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(CASE REPORT)

Peripartum dilated cardiomyopathy: Case report and literature review

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Abstract

Peripartum cardiomyopathy, also known as primary gravid cardiomyopathy, is a rare and underrecognized condition defined as systolic heart failure occurring during the last month of pregnancy or within the first five months postpartum, in the absence of any identifiable cause or preexisting heart disease. We report the case of a 31-year-old woman with no significant medical history, admitted for an episode of left-sided heart failure one month after delivery. The peripartum context raised suspicion for this diagnosis, prompting further investigations including echocardiography and cardiac MRI, which confirmed the condition.

Keywords: Peripartum cardiomyopathy; Heart failure; pregnancy; Transthoracic echocardiography; Cardiac MRI

1. Introduction

- **Peripartum cardiomyopathy** is a rare cause of dilated cardiomyopathy that occurs at the end of pregnancy or in the months following delivery. The diagnosis is based on the combination of clinical signs of heart failure and left ventricular systolic dysfunction as demonstrated by echocardiography. Although several pathophysiological hypotheses have been proposed, the exact causes of this condition remain unknown. The clinical course is unpredictable — sometimes favorable with complete remission, but often marked by persistent or worsening heart failure, which can be life-threatening. The risk of recurrence in a subsequent pregnancy, even after apparent remission, is very high.
- **Cardiac MRI** is an advanced imaging technique whose usefulness is now well established in the assessment of idiopathic dilated cardiomyopathies. However, there is limited data regarding the specific role of cardiac MRI in the context of peripartum cardiomyopathy.

2. Clinical Case

We report the case of a 31-year-old woman who was admitted to the cardiac intensive care unit following an episode of left-sided heart failure one month after an uncomplicated vaginal delivery. The patient was at 38 weeks of amenorrhea at the time of delivery, and the pregnancy had progressed normally. She had no significant medical history, particularly no history of heart disease.

Upon admission, the patient presented with orthopnea and tachycardia at 120 bpm, with a blood pressure of 100/65 mmHg. Cardiac auscultation revealed a grade 2/6 systolic murmur consistent with mitral regurgitation, associated with a left-sided protodiastolic gallop. Pulmonary auscultation revealed crackles in both lung fields without peripheral signs of right heart failure.

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A resting electrocardiogram showed sinus tachycardia at 120 bpm, with no repolarization abnormalities or signs of ventricular hypertrophy. A frontal chest X-ray revealed cardiomegaly, interstitial syndrome, and signs of pulmonary vascular congestion.

Biologically, there were no electrolyte disturbances (serum sodium: 138 mmol/L, potassium: 3.9 mmol/L) and no signs of renal dysfunction. Troponin and NT-proBNP levels were elevated, at 0.20 ng/mL (normal < 0.04 ng/mL) and 500 pg/mL (normal < 300 pg/mL), respectively.

Transthoracic echocardiography revealed features of hypokinetic dilated cardiomyopathy (Figure 1), with impaired left ventricular function. The left ventricular end-diastolic diameter measured 65 mm, and the end-systolic diameter measured 51 mm, with an ejection fraction of 31% by biplane Simpson method (Figure 2). Left ventricular filling pressures were elevated (Figure 3), and a grade II functional mitral regurgitation was noted.

Coronary angiography demonstrated angiographically normal coronary arteries.

