Myocardial Edema in Takotsubo Syndrome
— Serial Cardiovascular Magnetic Resonance Imaging of the Natural Course —

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Figure. Cardiovascular magnetic resonance imaging. (A,B) Obvious myocardial edema is not seen in the acute phase. Apical wall thickness was 5.6mm and the signal intensity (SI) ratio to the skeletal muscle was 2.7. (C,D) Thickened apical wall with circumferential myocardial edema in the subacute phase (arrows). Apical wall thickness, 8.9mm; SI ratio, 3.6. (E,F) Complete resolution of apical wall thickening and myocardial edema in the chronic phase. Apical wall thickness, 5.0mm; SI ratio, 2.3.
A 67-year-old woman lost consciousness after sudden chest pain without obvious stressful trigger. Consciousness spontaneously recovered in the emergency department. Electrocardiogram showed significant ST-elevation in leads II, III, aVF, and V4–6. Coronary angiography indicated no obstructive coronary artery disease, and left ventriculogram showed typical apical ballooning. Troponin I was 5.66 ng/mL on admission, and peak creatine kinase was 695 U/L. On day 2, initial cardiovascular magnetic resonance imaging (CMR) showed apical ballooning without obvious myocardial edema on T2-weighted imaging, except for extreme high signal intensity of the endocardium due to slow blood flow in the apical ballooning area (Figure A,B; Movie S1). One week later, second CMR showed thickened apical wall with apparent myocardial edema (Figure C,D; arrows), even though the wall motion was completely recovered (Movie S2). Three months later, the final CMR showed no apical wall thickening or myocardial edema (Figure E,F).

This is the first study to show the pathological and functional change of myocardium in takotsubo syndrome (TTS) on serial CMR from the acute phase to the chronic phase. Myocardial edema is one of the typical features in TTS, but there is little information on the relationship between myocardial edema and wall motion abnormality. In the present case, obvious myocardial edema was not seen in the acute phase regardless of apical ballooning. In the subacute phase, myocardial edema with no wall motion abnormality was observed. The definitive etiology of TTS is not still established, although several hypotheses, such as catecholamine-induced myocardial stunning, multivessel coronary artery spasm, and microcirculatory impairment, have been postulated. In addition, myocardial edema is recognized as one of the possible pathogenic cause of TTS. The present findings, however, suggest that myocardial edema is not the primary pathogenesis of TTS but a secondary phenomenon. Recently, Shin et al reported that worse outcome was observed in patients with than without transient apical wall thickening on echocardiography during the recovery process of TTS. The present case indicates that transient apical wall thickening is due to delayed myocardial edema and it may have an impact on the outcome of TTS.

Disclosures
The authors declare no conflict of interest.

References

Supplementary Files
Supplementary File 1
Movie S1. Cine cardiovascular magnetic resonance imaging in the acute phase.

Supplementary File 2
Movie S2. Cine cardiovascular magnetic resonance imaging in the subacute phase.

Please find supplementary file(s); http://dx.doi.org/10.1253/circj.CJ-17-0065