Peripartum Mid-Ventricular-Type Takotsubo Cardiomyopathy
After Cesarean Delivery

The Role of Early and Repeat Cardiac Magnetic Resonance Imaging

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Summary

There are several causes of heart failure during pregnancy and the peripartum period, which include peripartum cardiomyopathy, Takotsubo cardiomyopathy or stress cardiomyopathy, exacerbation of a preexisting cardiomyopathy, and acute myocarditis. It is important to determine the cause of the heart failure as the medical treatment may be different based on the diagnosis. However, it has been sometimes challenging to diagnose the cause because of the limited diagnostic tools, especially in pregnant women. Cardiac MRI can characterize myocardial injury and can be used to track the changes in myocardial tissue. We herein report a 35-year-old woman diagnosed with peripartum mid-ventricular-type Takotsubo cardiomyopathy, who was referred to our hospital due to worsening dyspnea the day after cesarean delivery. On admission, electrocardiography showed sinus tachycardia and poor progression of R waves in the precordial leads. Bedside echocardiography revealed severe hypokinesis in the mid- and apical left ventricle (LV) with a LV ejection fraction of 20%. Cardiac catheterization showed normal coronary arteries, and myocardial biopsy revealed contraction band necrosis. On acute phase (Day 4), cardiac MRI showed prolonged native T1 and T2, and severe hypokinesis and decreased regional longitudinal peak strain in the mid-anterior LV wall. During the 1st week, precordial ST fluctuation was observed, and LV wall motion had gradually recovered. Repeat cardiac MRI revealed normalized LV wall motion and shortened values for global native T1 and T2. Thus, she was diagnosed with peripartum Takotsubo cardiomyopathy. Serial cardiac MRI may be able to differentiate Takotsubo cardiomyopathy during pregnancy and the peripartum period from other preexisting cardiomyopathies.

Key words: Myocardial biopsy

There are several causes of heart failure during pregnancy and the peripartum period, including peripartum cardiomyopathy, Takotsubo cardiomyopathy or stress cardiomyopathy, exacerbation of a preexisting cardiomyopathy, and acute myocarditis.1,2) Takotsubo cardiomyopathy is often characterized with LV apical ballooning and acute ST segment elevation followed by giant negative T wave in multiple leads. There are various types of variants of Takotsubo cardiomyopathy, such as midventricular, basal, and focal types.3) T2 mapping by cardiac MRI is reported to be useful in identifying patients with myocardial inflammation such as Takotsubo cardiomyopathy and acute myocarditis.4)

Case Report

A 35-year-old gravida 2, para 1 woman with a history of postpartum depression was hospitalized in an obstetrics and gynecology department due to threatened premature labor at 33 weeks of gestation. No other peripartum abnormalities were observed. She was referred to our hospital because of rapidly progressing dyspnea and hypoxemia the day after cesarean delivery of twins at 37 weeks. On admission, she was afebrile, 154 cm in height, and 46.5 kg in weight. Her blood pressure was 95/66 mmHg, pulse was 136/minute, and respiratory rate was 35/minute. Oxygen saturation was 90% with an oxygen inflow of 10 L/min. Her blood pressure was 95/66 mmHg, pulse was 136/minute, and respiratory rate was 35/minute. Oxygen saturation was 90% with an oxygen inflow of 10 L/min. Physical examination revealed gallop rhythm but no jugular venous distention, cardiac murmur, severe abdominal guarding, or leg edema. Chest X-rays demonstrated pulmonary congestion without cardiomegaly (Figure 1A). Sinus tachycardia and poor progression of R waves in the precordial leads were noted on electrocardiography on Day 0 (Figure 1B). Laboratory findings revealed increases in white blood cell count (17,500/μL), aspartate transaminase (41 U/L), creatine kinase (156 U/L),...
brain natriuretic peptide (647.6 pg/mL), and troponin I (3,051.4 pg/mL), but no anemia or renal insufficiency. Bedside emergent echocardiography revealed severe hypokinesis in the mid- and apical left ventricle (LV), and mild hypokinesis in the basal segments, with an LV ejection fraction of 20%. No LV outflow tract obstruction or thickening of the LV wall was observed. Intravenous furosemide (20 mg) was administered intravenously. Since we believed that she may have had acute heart failure due to low cardiac output syndrome, a low dose of dobutamine and noninvasive positive pressure ventilation were initiated, which alleviated her symptoms and reduced the heart rate to 90 bpm. Cardiac catheterization showed normal coronary arteries, a cardiac index of 2.39 L/minute/m², and a normal pulmonary capillary wedge pressure of 7 mmHg under dobutamine support. Left ventriculography was not performed. Myocardial biopsy specimens from the mid-portion of the right ventricular septum revealed interstitial widening and contraction band necrosis but no abnormal inflammatory infiltrate defined as ≥ 14 leukocytes/mm² (Figure 1C). On Day 1, electrocardiography demonstrated negative T waves in precordial leads or precordial ST fluctuation with no giant negative T waves was noted (Figure 1B). Cardiac MRI was performed on Day 4. Severe hypokinesis and decreased regional longitudinal peak strain in the mid-anterior LV wall, and preserved wall motion of the basal- and apical LV walls were observed on cine MR images with feature tracking analysis. No LV hypertrophy was observed. T1/ T2 mapping revealed prolonged global native T1 (1,552 ms; normal, 1,294 ± 39 ms), markedly high T2 (81.3 ms; normal, 45 ± 5 ms), and increased global ECV (41%)(normal, 26.1 ± 1.4%, Figure 3A-C). Diffuse faint enhancement was observed in the LV wall on late gadolinium enhancement (LGE) MRI (Figure 3D). Thus, acute coronary syndrome due to coronary atherosclerosis was ruled out. Dobutamine was successfully tapered. The follow-up echocardiography on Day 11 showed recovered LV ejection fraction with mild hypokinesis in the mid to apical segments. Cardiac MRI on Day 22 showed normalized LV wall motion by colored strain analysis, shortened global native T1 (1,398 versus 1,552 ms) and T2 (55.0 versus 81.3 ms), and decreased global ECV (35.4 versus 41.0%) (Figure 3E-G). However, the native T1 (1,415 ms) and ECV (36.7%) in the mid-lateral LV were larger than the global native T1 and ECV, suggesting less improvement of the altered myocardial tissue. The faint enhancement was diminished on the LGE MR images (Figure 3H). She was discharged home with ACE-I and beta-blockers on Day 23. Repeat electrocardiography 1 month and 5 months after the onset was normal with no ST-T changes. Three months after the onset, she had no symptoms of heart failure. Although the native T1 and T2 values were normalized, the global ECV (32.7%) remained slightly elevated on the repeat cardiac MRI at the 3rd month.

