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Case Report

Peripartum cardiomyopathy: a case report and review of current diagnostic and therapeutic strategies

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is a rare but serious cause of heart failure in late pregnancy or the postpartum period. We report a unique case of PPCM with delayed onset and predominant right-sided heart failure in a 41-year-old woman, presenting on postpartum day 14 with peripheral edema and fatigue. Initial findings, including severe anemia and hypoalbuminemia, obscured the diagnosis. Echocardiography revealed biventricular dysfunction and biatrial enlargement. Treatment with beta-blockers and diuretics led to significant recovery. This case highlights the diagnostic challenge of atypical PPCM and underscores the need for vigilance and cardiac evaluation in postpartum patients with heart failure signs.

Keywords: Peripartum cardiomyopathy, Postpartum heart failure, Echocardiography, Bromocriptine, Left ventricular dysfunction

INTRODUCTION

Peripartum cardiomyopathy (PPCM), also known as Meadows syndrome, was first described in 1997 by a working group of the National Heart, Lung, and Blood Institute (NHLBI). PPCM is defined as congestive heart failure with a left ventricular ejection fraction (LVEF) less than 45% and/or left ventricular dilatation (end-diastolic diameter >2.7 cm/m^2), occurring during the third trimester of pregnancy or within five months postpartum, in the absence of pre-existing cardiac disease and with a negative etiological work-up.[1] PPCM is a rare clinical entity with a physiopathology that remains partially understood. The aim of this study is to describe the clinical, diagnostic, and therapeutic characteristics of this condition. Despite its rarity, early recognition is crucial, particularly by obstetricians, due to its potentially severe progression and the need for prompt multidisciplinary management.

CASE REPORT

A 41-year-old woman, gravida 2 para 2, was admitted to the cardiac intensive care unit due to acute decompensated heart failure occurring on postpartum day 14 following an uncomplicated vaginal delivery. This was her first full-term pregnancy, which had progressed without incident, and she had no prior medical history. The immediate postpartum period was uneventful until the gradual onset of peripheral edema extending to the gluteal region, orthopnea, and asthenia.

Upon admission, the patient was conscious, with a blood pressure of 110/80 mmHg, marked tachycardia at 148 bpm, orthopnea alleviated by a semi-recumbent position, and oxygen saturation at 94% on ambient air. She was afebrile and reported no chest pain. Clinical examination revealed pale conjunctivae and bilateral lower limb pitting edema extending to the gluteal region. Abdominal

examination demonstrated distension with shifting dullness, without tenderness or guarding, and lochia of normal quantity. Cardiopulmonary auscultation identified regular heart sounds without murmurs or friction rubs, and clear lung fields without crackles. However, signs of right-sided heart failure were present, including jugular venous distension, massive lower extremity edema, and significant ascites.

Laboratory investigations showed severe anemia (hemoglobin 5.6 g/dl, hematocrit 19.7%), mild inflammatory response (C-reactive protein 30 mg/l), preserved renal function, and hypoalbuminemia (25 g/l). Liver enzymes were moderately elevated (AST 53 IU/l, ALT 36 IU/l, LDH 503 IU/l), along with increased total and direct bilirubin levels (6.2 mg/l and 3 mg/l, respectively). Electrolyte panel was within normal limits.

Abdominopelvic ultrasound revealed massive ascites with an empty uterus and a normal endometrial stripe extending to the uterine fundus. Doppler ultrasound of the lower extremities confirmed preserved arterial flow and excluded deep vein thrombosis.

An electrocardiogram performed in the emergency department demonstrated rapid atrial fibrillation with an average ventricular rate of 148 bpm, right axis deviation, and no repolarization abnormalities (Figure 1).

