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Case Report

Comprehensive Management of Peripartum Cardiomyopathy with Acute Renal Failure: From Prepartum to Renal Replacement Therapy – A Case Report

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ABSTRACT

Introduction: Peripartum cardiomyopathy (PPCM) is a rare but potentially serious that poses significant diagnostic and therapeutic challenges, particularly in previously healthy women. We report a complex case of severe PPCM in a primigravida patient who needed intensive multidisciplinary care due to respiratory failure and acute renal failure.

Case Presentation: A 21-year-old primigravida in 34 weeks of gestation presented with progressive dyspnea and signs of acute heart failure. Despite an unremarkable medical history, she exhibited severe left ventricular dysfunction (LVEF of 28%), cardiomegaly, and pulmonary congestion. Rapid deterioration necessitated urgent caesarean delivery under general anesthesia due to maternal and fetal distress. Postoperatively, the patient developed cardiogenic shock and acute kidney injury (AKI) necessitating vasopressor and continuous renal replacement therapy (CRRT). Intensive heart failure management, including bromocriptine therapy and guideline-directed medical treatment, led to gradual hemodynamic stabilization. She was successfully weaned off mechanical ventilation and CRRT within days, ultimately being discharged after 12 days of treatment with improved cardiac function.

Conclusion: This case underscores the unpredictable nature and rapid progression of PPCM, even in the absence of traditional risk factors. Early recognition, prompt multidisciplinary intervention, and individualized therapy are critical to improving maternal and neonatal outcomes.

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INTRODUCTION

Peripartum cardiomyopathy (PPCM) is an uncommon yet serious condition that may result in significant maternal morbidity and mortality during late pregnancy or postpartum. It is characterized by left ventricular (LV) systolic dysfunction with an ejection fraction of less than 45%, occurring at the end of pregnancy or within the postpartum period (typically within one month after delivery), with no identifiable etiology for heart failure [1]. The incidence varies widely across regions, with estimates of 1:1124 in Singapore, 1:1:2900 in Malaysia, and up to 1:20,000 in Japan [1–4]. Hoevelman *et al.* reported 1.9% in-hospital mortality of PPCM across all regions, while 6-month all-cause mortality was 8.0%, and 12-month all-cause mortality was 9.8% [5].

The clinical classification of PPCM is categorized as mild PPCM (subacute heart failure with stable hemodynamics), moderate PPCM (acute heart failure, stable hemodynamics, and respiratory insufficiency), and severe PPCM (cardiogenic shock, unstable

hemodynamics, and respiratory insufficiency) [1]. Approximately 21.5% of patients with PPCM required inotropic support; 3.1% required mechanical support; 10% underwent invasive ventilation; and 18.3% experienced acute kidney injury (AKI) [5,6].

Risk factors for PPCM include older age, multiparity, multifetal pregnancy, ethnicity (African-American), family history, malnutrition, smoking, diabetes, preeclampsia, and prolonged use of tocolytic beta-agonists [1,7]. An illustration outlining the risk factors, diagnostic plan, classification, and management of PPCM is presented in Fig.1. Nonetheless, our patient did not meet any of the risk factors, yet she was in moderate-severe PPCM.

CASE PRESENTATION

A 21-year-old female (primigravida) in 34th weeks of gestation came to the emergency room due to complaints of progressive dyspnea on exertion and bilateral leg swelling since two weeks earlier. She routinely attended check-ups with a midwife and an obstetrician. She had normal blood pressure and was receiving standard antenatal care. History of the previous episode of dyspnea on effort or family history of any heart disease was denied. At the emergency room, she had normal blood pressure, a heart rate of 136 bpm, a respiratory rate of 26 x/m, and SpO_2 of 90% on a simple mask at 8 lpm, and 97% on a non-rebreather mask at 15 lpm. The physical examination revealed 2+ pitting edema in both legs on inspection and bilateral basal rales and S3 gallop on auscultation. The fetal heart rate examination showed 172 beats per minute, suggesting fetal distress.