Discussion

In patients with heart failure during pregnancy and the peripartum period, differential diagnoses of heart failure include peripartum cardiomyopathy, Takotsubo or stress cardiomyopathy, exacerbation of the pre-existing dilated familial cardiomyopathy, and myocarditis.1,2) There are variants of Takotsubo cardiomyopathy such as the midventricular type.3) Our patient was diagnosed with mid-ventricular-type Takotsubo cardiomyopathy based on her history of cesarean delivery, postpartum depression, interstitial widening and contraction band necrosis, precordial ST fluctuation, markedly high T2 on a cardiac MRI during acute phase, and early recovery of LV wall motion abnormality. Although we confirmed the improvement of mid-ventricular wall motion by colored strain analysis with feature tracking software, the altered myocardial tissue had not completely recovered by Day 22.

Only a few reports have evaluated the clinical courses and recovery of LV wall motion in patients with Takotsubo cardiomyopathy and peripartum cardiomyopathy. For example, Yang, et al observed earlier complete recovery of the LV ejection fraction within a month in patients with Takotsubo cardiomyopathy compared to those with peripartum cardiomyopathy.7) Aikawa, et al. reported that native T1 and ECV decreased after 3 months from onset when compared to those during acute phase, but had never normalized at the 3rd month in Takotsubo cardio-

Figure 1. A: Chest X-ray showing pulmonary congestion on Day 0. B: ECG demonstrating negative T wave in precordial leads on Day 1 and precordial ST fluctuation from Days 2 to 7. C: Hematoxylin-eosin staining showing significant interstitial edema and contraction band necrosis, but no inflammatory cell infiltration.
Figure 2. Clinical course and data during hospitalization. CTR indicates cardiothoracic ratio; SBP, systolic blood pressure; CRP, C-reactive protein; SpO2, oxygen saturation; BiPAP, bilevel positive airway pressure; NC, nasal cannula; and BNP, brain natriuretic peptide.

Figure 3. Cardiac MRI on Day 4 (A-D) and on Day 22 (E-H). Long-axis 4-chamber cine image at end-systole analyzed by colored strain analysis with feature tracking software (Circle CVI42<sup>®</sup>) showing depressed mid-anterior LV systolic function (A), global and focal native T1 mapping (B), extracellular volume fraction (ECV) (C), and late gadolinium enhancement (LGE) (D) on Day 4. Repeated cine image at end-systole analyzed by colored strain analysis showing normalized LV systolic function (E), global and focal native T1 mapping (F), ECV (G), and LGE (H) on Day 22.

myopathy. Consistent with their report, the global ECV value in our patient remained slightly elevated in the 3rd month. To the best of our knowledge, this is the first report demonstrating that significant improvement of native T1, T2, and ECV could occur within a few weeks from the onset of Takotsubo cardiomyopathy. Contrary to our patient with a very high T2 value, those with peripartum cardiomyopathy may not have a high T2 value in the
acute phase. In addition, patients with peripartum cardiomyopathy appeared to have unchanged ECV and T2 during follow-up although they had a lower global native T1 value at follow-up. Patients with acute myocarditis could have high native T1, T2, and ECV. Therefore, early cardiac MRI by itself is not enough to differentiate Takotsubo cardiomyopathy from acute myocarditis by itself. However, LV hypertrophy was not observed in our patient. In addition, the myocardial specimens obtained from the mid portion of the right ventricular septum did not show inflammatory infiltrates. Although it is unclear whether the pathological findings in the affected LV wall were completely the same as those in the RV septum, we believe that the pathological findings from the right ventricle could exclude acute myocarditis.

Early cardiac MRI rules out acute coronary syndrome caused by coronary atherosclerosis when the regional wall motion abnormalities of the myocardium or late gadolinium enhancement of the LV extends beyond a single epicardial coronary distribution. In our patient, the findings of early cardiac MRI coupled with coronary angiography could rule out myocardial infarction with/without a non-obstructive coronary artery.

Feature tracking cardiac MRI could evaluate global and regional myocardial function by measuring myocardial strain. We believe that when combined with T1/T2 mapping and ECV, serial cardiac MRI at early phase and 2-3 weeks after onset might have the potential to differentiate Takotsubo cardiomyopathy during the peripartum period from other preexisting cardiomyopathies and peripartum cardiomyopathy.

Disclosure

Conflicts of interest: None.

References