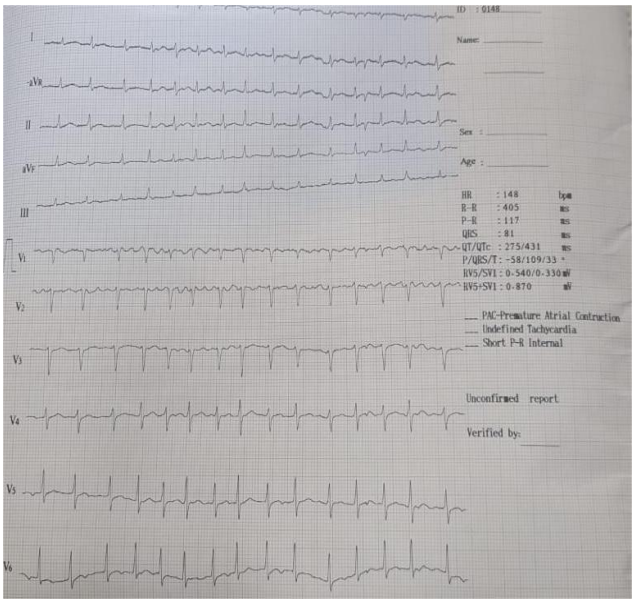

Figure 1: Electrocardiogram showing a rapid atrial fibrillation with an average ventricular rate of 148 bpm and right axis deviation.

Transthoracic echocardiography (TTE) revealed a dilated cardiomyopathy with biventricular systolic dysfunction, an estimated left ventricular ejection fraction of 42%, significant biatrial enlargement, and a dilated inferior vena cava measuring 26 mm, suggestive of elevated right-sided filling pressures (Figure 2).

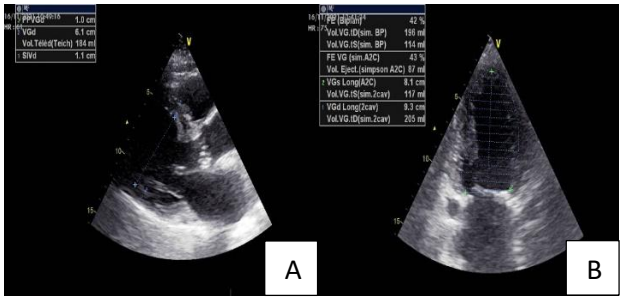

Figure 2 (A and B): Transthoracic echocardiogram showing a dilated left ventricle with reduced ejection fraction.

Given the postpartum status, elevated D-dimer levels, and right heart involvement, a chest computed tomography (CT) angiogram was performed. It showed cardiomegaly with pulmonary arterial hypertension (Figure 3), but no evidence of pulmonary embolism. There was a small pericardial effusion, minimal right-sided pleural effusion, a thin left pleural effusion, and bilateral alveolar-interstitial pneumonia. Coronary angiography revealed normal coronary arteries.

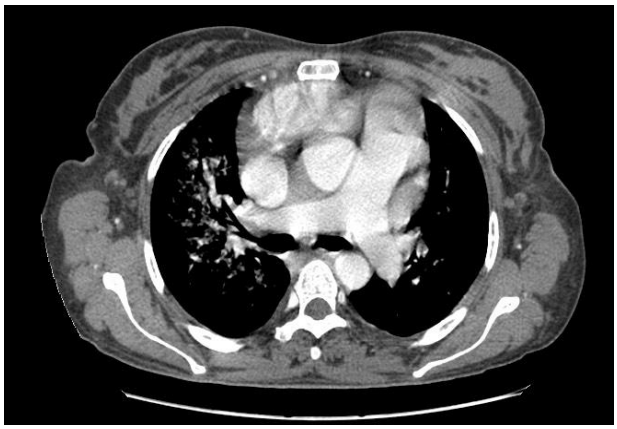

Figure 3: Chest CT angiogram showing cardiomegaly with pulmonary arterial hypertension.

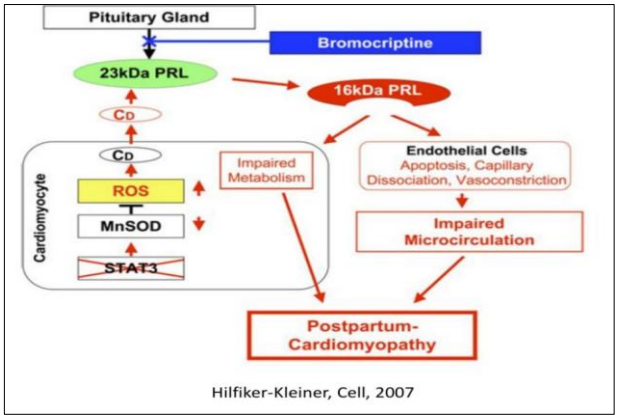

Figure 4: Mechanism proposed by Hilfiker-Kleiner's team involves a cleaved fragment of prolactin contributing to the development of PPCM.

