Peripartum Serial Echocardiographic Findings in a Patient with Life-threatening Peripartum Cardiomyopathy

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Abstract:
A 35-year-old woman was referred to our hospital for the management of acutely decompensated heart failure due to peripartum cardiomyopathy (PPCM). Generally, cardiac examinations are performed after the manifestation of heart failure in patients with PPCM. Thus, reports of serial cardiac examinations before the onset of PPCM are scarce. In this case, we were able to document the serial echocardiographic findings before the onset of life-threatening PPCM. We found that the left ventricular systolic function was preserved at 35 weeks of gestation but declined acutely after delivery at 38 weeks. Although speculative, these findings suggest that left ventricular dilation might precede the onset of PPCM.

Key words: peripartum cardiomyopathy, echocardiography, predictor

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Peripartum cardiomyopathy (PPCM) is characterized by systolic cardiac dysfunction and presents in the last month of pregnancy or within five months of delivery in women without pre-existing cardiac disease (1). A diagnosis of PPCM is confirmed by the exclusion of other underlying disorders and strict echocardiographic indications of left ventricular (LV) dysfunction, defined as an LV ejection fraction (LVEF) less than 45% (2). The reported incidence of PPCM varies globally and ranges from 1 in 1,421 to 1 in 9,861 deliveries (3). While half of the patients regain a normal LVEF, some patients require inotropes and mechanical circulatory support and may even require a heart transplant to survive (4). However, the etiology of PPCM remains unknown, and it is difficult to predict the onset of PPCM before the disease becomes apparent.

Case Report
A 35-year-old woman (gravida 1, para 1; uneventful pregnancy with history of first delivery at 32 years of age) was referred to our cardiac emergency department for the management of heart failure due to PPCM. The patient had a benign medical history before the current delivery of twin pregnancy. Her blood pressure had been within the normal range throughout the pregnancy, ranging from 111/64 to 129/75 mmHg in the absence of antihypertensive agents, and she had not developed proteinuria during the pregnancy. She was admitted to the obstetric hospital for the management of her pregnancy at 32 weeks of gestation. On admission, she was asymptomatic. However, chest radiography showed cardiac enlargement [cardiothoracic ratio (CTR), 54%] (Fig. 1A), and transthoracic echocardiography showed a slightly dilated cardiac chamber and preserved LV function [LV end-diastolic dimension (LVDd)/LV end-systolic...
The patient was referred to our tertiary medical center for further management.

On referral to our hospital, the patient’s pulse rate was 103 beats/minute, her blood pressure was 150/97 mmHg, respiratory rate was 29 breaths/minute, and room air oxygen saturation was 94%, with a New York Heart Association (NYHA) functional class III. Her BNP level was elevated at 2,696 pg/mL. Chest radiography showed further cardiac enlargement (CTR, 64%) and pulmonary congestion (Fig. 1E). On transthoracic echocardiography, LVDd/LVDs was 58/53 mm, and LV wall motion exhibited severely reduced contraction, with an LVEF of 24% (Fig. 1F) and only mild mitral regurgitation.

Emergency right heart catheterization indicated progressive heart failure with a high pulmonary capillary wedge pressure (25 mmHg), high pulmonary arterial pressure (40/23/30 mmHg), normal right atrial pressure (2 mmHg), and normal cardiac index (4.6 L/min/m²). We then detected an increased heart rate (from 95 to 138 beats/minute) and a gradual decrease in her cardiac index (down to 2.8 L/min/m²). Considering her low stroke volume index (20 mL/m²), we initially administered inotropes, but her heart failure continued to worsen. Thus, intra-aortic balloon pumping (IABP) was used for circulatory support. Cabergoline, which is a potent dopamine receptor agonist, was

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**Table:**

|                | At 32 weeks of gestation | At 35 weeks of gestation | Immediately after delivery (38 weeks) | 6 months after delivery |
|----------------|--------------------------|--------------------------|--------------------------------------|------------------------|
| **LVDd**       | 51 mm                    | 54 mm                    | 58 mm                                | 44 mm                  |
| **LVDs**       | 34 mm                    | 36 mm                    | 53 mm                                | 29 mm                  |
| **LVEF**       | 54%                      | 62%                      | 24%                                  | 53%                    |
| **BNP**        | 138 pg/mL                | 154 pg/mL                | 2696 pg/mL                           | 9 pg/mL                |

