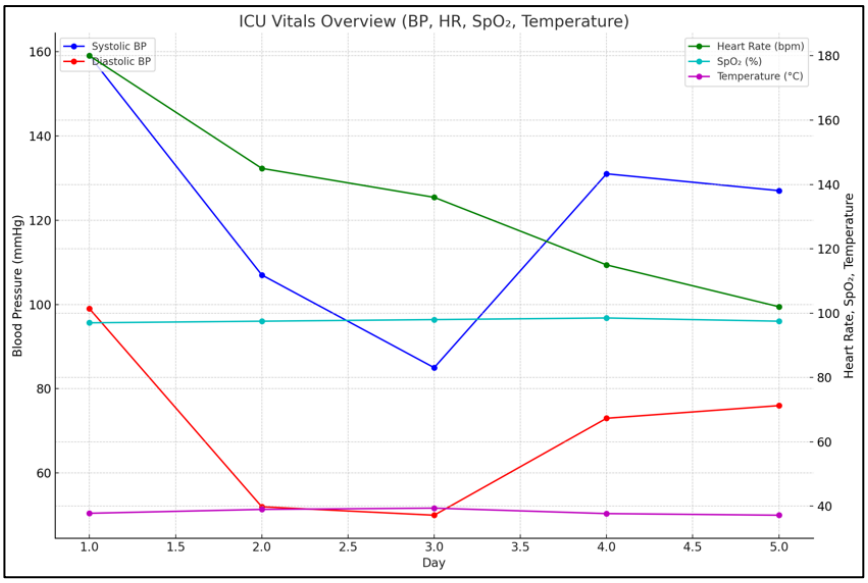

Figure 1. ICU Vitals Overview (BP, HR, SpO_2, Temperature)

The ventilator mode was shifted to spontaneous breathing with a pressure support of 10 cmH_2O, PEEP 5 cmH_2O, and FiO_2 50%, and nutrition was increased to 1500 kcal/24 h. On day 4, clinical improvement allowed the discontinuation of norepinephrine and dexmedetomidine, with extubation following weaning to spontaneous mode (pressure support 8 cmH_2O, PEEP 5 cmH_2O, FiO_2 40%). Daily oral bromocriptine (2.5 mg) was initiated, and a repeat chest radiograph was taken. By day 5, the patient was hemodynamically stable post-extubation, with SpO_2 of 97-99% on a nasal cannula at 3 L/min, and was transferred to the semi-intensive care unit. An overview of the ICU Vitals is presented in Fig. 1.

DISCUSSION

Peripartum Cardiomyopathy (PPCM) is a form of heart failure with reduced left ventricular ejection fraction (LVEF) occurring during the last month of pregnancy or within six months postpartum, without prior heart disease. Diagnosis requires an LVEF of <45% with or without ventricular dilation. The symptoms of PPCM can mimic the normal physiological changes during pregnancy, leading to a delayed diagnosis. Risk factors include advanced age (>30 years), multiparity, hypertension, and family history of heart disease [4,7].

The etiology of PPCM is multifactorial. One key mechanism involves the immune response and hemodynamic stress during pregnancy, which may cause myocardial apoptosis. Prolactin, in its 16 kDa form, has been implicated in the pathophysiology of the disease, as it induces vasoconstriction and endothelial damage. Genetic mutations, particularly in STAT3, predispose individuals to PPCM. Studies have suggested that an imbalance between proangiogenic and antiangiogenic factors plays a role in endothelial dysfunction, increasing the risk of PPCM in genetically predisposed individuals [8,9].

The clinical presentation of PPCM includes dyspnea, fatigue, peripheral edema, and orthopnea. These symptoms overlap with those of other conditions, making differential diagnosis crucial. Echocardiography revealed left ventricular dilation and systolic dysfunction, which are hallmarks of PPCM. Elevated BNP levels and signs of pulmonary edema on chest radiography help confirm the diagnosis [10].

A significant complication of PPCM is thromboembolism due to left ventricular dilation, poor contractility, and endothelial injury. Hypercoagulability during pregnancy increases this risk significantly. Management includes volume control using diuretics and neurohormonal modulation using beta-blockers. In severe cases, bromocriptine inhibits prolactin-induced endothelial injury. Anticoagulation therapy is recommended for patients with low LVEF, particularly during the postpartum period [6].

Acute decompensated heart failure (ADHF), often precipitated by infections such as community-acquired pneumonia (CAP), complicates PPCM. Early administration of broad-spectrum antibiotics, such as ceftriaxone and levofloxacin, followed by de-escalation based on culture results, is essential. Additionally, careful fluid management and supportive care, including sedation and analgesia, are vital for ensuring patient comfort and recovery. PPCM is a serious condition that requires early diagnosis and comprehensive management. Understanding its multifactorial etiology and addressing complications such as thromboembolic events and infections are crucial for improving maternal outcomes [4,11].

CONCLUSION

Acute decompensated heart failure (ADHF) with acute pulmonary edema complicated by community-acquired pneumonia (CAP) in a patient with peripartum cardiomyopathy (PPCM) represents a critical medical emergency that requires aggressive and coordinated management in a specialized unit. Although rare, PPCM is a potentially fatal condition that is challenging to diagnose, predict, and manage. Delays in diagnosis and treatment, along with worsening LVEF and ventricular dilation, significantly worsen prognosis. The management of heart failure in PPCM and CAP must be tailored to the unique considerations of pregnancy and breastfeeding to ensure optimal outcomes.

DECLARATIONS

None

CONSENT FOR PUBLICATION

The Authors agree to be published in the Journal of Society Medicine.

FUNDING

None

COMPETING INTERESTS

The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS

All authors have made substantial contributions to this case report. PS was responsible for patient management, data collection, and initial drafting of the manuscript. All authors reviewed and approved the final version of the manuscript, ensuring its accuracy and integrity, and are accountable for all aspects of the work.

ACKNOWLEDGMENTS

None

REFERENCE

1. Davis MB. Peripartum cardiomyopathy. J Am Coll Cardiol. 2020;75(2):207–221.
2. Honigberg MC. Peripartum cardiomyopathy. BMJ. 2019; 364: 5287.
3. Wilson JH. Peripartum cardiomyopathy. In: Maternal Cardiac Care. 2023;78–82.
4. Johan B. Pathophysiology, diagnosis, and management of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Study Group on peripartum cardiomyopathy. Eur J Heart Fail. 2019; 21:827–843.
5. Teerlink JR. Acute decompensated heart failure update. Curr Cardiol Rev. 2015;11(1):52–63.
6. Njoroge JN, Teerlink JR. Pathophysiology and therapeutic approaches to acute decompensated heart failure. Circ Res. 2021; 128:1468–1486.
7. Evans, et al. Surviving Sepsis Campaign: International Guidelines for the Management of Sepsis and Septic Shock 2021. Crit Care Med. 2021;49(11): e1063-e1143.
8. Hariharan R, Yuji O. Community-acquired pneumonia. Treasure Island (FL): StatPearls Publishing; 2024 Jan.
9. Diagnosis and Treatment of Adults with Community-acquired Pneumonia. An Official Clinical Practice Guideline of the American Thoracic Society and Infectious Diseases Society of America, 2019.
10. Michael SN, Antoni T. Severe community-acquired pneumonia. Eur Respir Rev. 2022; 31:220123.
11. Kementrian Kesehatan. Tatalaksana pedoman penggunaan antibiotik. 2021.