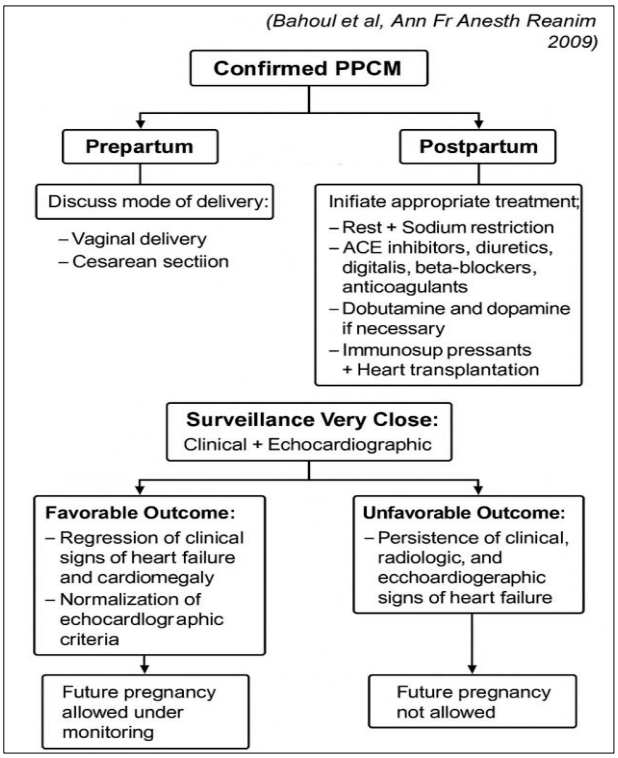

Figure 5: Management algorithm for peripartum cardiomyopathy (PPCM): from diagnosis to prognosis-based recommendations.

Under medical management combining loop diuretics and beta-blockers, the patient showed notable clinical and echocardiographic improvement, with the left ventricular ejection fraction improving to 50% after six months of therapy.

DISCUSSION

Peripartum cardiomyopathy is an idiopathic form of cardiomyopathy that typically occurs in late pregnancy or in the months following delivery, most frequently during the first postpartum month.[2] It is a diagnosis of exclusion in a pregnant or postpartum woman presenting with heart failure symptoms and left ventricular systolic dysfunction in the absence of other identifiable causes.[3] The key diagnostic criterion is an LVEF less than 45%, with or without ventricular dilation. Clinical outcomes vary widely, from full recovery of cardiac function to persistent dysfunction or rapid progression to advanced heart failure.[3] The incidence ranges from 1 in 1,500 to 1 in 4,000 live births depending on geographic region.[4]

Several risk factors have been associated with PPCM. The most commonly reported is maternal age over 30 years, followed by multiparity, which increases cumulative cardiovascular stress. Multiple gestations represent another significant risk due to greater hemodynamic load. Obesity contributes through its impact on metabolic and cardiovascular regulation. Hypertension and preeclampsia are strongly associated as well, reflecting vascular dysregulation. Prolonged tocolysis, though less frequent, has also been implicated, likely through its hemodynamic effects.[5]

Various pathophysiological hypotheses have been proposed. One involves inadequate adaptation to pregnancy-induced hemodynamic changes, such as increased cardiac output and plasma volume with decreased peripheral vascular resistance. Other theories include an abnormal autoimmune response with generation of cardiomyocyte-specific autoantibodies, or viral myocarditis.[6]

A novel mechanism proposed by Hilfiker-Kleiner's team involves a cleaved fragment of prolactin.[6] Myocardial oxidative stress increases cathepsin D activity, leading to the release of a 16 kDa prolactin fragment with anti-angiogenic and pro-apoptotic properties. This peptide impairs myocardial perfusion and contractility, thereby contributing to the development of PPCM (Figure 3).[8]