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**Figure 1.** Serial chest radiograph and transthoracic echocardiogram from 32 weeks of gestation to 6 months after delivery. (A) Chest radiograph at 32 weeks of gestation. (B) Transthoracic echocardiogram at 32 weeks of gestation. (C) Chest radiograph at 35 weeks of gestation. (D) Transthoracic echocardiogram at 35 weeks of gestation. (E) Chest radiograph at referral to our hospital (38 weeks of gestation). (F) Transthoracic echocardiogram at referral to our hospital (38 weeks of gestation). (G) Chest radiograph 6 months after delivery. (H) Transthoracic echocardiogram 6 months after delivery. BNP: B-type natriuretic peptide, LVDd: left ventricular end-diastolic dimension, LVDs: left ventricular end-systolic dimension, LVEF: left ventricular ejection fraction.
In addition, some investigators have suggested acute myocardial angiogenic imbalance (7), and genetic susceptibility (2). In a multivariate analysis, the risk factors for PPCM are elevated plasma BNP level (154 pg/mL at 35 weeks) and an elevated LVEF (62%) at 35 weeks of gestation but declined acutely (LVEF, 24%) after delivery at 38 weeks. Serial echocardiography revealed that the deleterious effects on the systolic function occurred within 3 weeks. Second, LV dilation (LVDd/LVDs, 51/34 mm at 32 weeks; 54/36 mm at 35 weeks) and an elevated BNP level (154 pg/mL at 35 weeks) might precede the onset of PPCM.

**Discussion**

PPCM is a life-threatening disease, but its precise etiology and progression remain largely unknown. Cardiac examinations in patients with PPCM are usually performed after the manifestation of heart failure. Thus, reports of serial cardiac examinations before the onset of PPCM are scarce. In our case, we were able to document serial cardiac examinations before the onset of life-threatening PPCM. The major findings in our report were as follows: First, the LV systolic function was preserved (LVEF, 62%) at 35 weeks of gestation but declined acutely (LVEF, 24%) after delivery at 38 weeks. Second, LV dilation (LVDd/LVDs, 51/34 mm at 32 weeks; 54/36 mm at 35 weeks) and an elevated BNP level (154 pg/mL at 35 weeks) might precede the onset of PPCM.

The causes of PPCM are reportedly multifactorial, including inflammatory cytokines (5), cleavage of prolactin to an angiostatic N-terminal 16 kDa prolactin fragment (6), cardiac angiogenic imbalance (7), and genetic susceptibility (2). In addition, some investigators have suggested acute myocarditis as a possible cause of PPCM based on endomyocar-

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**Figure 2.** Coronary angiography, and cardiac magnetic resonance imaging. (A) Right coronary angiography showed a normal right coronary artery. (B) Left coronary angiography showed a normal left coronary artery. (C) The myocardium was not enhanced on late gadolinium-enhanced cardiac magnetic resonance imaging. (D) The myocardium had no high-signal-intensity areas on T2-weighted cardiac magnetic resonance imaging. LAD: left anterior descending coronary artery, LCX: left circumflex artery, LV: left ventricle, RCA: right coronary artery, RV: right ventricle.
The literature regarding the LV size in twin pregnancy is lacking. However, we speculate that the patient’s LV size of 54 mm might be slightly dilated, as Japanese women are generally relatively lean and small, based on the findings of a report on the cardiac function in twin pregnancy from Western countries (15).

LV dilation and BNP elevation beyond the normal range in pregnancy (although the twin pregnancy might have influenced these changes) preceded the decline in the LVEF in our patient. Our case suggests that LV dilation and BNP elevation may precede heart failure decompensation and might be predictors for the development of PPCM. Further studies are required to test this hypothesis.

**Conclusion**

Our case demonstrated serial cardiac changes before the onset of PPCM. We found that the LVEF declined acutely after 35 weeks of gestation, and LV dilation might have preceded the decline in the LVEF, suggesting that LV dilatation...
might be a predictor for the development of PPCM. Further studies are warranted to investigate the underlying mechanism, natural course, and predictors of PPCM.

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The authors state that they have no Conflict of Interest (COI).

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