Investigation

Electrocardiography (Fig.2A) revealed sinus tachycardia, while chest X-ray (Fig.2B) suggested cardiomegaly with pulmonary congestion and left pleural effusion. Bedside echocardiography at the emergency room (Fig.2C) showed global hypokinesis with left atrial and left ventricular dilatation (LVIDD 5.8 cm) and decreased LVEF 28% by Teich.

Differential Diagnosis

We assessed the patient as having moderate peripartum cardiomyopathy. The clinical presentation of this patient that meets the criteria of PPCM includes dyspnea on exertion, and swelling of the lower extremity, with echocardiography revealing LV systolic dysfunction with a reduced LVEF 28% by Teich, occurring at the end of pregnancy with no other identifiable causes of heart failure, such as pre-existing idiopathic or familial dilated, acquired cardiomyopathy, Takotsubo syndrome during the last trimester or following an emergency delivery, or pre-existing congenital or valvular heart disease [8].

Treatment

The patient received intravenous vasodilators for relaxation if systolic blood pressure > 110 mmHg (nitroglycerin 10 mcg/minute) along with diuretics. Due to emergency conditions (respiratory distress from cardiogenic acute lung edema secondary to PPCM and fetal distress), a decision was taken to terminate the pregnancy through caesarean delivery under general anesthesia, requiring intubation of the patient. Estimated fetal weight according to gestational age; no fetal growth restriction was found. The baby's Apgar Score was 5-7. Post-operatively, the patient was monitored in the intensive care unit (ICU), remaining intubated and on mechanical ventilation.

In the ICU, she developed unstable hemodynamics, with a blood pressure of 85/53 mmHg (107/63 mmHg on Dobutamine 5 mcg/kgBW/minute), a heart rate of 138 bpm, a respiratory rate of 24 x/m, SpO_2 95% on ventilator mode PC/CMV, PEEP 12cmH_2O, FiO2 100%. The patient was treated based on position statement of ESC 2019 about Management of Peripartum Cardiomyopathy using BOARD scheme treatment (Bromocriptine, Oral HF drugs after tapering down inotropic, Anticoagulant, Relaxant IV vasodilators if SBP > 110mmHg, Diuretics) [1]. We give patients ACE Inhibitors and beta blockers (if they are no longer congested) to treat heart failure.

During ICU monitoring, she showed a trend of anuria for 12 hours, elevated urea levels (from 37.7 to 52.8 mg/dL), creatinine (from 1.18 to 2.1 mg/dL), decreased eGFR (from 65.8 to 32 ml/minute/1.73 m^2), and hyperkalemia (from 3.8 to 6.79 mmol/L). Additionally, the blood gas analysis indicated uncompensated respiratory acidosis. Following a multidisciplinary team discussion, it was determined to consult a nephrologist and initiate continuous renal replacement therapy with a setting mode of CVVHDF, a blood flow of 100 ml/minute, and a fluid loss rate of 80 ml/h. Hemodynamics improved after three days; the patient was successfully weaned from the ventilator after 2 days, and renal function got better (urea 32.4 mg/dL, creatinine 1.00 mg/dL, eGFR 80 ml/minute/1.73 m2), and CRRT was discontinued.

Outcomes and Follow-Up

Shortness of breath and leg swelling were relieved, the patient was transferred to ward following 6 days treated in the ICU, and was discharged after 12 days of treatment.