From a clinical perspective, the condition typically manifests as rapidly progressive acute heart failure, often severe in nature, and in some cases predominantly affecting the left ventricle, with deterioration occurring within a matter of hours.[6] Chest pain is reported in approximately 50% of cases, typically presenting as atypical precordial discomfort, angina-like pain, or symptoms resembling myocardial infarction. Electrocardiographic findings are often nonspecific, although left bundle branch block or T-wave inversions may occasionally be detected. Transthoracic echocardiography remains the gold standard for diagnosis, as it confirms ventricular dilation, reveals a LVEF below 45%, and may also demonstrate right ventricular involvement.[9] Moreover, it provides valuable information on potential complications, including intracavitary thrombosis and pericardial effusion, while aiding in the exclusion of underlying structural heart disease. Cardiac MRI provides prognostic insights through late gadolinium enhancement, typically located in the subepicardial region, with the extent of enhancement appearing to correlate with functional recovery.[10]

Management is based on established guidelines for the treatment of chronic heart failure (Figure 5), including a combination of beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, and diuretics. In severe cases, intravenous inotropic support may be required. Given the thromboembolic risk, anticoagulation therapy is recommended. Even in cases of complete recovery of systolic function, long-term medical therapy is advised, as treatment discontinuation does not consistently prevent relapse.[11] In non-responding patients, cardiac resynchronization therapy, left ventricular assist devices, or even heart transplantation may be considered, depending on clinical progression. Bromocriptine, a prolactin antagonist, has demonstrated significant efficacy in this setting, further supporting the pathogenic role of prolactin fragments.[6]

An open-label study involving 20 patients compared the combination of ACE inhibitors and bromocriptine with ACE inhibitor therapy alone. Bromocriptine was administered at a dose of 2.5 mg twice daily for two weeks, followed by 2.5 mg once daily for an additional six weeks. At six months, patients receiving bromocriptine exhibited higher left ventricular ejection fraction on cardiac MRI, reduced mortality, and a significantly lower incidence of major adverse clinical events, including death, NYHA class III/IV heart failure, or an ejection fraction below 35%.[12]

Effective contraception is crucial in the management of these patients, particularly following an initial episode of peripartum cardiomyopathy. In cases of persistent left ventricular dysfunction, subsequent pregnancy is strictly contraindicated due to the associated life-threatening risk. If complete functional recovery is achieved, pregnancy may be considered; however, patients must be thoroughly counselled regarding the potential risks.

The progression of peripartum cardiomyopathy (PPCM) is unpredictable. An initial ejection fraction (EF) of <30% and a ventricular dilation >27 mm/m² are markers of poor prognosis.[13] However, complete recoveries are possible, even in initially severe cases.[14] Thus, the persistence of dysfunction beyond the sixth postpartum month is a negative prognostic factor, although late improvements, sometimes extending beyond three years, have been reported.[14]

This case is distinctive due to its atypical presentation, complex diagnostic context, and favorable recovery. The patient developed symptoms on postpartum day 14, a timing that stresses the need for continued clinical vigilance beyond immediate delivery. Unlike the classical form, the presentation was marked by right-sided heart failure signs: massive ascites, peripheral edema, and jugular distension rather than overt pulmonary congestion, making diagnosis more challenging. Additionally, severe anemia and hypoalbuminemia further complicated the clinical picture, potentially masking the underlying cardiac dysfunction. Despite this, a comprehensive diagnostic approach, including echocardiography, chest CT angiography, and coronary angiography, allowed clinicians to exclude thromboembolic and ischemic causes and confirm PPCM. The patient responded well to guideline-directed therapy, with a recovery of left ventricular ejection fraction from 42% to 50% within six months. This case not only illustrates an uncommon form of PPCM but also underscores the importance of multidisciplinary evaluation and timely management, offering valuable educational insight for both cardiologists and obstetricians.

CONCLUSION

Peripartum cardiomyopathy is a rare but life-threatening condition that requires prompt recognition and multidisciplinary management. Although its exact pathophysiology remains unclear, emerging evidence particularly the role of prolactin fragments has opened new therapeutic avenues. Early diagnosis through echocardiography and exclusion of other cardiac diseases is essential. Management is based on standard heart failure treatment, with promising results seen with bromocriptine in select cases. Prognosis varies, but even severe cases may recover fully with appropriate care. Long-term follow-up and effective contraception are crucial, especially for patients with persistent cardiac dysfunction. Enhancing awareness among healthcare professionals is key to improving outcomes for both mother and child.

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