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Primipara with peripartum cardiomyopathy: A case report

Abstract

Peripartum Cardiomyopathy (PPCM) is a rare but potentially fatal idiopathic cardiomyopathy that predominantly occurs in the late stages of pregnancy or within several months postpartum. It is clinically manifested as heart failure, posing a severe threat to the life of pregnant and postpartum women. In recent years, the incidence of PPCM has been on the rise. Timely diagnosis and treatment can significantly reduce mortality. We report a case of a 29-year-old primiparous woman who presented with chest pain as the initial symptom of PPCM 5 days after cesarean section. At admission, she had Left Ventricular Systolic Dysfunction (LVEF 32%). One month later, her cardiac function had significantly improved (LVEF 62%). This article, through the analysis of a case of PPCM that occurred early after cesarean section, focuses on the recognition points of atypical clinical manifestations and the optimization of treatment strategies. We aim to enhance clinicians' awareness of peripartum cardiomyopathy, emphasizing the importance of early diagnosis and comprehensive management to improve patient outcomes.

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Introduction

Peripartum Cardiomyopathy (PPCM) is an idiopathic cardiomyopathy with a unique temporal window, traditionally defined as a condition in which women with no prior history of heart disease develop myocardial contractile dysfunction with a Left Ventricular Ejection Fraction (LVEF) below 45% for the first time during the last month of pregnancy or within 5 months postpartum, excluding other causes of heart failure [1,2]. Some studies have found that peripartum cardiomyopathies occurring earlier or slightly later in pregnancy also fully meet the characteristics of PPCM. Therefore, the 2019 ESC updated expert consensus has broadened the previously strict time frame for PPCM to the late pregnancy period and several months postpartum [3]. Moreover, some studies propose classifying PPCM into early-onset PPCM (occurring during months 1-9 of pregnancy) and late-onset PPCM (occurring 6-12 months postpartum) to more comprehensively encompass the temporal variability in disease manifestation [4]. Other research has shown that most cases of PPCM occur postpartum, primarily in the first month after delivery [5].

The pathogenesis of PPCM has not yet been fully elucidated, and it is likely to involve multiple factors, including genetic factors as well as immune responses, hemodynamic changes, and hormonal dysfunction [5]. PPCM exhibits diverse clinical manifestations, and its early symptoms are often mistaken for normal physiological changes during pregnancy, leading to delayed diagnosis. Without timely intervention, PPCM may progress to permanent refractory heart failure, posing serious threats to both maternal and fetal health. Therefore, early recognition and intervention for PPCM are crucial for improving maternal and neonatal outcomes.

Case presentation

A 29-year-old woman was admitted to the Department of Cardiology of the First Affiliated Hospital of Dali University on October 13, 2022, with complaints of "recurrent chest pain for over 2 months, and then aggravated for 3 days". Five days after delivering a female infant via cesarean section two months prior, the patient developed fixed, stabbing pain in the left precordial region, lasting several seconds before resolving spontaneously. The pain had no identifiable aggravating or relieving

factors, did not radiate to the shoulders or back, and was not accompanied by chest tightness, dyspnea, or other discomforts. The patient did not pay attention to it and did not seek medical treatment. Three days before admission, she was awakened at night by breathlessness, which could be relieved by sitting up, without profuse sweating. Subsequently, she developed stabbing precordial pain that could radiate to the shoulders and back, accompanied by chest tightness, slight exertion-induced panting, decreased exercise tolerance, and relief with rest. She presented to our hospital for further systematic diagnosis and treatment. No prior medical history, denies history of hypertension, diabetes mellitus, hyperlipidemia or heart disease. Married, with one previous cesarean delivery 2 months ago.

Physical examination upon admission: Height: 155 cm, Weight: 92 kg, Body Mass Index (BMI): 38.3 kg/m^2. Temperature (T) 36.6℃, Pulse (P) 113 bpm, Respiratory rate (R) 20 breaths per minute, Blood Pressure (BP) 114/74 mmHg. The patient is conscious, with no jugular venous distention. The hepatojugular reflux sign is negative. Bilateral lung sounds are clear, with a few moist rales heard in the lower lobes of both lungs. The apical impulse is located 1.0 cm lateral to the midclavicular line at the 5th intercostal space. Cardiac dullness is enlarged to the left on percussion. The heart rate is 113 bpm, with a regular rhythm. No significant murmurs are heard in all valve auscultation areas. The abdomen is soft, without tenderness or rebound tenderness. There is no edema in both lower limbs.