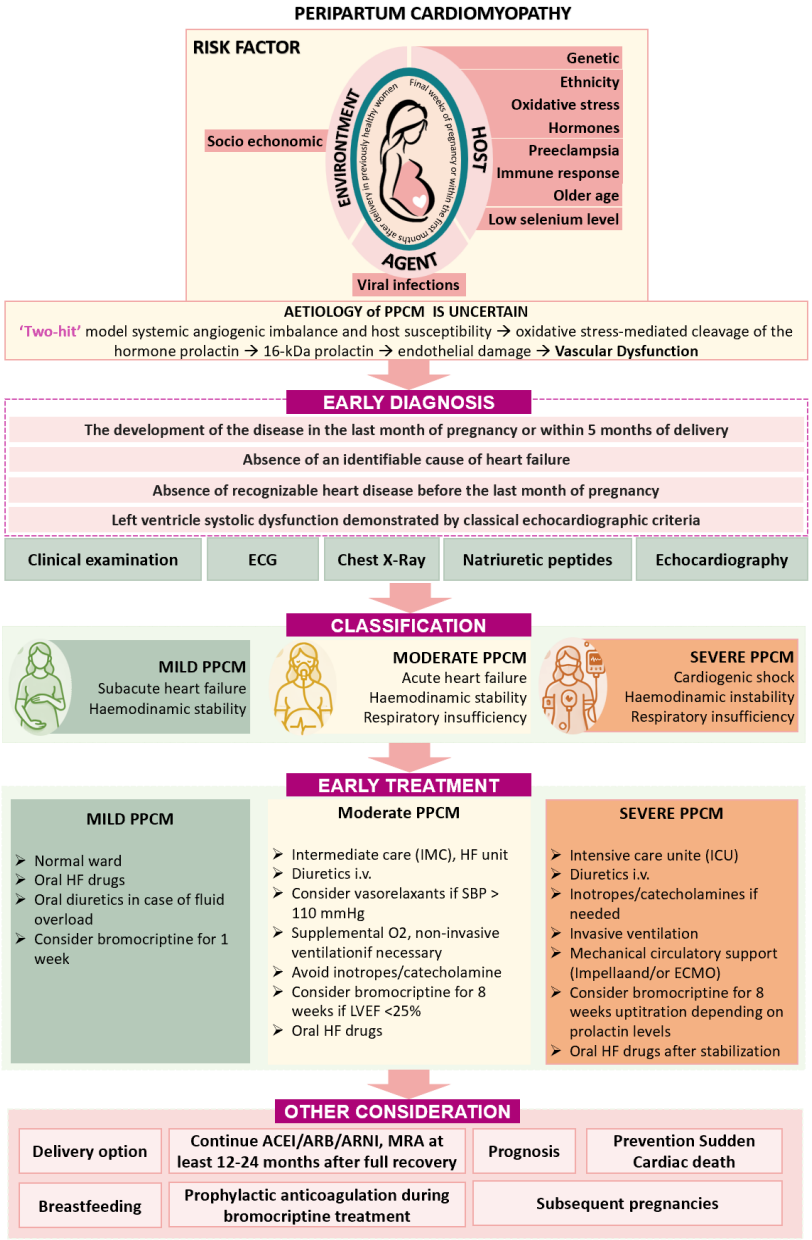


Fig. 1. The following illustration describes the risk factors for patients experiencing peripartum cardiomyopathy, steps for early diagnosis, and treatment based on the degree of PPCM. Comprehensive management after the acute phase till preparation for planning the subsequent pregnancy

DISCUSSION

The etiology of peripartum cardiomyopathy remains unclear and is most likely multifactorial. Nutritional deficiencies, viral myocarditis, and autoimmune processes have all been suggested as potential but unproven mechanisms for the development of PPCM. Hemodynamic stress associated with pregnancy has been proposed as a possible cause [9]. The development of two vascular-hormonal animal experimental models of pregnancy-related cardiomyopathy generated novel theories concerning the pathophysiology of peripartum cardiomyopathy in humans [10].