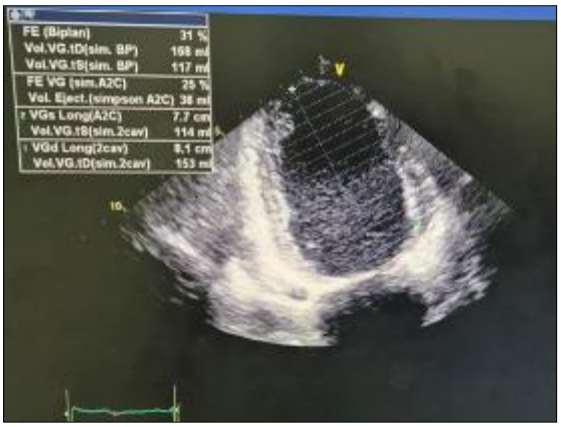

Figure 1 Impaired systolic function at 31%

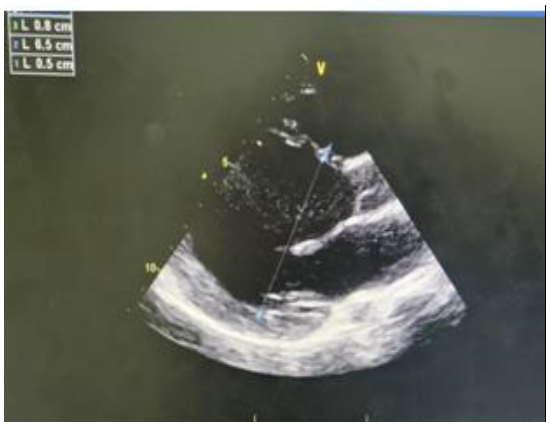

Figure 2 Left ventricular dilation

A cardiac MRI was performed: cine-MRI sequences confirmed global hypokinesia of the left ventricle, with a left ventricular ejection fraction (LVEF) calculated at 31% (Figure 4). On late gadolinium enhancement sequences obtained 10 minutes after contrast injection, a nodular mid-myocardial enhancement was observed, limited to the septal wall (Figure 5).

The clinical course under medical therapy—including loop diuretics, mineralocorticoid receptor antagonists, SGLT2 inhibitors, angiotensin receptor-neprilysin inhibitors (ARNI), and beta-blockers—was marked by both clinical and echocardiographic improvement, with the ejection fraction increasing to 50% after six months of treatment.

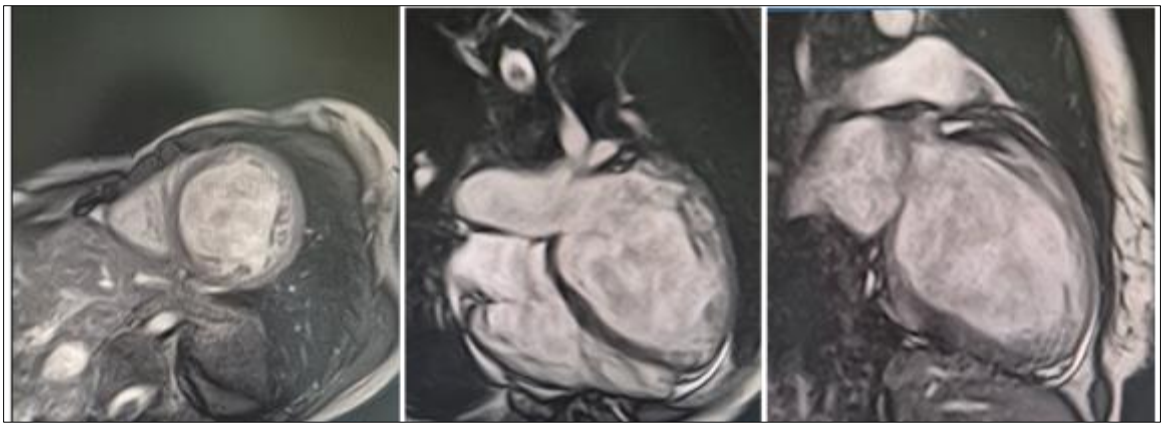

Figure 3 Cine MRI sequences showing a dilated left ventricle with global hypokinesia

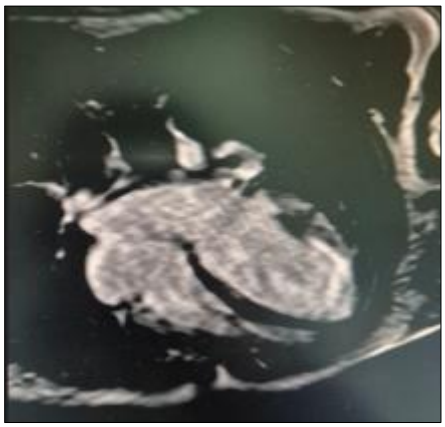

Figure 4 Delayed enhancement sequence in the four-chamber view showing a very limited mid-myocardial late enhancement in the septal wall

3. Discussion

Peripartum cardiomyopathy (PPCM) is defined by the European Society of Cardiology (ESC) as "a dilated cardiomyopathy presenting in the peripartum period in previously healthy women" [1]. It is characterized by left ventricular systolic dysfunction with reduced left ventricular ejection fraction (LVEF) on echocardiography, occurring in the last month of pregnancy or within the first five months postpartum [1]. Its incidence ranges from 1 in 1,500 to 1 in 4,000 live births [2], with significant geographic variation.

Several risk factors for PPCM have been identified, including maternal age over 30 years, multiparity, multiple pregnancies, obesity, hypertension, preeclampsia, and prolonged tocolysis [3]. Numerous pathophysiological hypotheses have been proposed, such as inadequate adaptation to the hemodynamic changes of pregnancy—linked to increased cardiac output, expanded plasma volume, and altered peripheral vascular resistance. An abnormal autoimmune response to pregnancy involving the production of specific cardiac autoantibodies has also been suggested. Other studies point to viral myocarditis as a potential trigger [4].