Auxiliary examinations: BNP 946 pg/ml (normal range: <100 pg/ml); Biochemical blood tests: Alanine aminotransferase 121 U/L (normal range: 0-50 U/L); Aspartate aminotransferase 55 U/L (normal range: 0-50 U/L); Gamma-glutamyl transferase 83 U/L (normal range: 11-50 U/L); Uric acid 628 umol/L (normal range: 150-360 umol/L); Calcium 2.11 mmol/L (normal range: 2.15-2.55 mmol/L); Bicarbonate 19.50 mmol/L (normal range: 22-29 mmol/L); Triglycerides 1.57 mmol/L (normal range: 0.40-1.53 mmol/L); Apolipoprotein A1 0.67 g/L (normal range: 1.0-1.6 g/L); Homocysteine 15.3 umol/L (normal range: 0-15 umol/L); NT-pro BNP: 3528.00 pg/ml (0-125 to rule out chronic heart failure; 0-300 to rule out acute heart failure; for acute heart failure diagnosis stratified by age, <50 years: >450); Other tests: Blood routine, urine routine, stool routine, coagulation tests, thyroid function, rheumatoid arthritis, antinuclear antibodies, anticardiolipin antibodies, procalcitonin, interleukin, sputum smear, sputum culture, HIV/HCV/HBV tests showed no significant abnormalities. Electrocardiogram (ECG): Sinus tachycardia, T wave changes in some leads; Abdominal ultrasound: Mild fatty liver appearance, multiple polypoid lesions in the gallbladder; Echocardiogram: LVEDD 54 mm, LAD 43 mm, LVESD 46 mm and LVEF 33%. Left ventricular enlargement, mild mitral regurgitation, reduced left ventricular systolic and diastolic function, mild pulmonary and tricuspid valve regurgitation, minimal pericardial effusion. Chest CT: Scattered infections in both lungs, minimal pleural effusion on both sides, enlarged cardiac silhouette, fatty liver. Echocardiography with contrast: Left ventricular opacification showed normal left ventricular wall motion, LVEF 32%; no obvious contrast agent filling defects in the left ventricular cavity; no obvious perfusion defects in the left ventricular myocardium. Other imaging: Head CT and neck ultrasound showed no significant abnormalities.

Based on clinical manifestations, symptoms, physical signs, echocardiography, and contrast echocardiography results, the diagnosis was PPCM. Upon admission, the patient was prescribed a low-salt, low-fat diet and treated with recombinant human brain natriuretic peptide and furosemide injection via micro-infusion to improve cardiac function, sacubitril/valsartan to reverse ventricular remodeling, metoprolol succinate in combination with digoxin to control ventricular rate, spironolactone + furosemide for diuresis, empagliflozin to improve myocardial remodeling, Qiliqiangxin capsules for promoting blood circulation and unblocking collaterals, polyene phosphatidylcholine for liver protection, and sodium bicarbonate to lower uric acid levels, among other symptomatic and supportive treatments. The initial dose of metoprolol succinate was 47.5 mg once daily (qd). After 2 days, the patient's heart rate remained slightly elevated. The senior physician recommended increasing the dose of metoprolol succinate to 71.25 mg qd and maintaining digoxin at 0.125 mg qd. After 2 days of treatment with the adjusted regimen, the patient's heart rate was well-controlled throughout the day, with fluctuations 80-95 bpm. To effectively control the ventricular rate and improve long-term prognosis, the dose of metoprolol succinate was increased, and the dose of digoxin was reduced to 0.0625 mg qd, with metoprolol succinate adjusted to 47.5 mg twice daily (bid). The patient's heart rate has decreased compared to before, fluctuating between 65-77 bpm, with significant improvement in other indicators. The patient requested discharge.

Discharge diagnosis: (1) Acute left heart failure, Class IV heart function (NYHA); (2) Peripartum cardiomyopathy; (3) Cardiomegaly; (4) Sinus tachycardia; (5) Hepatic dysfunction; (6) Hyperuricemia; (7) Mild fatty liver; (8) Gallbladder polyps.

At discharge, the patient had no recurrence of chest pain, no discomfort such as chest tightness or dyspnea, could lie flat at night, and had no moist rales heard in both lungs. Rechecked NT-proBNP was 440.90 pg/ml. The patient was discharged with the following medications: sacubitril/valsartan 100 mg bid, metoprolol succinate 47.5 mg bid, digoxin 0.0625 mg qd, Qiliqiangxin capsules 1.2 g tid, empagliflozin 10 mg qd, spironolactone 20 mg qd, furosemide 20 mg qd, and polyene phosphatidylcholine 456 mg tid.