In the initial model, in a STAT3 mutant mouse, oxidative stress resulted in the cleavage of prolactin, the hormone responsible for regulating breastfeeding. The 16-kDa prolactin fragment induced cardiac and vascular dysfunction, as well as pro-apoptotic and vasculotoxic effects [11]. The second vascular-hormonal mouse

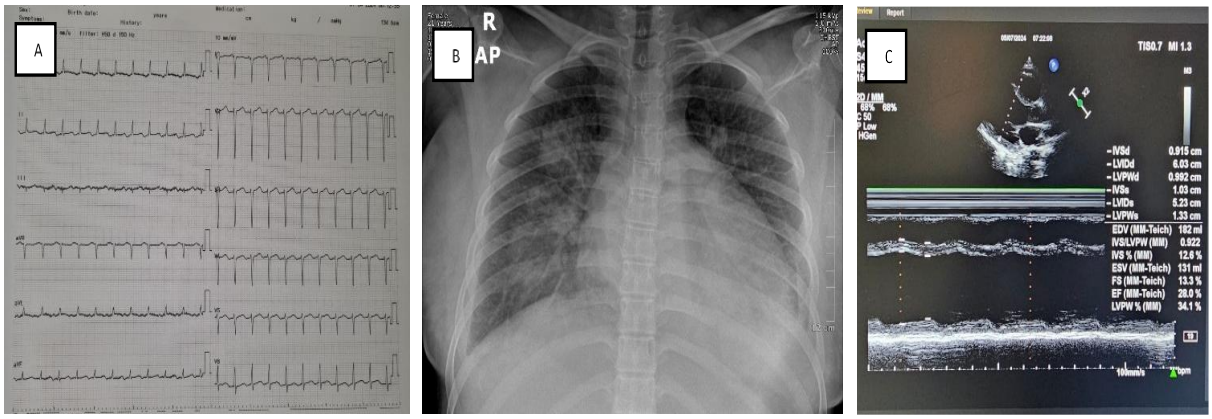


Fig.2. (A) Electrocardiography on admission showed sinus tachycardia at 136 beats per minute. **(B)** Chest X-ray showed cardiomegaly, pulmonary congestion, and left pleural effusion. **(C)** Echocardiography of LV study showed dilatation of LV with reduced LV Ejection Fraction (28% by Teich

model of peripartum cardiomyopathy was developed by genetically eliminating PGC-1a, the proliferator-activated receptor, in the cardiac tissue. This contributed to reduced expression of proangiogenic vascular endothelial growth factor (VEGF) and vasculotoxicity induced by the activation of the 16-kDa prolactin fragment [12].

The treatment of peripartum cardiomyopathy is determined by the severity of the condition [13]. During pregnancy, patients can be given loop diuretics, beta blockers, hydralazine/isosorbide dinitrate, digoxin, and low molecular weight heparin (LMWH). Early delivery can be considered reasonable under unstable conditions. ACE inhibitors, ARBs, and aldosterone receptor antagonists are contraindicated in pregnancy due to their teratogenic effects [2,9]. The initial evaluation in the emergency room revealed that the patient was experiencing moderate peripartum cardiomyopathy (acute heart failure, with stable hemodynamics and respiratory insufficiency). The patient received intravenous diuretics, intravenous vasodilators, and oxygen supplementation (intubation and ventilator support were performed since the patient did not respond to non-invasive ventilation and was planned for a sectional cesarean with general anesthesia). After surgery, the patient was monitored at the ICU, where bromocriptine and anticoagulants were started, and thorough hemodynamic monitoring was performed. Signs of shock were apparent, necessitating an infusion of inotropic support, whereas oral anti-heart failure medication was given after hemodynamics stabilized.

She showed a trend of anuria for 12 hours during observation in the ICU, with an increase in urea (from 37.7 to 52.8 mg/dL), creatinine (from 1.18 to 2.1 mg/dL), decreased eGFR (from 65.8 to 32 ml/minute/1.73m^2), and hyperkalemia (from 3.8 to 6.79 mmol/L). KDIGO defines acute AKI as any elevated serum creatinine of more than 0.3 mg/dl in 48 hours, an elevation of serum creatinine over 1.5 times from baseline, presumed happened within the last 7 days, or a urine production less than 0.5 ml/kg/h sustained for 6 hours [14]. The treatment of acute kidney injury in this patient includes avoiding nephrotoxic drugs, ensuring volume status, considering functional hemodynamic monitoring, and monitoring serum creatinine and urine output.