The research team led by Hilfiker-Kleiner highlighted the role of a prolactin cleavage product in PPCM pathogenesis [5]. It has been shown that increased oxidative stress within the myocardium enhances the enzymatic activity of cathepsin D, leading to its release into the bloodstream, where it cleaves prolactin into a 16-kDa peptide. This peptide has anti-angiogenic and pro-apoptotic properties, resulting in inadequate myocardial vascularization and contractile dysfunction of cardiomyocytes.

The classical presentation is that of global heart failure, sometimes limited to left-sided symptoms, typically severe and with a very rapid onset—sometimes within just a few hours [4]. Chest pain occurs in nearly 50% of cases, either as

atypical precordial pain or angina-like, and sometimes mimicking myocardial infarction. The electrocardiogram (ECG) usually shows no specific features, but may occasionally reveal a left bundle branch block or negative T waves.

Transthoracic echocardiography is the key diagnostic tool. It confirms the diagnosis and allows for monitoring of the disease progression. It typically shows ventricular dilation and reduced ejection fraction below 45%, and may also reveal right ventricular involvement [6]. It is also essential for identifying potential complications such as intracavitary thrombi or associated pericardial effusion. Finally, echocardiography helps rule out preexisting cardiac diseases such as hypertrophic, rheumatic valvular, or ischemic heart disease.

Cardiac MRI would help better understand the pathophysiological mechanisms involved in the onset of peripartum cardiomyopathy (PPCM), assist in the positive diagnosis, and contribute to the follow-up of affected patients. It allows for reliable and reproducible measurements of ventricular volumes and LVEF, which are important for monitoring the disease. Additionally, the presence and persistence of myocardial contrast enhancement in late-phase images could serve as a prognostic factor for the disease and may be correlated with the absence of functional recovery of the left ventricle in the medium and long term. Prospective studies involving large numbers of patients are needed to confirm or refute this hypothesis [7].

The treatment of PPCM follows the guidelines for chronic heart failure, with a combination of beta-blockers, mineralocorticoid receptor antagonists, SGLT2 inhibitors, Angiotensin Receptor-Neprilysin Inhibitors, and diuretics. In severe cases, intravenous inotropic treatment may be necessary. Given the risk of thromboembolic events, anticoagulant therapy is recommended. In cases of complete recovery of systolic function, long-term treatment should continue, although discontinuation does not seem to lead to further deterioration of systolic function [8]. In the absence of recovery, and depending on the clinical progression, options such as cardiac resynchronization therapy, left ventricular assist devices, and, as a last resort, heart transplantation may be considered.

Bromocriptine (anti-prolactin) has proven effective, based on the pathophysiological hypothesis that low molecular weight prolactin is cardiotoxic and triggers PPCM. A study compared treatment with ACE inhibitors alone versus ACE inhibitors combined with bromocriptine in an open-label trial with 20 women. The diagnosis was made within a month postpartum. The patients received 2.5 mg of bromocriptine twice daily for 2 weeks, followed by once daily for 6 weeks. At six months, the patients treated with bromocriptine showed, compared to the control group: a higher LVEF measured by MRI, lower mortality, and a lower incidence of the primary endpoint (defined by death and/or heart failure NYHA class III/IV and/or LVEF < 35%) [9].

Effective contraception is crucial for these young women after their first episode of PPCM. In cases of persistent dysfunction, any subsequent pregnancy should be discouraged and contraindicated due to the high risk of mortality. If systolic function has normalized, a subsequent pregnancy may be considered, but the risks of complications should be clearly explained. The clinical course of peripartum cardiomyopathies is variable. The initial left ventricular dysfunction (LVEF < 30%) and ventricular dilation (greater than 27 mm/m^2) are poor prognostic factors [10]. However, individual prediction remains difficult, and it is common to observe full recovery even in patients with initially severe systolic dysfunction [11]. The persistence of left ventricular dysfunction beyond six months postpartum is a poor prognostic indicator Prognosis**.** However, some data also report late full recoveries, with delays of more than 3 years [11].

4. Conclusion

Peripartum cardiomyopathy is a severe cardiac complication of pregnancy. Often underdiagnosed and of multifactorial origin, its extremely rapid and entirely unpredictable progression justifies multidisciplinary management in a specialized center to improve maternal and fetal prognosis.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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