Follow-up after discharge: One month after discharge, echocardiography showed LVEDD 48 mm, LAD 34 mm, LVESD 32 mm and LVEF 62%. The patient had returned to normal work and daily life.

Discussion

Patients with PPCM typically present with symptoms characteristic of heart failure, including dyspnea, fatigue, cough, orthopnea, and nonspecific symptoms of cardiac congestion such as abdominal discomfort, chest pain, and palpitations [6]. Cardiogenic shock can occur in a minority of patients, while arrhythmia and arterial thromboembolism are rare. Physical examination may reveal signs of left heart failure such as lung rales, signs of right heart failure such as jugular venous distension and edema, and S3 gallop. Electrocardiogram often shows nonspecific ST-segment and/or T-wave abnormalities. Chest X-ray or chest CT may show pulmonary edema, cardiomegaly, and pleural effusion. Echocardiographic demonstration of LVEF <45% and or (M)-type echocardiographic left ventricular shortening fraction <30%. Cardiac MRI is helpful in differentiating other cardiomyopathies such as ischemic cardiomyopathy, stress cardiomyopathy or infiltrative disease. Laboratory tests may show elevated BNP and NT-pro BNP, with possible slight elevation of cardiac troponin T (cTnT).

Diagnosing PPCM requires a high degree of suspicion, as its

early symptoms of heart failure are often difficult to distinguish from the discomforts of pregnancy, making diagnosis easily delayed. The diagnostic criteria are: (1) Heart failure occurring in the last month of pregnancy or within several months postpartum; (2) Exclusion of other causes of heart failure; (3) Left ventricular systolic dysfunction with LVEF <45%, with or without left ventricular enlargement [7]. This case meets all the above diagnostic criteria.

The exact pathogenesis of PPCM remains highly controversial. Currently, the most widely accepted model is the "two-hit" model [5]. This model includes genetic susceptibility caused by abnormal gene expression, vascular dysfunction, inflammatory responses, autoimmune reactions triggered by anti–cardiac tissue antibodies, and oxidative stress reactions caused by myocardial reperfusion injury. These multifactorial influences act on women in the late stages of pregnancy and early postpartum period who have underlying susceptibility, leading to the development of PPCM. As a pregnancy-related cardiovascular emergency, PPCM progresses rapidly and has a relatively high mortality rate during the acute phase. It remains the leading cause of death from heart failure in pregnant women [8]. Studies have reported that the morbidity and mortality rates of PPCM are approximately 7%-50% [9], depending on a variety of factors such as black females, multiple pregnancies, and LVEF <30%, which are associated with the worst maternal prognosis [10]. Mortality in PPCM varies significantly across regions and studies.

It is noteworthy that advanced maternal age, multifetal pregnancy, and pre-eclampsia have been identified as significant risk factors [11]. Therefore, paying more attention to this type of pregnancy high-risk group, increasing vigilance, and strengthening prevention are important ways to reduce the incidence of PPCM. The main diagnostic challenge lies in the fact that early symptoms of PPCM can easily be confused with normal pregnancy manifestations, often leading to delayed diagnosis. Echocardiographic LVEF <45% serves not only as one of the diagnostic criteria for PPCM but also predicts prognosis [12]. Current guidelines recommend NT-pro BNP testing and echocardiography for all suspected cases [13,14]. Both BNP and NT-pro BNP levels correlate with clinical outcomes; while they do not elevate during normal pregnancy, they typically show significant increases in PPCM [7]. In this case, NT-pro BNP was markedly elevated and subsequently decreased to normal levels as the condition improved. Electrocardiogram and chest X-ray findings are usually nonspecific, and a normal ECG does not exclude PPCM [15].

The treatment of peripartum cardiomyopathy has always been a challenge for clinicians due to the lack of evidence-based medical data. Currently, it mainly refers to the symptomatic treatment principles of acute heart failure caused by other etiologies, including inotropic support, diuresis, and vasodilation during the acute phase, and long-term treatment to improve myocardial remodeling during the chronic stable phase. Common pharmacological treatments include diuretics, beta-blockers, ACE inhibitors or ARBs, mineralocorticoid receptor antagonists, sacubitril/valsartan, hydralazine/nitrates, ivabradine, digoxin, heparin, warfarin, and direct oral anticoagulants (such as rivaroxaban, apixaban, edoxaban, and dabigatran). It is particularly important to always choose relatively safe drugs that have minimal impact on both the mother and the fetus during pregnancy and lactation. On the basis of ensuring no threat to fetal health, the patient's condition should be effectively controlled.