A multidisciplinary discussion with a nephrologist led to the consideration of renal replacement therapy. There is no established optimal time to start dialysis for AKI. The clinical signs of volume overload and the biochemical signs of solute imbalance (hyperkalemia, azotemia, and severe acidosis) are the principal criteria for initiating renal replacement therapy (RRT). In CRRT, continuous solute clearance of 25–35 ml/minute will stabilize serum markers after 48 hours [14]. Jiajia Zhu's study findings indicate a significant correlation between non-recovered peripartum cardiomyopathy and AKI at admission. Patients with peripartum cardiomyopathy who develop AKI during hospitalization generally exhibit poor recovery of cardiac function, potentially resulting in adverse long-term consequences [15].

A scoring system predicting LV recovery in PPCM at 6 months was derived from the ESC EORP Peripartum registry, with several indicators including LV ejection fraction at admission >35% (1 point), LVEDD < 53 mm (4 points) or LVEDD 53-61 mm (1 point), high human development index (1 point), QRS duration ≤ 80 ms (3 points) or QRS duration 81-109 ms (2 points), duration of symptoms ≤10 days (1 point), and pre-eclampsia (1 point) [16]. Maximum scores are 11, with higher scores correlating with a higher probability of LV recovery (LVEF ≥ 50%). In our patient, she had a total ESC EORP PPCM score of 6 points, and it correlates with 65% LV recovery at 6 months.

Management after outpatient treatment consists of continuing to administer bromocriptine at a dosage of 2.5 mg twice daily for 14 days, then 2.5 mg daily for an additional 42 days, alongside the provision of oral anticoagulants as prophylaxis for thromboembolism during bromocriptine administration [17]. During pregnancy, LMWH is a safe anticoagulant, while postpartum options include LMWH or warfarin. The patient received anti-heart failure treatment, including ACE inhibitors, beta blockers, and spironolactone. Discussion among doctor, patient, and patient's spouses over contraceptive strategies and subsequent pregnancies was done. Non-recovered PPCM patients had a higher risk of relapse of PPCM at the subsequent pregnancy; about 50% showed further reduced LVEF, as well as increased morbidity, mortality, and risk of premature delivery and abortion during pregnancy [11,18]. The increased risk of thromboembolism should discourage the use of estrogen-containing contraceptives during the early postpartum phase when LV dysfunction is obvious. Intrauterine devices or subcutaneous implants that release progesterone are both safe and efficient options [1].

After delivery, the choice of medication therapy for heart failure depends on whether the woman is breastfeeding [19]. Numerous medicines, including beta-blockers, MRAs, and ACE inhibitors, are present in human breast milk, but frequently at low quantities. All patients with PPCM should continue taking a combination of medications until LVEF fully recovers and for a minimum of 12-24 months following the complete recovery of LV function [20]. Prior to discharge, one month post-delivery, and whenever symptoms appear, a clinical evaluation, echocardiography, and BNP should be completed at regular intervals and in response to any concerning symptoms [1,9].

This case report provides a comprehensive case illustration of a primigravida patient with severe PPCM complicated by AKI, offering valuable insights into clinical manifestations, diagnostic findings, therapeutic intervention, and prognostic considerations. A collaborative effort between cardiologists, obstetricians, intensivists, and nephrologists is necessary, reinforcing the importance of a team-based strategy in treating PPCM and its complications. Although the patient had no identifiable risk factors for PPCM, undetected genetic predisposition or subclinical myocardial disease could have contributed to the condition, but genetic screening or myocardial biopsy was not performed due to financial and facility limitations. Further study is needed to evaluate existing knowledge gaps, improve risk stratification, and optimize therapeutic strategies for PPCM patients complicated by AKI.

CONCLUSION

Severe peripartum cardiomyopathy might lead to problems, including acute renal failure. Awareness of heart failure symptoms before, during, and following pregnancy may enhance the identification of PPCM patients. Appropriate strategy and treatment from prepartum to postpartum management requires good team collaboration to minimize morbidity and mortality.

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CONFLICT OF INTEREST

The authors declare there is no conflict of interest.

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