In addition, targeted drug therapy to improve the cure rate is a relatively new treatment approach. Adding the dopamine D2 receptor agonist bromocriptine to standard heart failure therapy can improve left ventricular function and prognosis in patients with PPCM. Studies have shown that after taking bromocriptine in combination with conventional heart failure medications for about 2 weeks, patients return to a stable level, and after taking it for 2 to 3 months, cardiac morphology and function are restored to normal level [16-19]. However, it should be noted that bromocriptine should be used in combination with prophylactic or therapeutic anticoagulant drugs [20]. Anticoagulation therapy is recommended for patients with systemic embolism or intracardiac thrombus. Patients with PPCM and significantly reduced LVEF should consider prophylactic anticoagulation therapy. The choice of anticoagulant drugs should be based on the stage of pregnancy and the patient's condition. Patients with atrial fibrillation should also be recommended anticoagulation therapy with low molecular weight heparin or warfarin according to the stage of pregnancy [20]. Anti-heart failure medication therapy should usually be maintained for at least 12 months. For those whose cardiac function has not returned to normal 6 to 12 months after onset, it is necessary to consider prolonging the medication period or even lifelong drug therapy [21]. After complete recovery of left ventricular function, heart failure drug therapy, including beta-blockers, ACEIs, and MRAs, should be continued for at least 6 months. If the medication is discontinued after the left ventricular ejection fraction returns to normal, it is necessary to closely monitor the changes in cardiac function by echocardiography. Timely diagnosis and early initiation of standard ant failure treatment can effectively relieve symptoms, promote cardiac function recovery, and reduce mortality.

The prognosis of PPCM varies greatly, ranging from complete recovery to persistent heart failure or death, which is influenced by the interplay of multiple factors. Therefore, it is crucial to conduct prognostic assessment based on risk factors to achieve individualized diagnosis and treatment. The research results of Fett et al. show that the shortest time for PPCM patients' LVEF to recover is 3 months, while the longest is 48 months, with 75% of patients recovering within 12 months of follow-up [22]. However, the traditional view holds that PPCM patients' LVEF usually returns to normal within 6 months, with little recovery after 6 months [2]. The patient in this case is a 29-year-old young woman with no underlying diseases. After excluding other cardiomyopathies through medical history, relevant auxiliary examinations, and echocardiography, a diagnosis of PPCM was made. Following treatment with the "quadruple therapy" (ARNI, beta-blocker, MRA, SGLT2i) for heart failure and symptomatic management, the patient's 1-month follow-up echocardiogram showed LVEF improved to 62%, allowing her to return to normal work and achieving early recovery. Compared with the traditional recovery rate, the treatment effect of this case is very good. The patient's self-monitored heart rate ranged 60-80 bpm. With digoxin withdrawal, guideline-directed quadruple therapy was continued under rigorous follow-up.

Literature reports that women with a history of PPCM have a risk of heart failure recurrence of over 20% in subsequent pregnancies, even if their LVEF has returned to normal. For patients with persistently reduced LVEF, the risk of worsening heart failure or even death in subsequent pregnancies is nearly 100%. Therefore, clinicians generally advise women with PPCM to avoid further pregnancies if possible. For those who do become pregnant again, close monitoring of cardiac function is neces-

sary, and termination of pregnancy should be considered in a timely manner if required. This primiparous patient remains at risk for future pregnancies under China's family planning policy. After comprehensive risk counseling, any subsequent pregnancy should be managed through coordinated cardiology and obstetric surveillance to mitigate maternal complications.

Conclusion

PPCM poses a serious threat to the life and health of parturient women. However, many issues regarding PPCM still need further research, such as the pathogenesis, the duration of medication after myocardial function recovery, and the risk prediction and management of subsequent pregnancies. At present, there is a lack of multicenter large-scale registry studies, which urgently need to be filled to provide more theoretical basis for the determination of pathogenesis, early diagnosis, standardized treatment, and improvement of prognosis.

Declarations

Author contributions: Literature search and manuscript preparation was done by T.T.C. Concept, design, manuscript editing, and final review of the manuscript was done by X.F.C. Ying Yang takes responsibility for the integrity of the work and acts as a guarantor.

Consent to participate: The author certifies that they have obtained all appropriate patient consent forms. The patient has given her consent for clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal the identity.

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