Remote Myocardial Remodeling of Biventricular Hypertrophic Cardiomyopathy After Alcohol Septal Ablation on Cardiac Magnetic Resonance

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A 44-year-old woman with known hypertrophic cardiomyopathy (HCM), presented with dyspnea and effort palpitation (New York Heart Association [NYHA] functional class 4). Physical examination indicated systolic ejection murmur. Creatine kinase MB mass (CKMB; 5.66 ng/ml), cardiac troponin T (cTnT; 50.8ng/L), and pro-B-type natriuretic peptide (pro-BNP; 4,142 pg/ml) were elevated compared with the reference ranges. Echocardiogram showed biatrial enlargement, biventricular hypertrophy, left ventricular outflow tract (LVOT) obstruction with peak gradient of 94 mmHg, and systolic anterior motion. Cardiac magnetic resonance (CMR) was done with a 3.0-T magnetic resonance scanner (Magetom Trio, Siemens Medical Systems, Erlangen, Germany). Cine images showed biventricular hypertrophy (Figures 1A-1B-1); and maximum left ventricular (LV) and right ventricular (RV) end-diastolic wall thickness were 34.2 mm and 20.7 mm, respectively. After a bolus of Gd-DTPA (Magnevist, Bayer Health Care Pharmaceuticals, Germany), first-pass sequence

Figure 1. Short-axis, 4-chamber and 3-chamber view cine images. (A-1,2) Septum, left ventricular (LV) anterior wall, and right ventricular (RV) free wall thinned after alcohol septal ablation (ASA), while LV lateral wall thickened. (B-1,2) Four-chamber view showed that the RV wall was hypertrophic before ASA, and thinned after ASA. (C-1,2) Left ventricular outflow tract slightly widened after ASA. Long arrows, septum, and LV anterior, lateral, and inferior wall. Short arrows, RV free wall.
indicated septum perfusion defect (Figure 2A-1). Ten minutes after use of the same contrast agent, late gadolinium enhancement (LGE) showed patchy biventricular hyperenhancement (Figures 2B-1–D-1). Based on LVOT gradient and NYHA functional class, alcohol septal ablation (ASA) targeting the septal perforator artery was done using 1.8 ml of ethanol. Five months after ASA, dyspnea was relieved, palpitation had disappeared and mild edema of the lower extremities occurred. CKMB (4.94 ng/ml), cTnT (46.1 ng/L), and pro-BNP (2,055 pg/ml) had decreased. Right atrial (RA) enlargement disappeared and LVOT gradient (44 mmHg) decreased on echocardiogram. CMR showed multiparameter alterations. Septal wall thickness at infarction site reduced from 32.3 mm to 22 mm. LV anterior and RV free wall thickness at the mid-LV level decreased from 18.6 mm and 11.5 mm, respectively, to 16.9 mm and 2 mm, respectively, and LV inferior wall thickness changed from 18.6 mm and 11.5 mm, respectively, to 16.9 mm and 2 mm, respectively, and LV inferior wall thickness changed from 8.3 mm to 8.5 mm. LV lateral thickness, however, increased from 8.9 mm to 15.8 mm (Figures 1A-1, 1C-1). LV mass reduced from 310.3 g to 235.6 g. LVOT diameter slightly widened (19.4 mm vs. 21.5 mm; Figures 1C-1, 2A-1). LV end-diastolic volume (LVEDV) changed from 144.7 ml to 147.7 ml, and LV end-systolic volume (LVESV, 48.8 ml vs. 71.8 ml), RV end-diastolic volume (106.1 ml vs. 126.8 ml), and RV end-systolic volume (38.7 ml vs. 56.2 ml) increased. Consequently, LV end-diastolic volume (106.1 ml vs. 126.8 ml), and RV end-systolic volume (38.7 ml vs. 56.2 ml) increased. LV lateral thickness in the present patient included reductions of LV anterior and RV free wall, and increases in the LV lateral wall, indicating heterogeneity in regional myocardial remodeling. It is interesting to find evident thinning in the RV free wall, suggesting that LV and RV remodeling might be different. It is necessary to investigate biventricular remodeling in biventricular HCM after ASA using CMR. LV remodeling occurred early and progressed on midterm follow-up, and the LA dimension decrease probably reflected the effect of ASA on improving diastolic function. 10,11 Current CMR findings showed reductions of LA dimension and LV mass, and increases of LVEDV and LVESV. Limited data have shown that ASA decreases RV workload, as reflected by reduced pulmonary artery pressure, increased RVEF, and decreased RV oxidative metabolism.12,13 The present biventricular HCM patient had decreased RA dimension, reflecting reduced RV preload, probably indicating RV remodeling on improving diastolic function. LGE CMR enables assessment of infarction size and location.10,11 In the present case, visualization of LGE in the targeted area, reflected the infarction effect of ethanol. CMR may be an excellent modality to follow up biventricular HCM after ASA.

HCM is a genetic disease with heterogeneous clinical symptoms.2–4 CMR allows visualization of abnormalities in HCM patients.5–7 One case report showed biventricular hypertrophy in HCM on CMR.5 CMR also enables identification of cardiac morphologic and functional alterations in HCM patients after ASA.1,9 CMR for biventricular HCM after ASA, however, has not previously been reported. The present patient with biventricular hypertrophy and scarring underwent CMR before and after ASA. Septal thickness and LV mass reduced after ASA, in line with previous studies.10,11 Some investigators reported a reduction in remote wall thickness.10 Changes in remote wall thickness in the present patient included reductions of LV anterior and RV free wall, and increases in the LV lateral wall, indicating heterogeneity in regional myocardial remodeling. It is interesting to find evident thinning in the RV free wall, suggesting that LV and RV remodeling might be different. It is necessary to investigate biventricular remodeling in biventricular HCM after ASA using CMR. LV remodeling occurred early and progressed on midterm follow-up, and the LA dimension decrease probably reflected the effect of ASA on improving diastolic function.10,11 Current CMR findings showed reductions of LA dimension and LVEF, and increases of LVEDV and LVESV. Limited data have shown that ASA decreases RV workload, as reflected by reduced pulmonary artery pressure, increased RVEF, and decreased RV oxidative metabolism.12,13 The present biventricular HCM patient had decreased RA dimension, reflecting reduced RV preload, probably indicating RV remodeling on improving diastolic function. LGE CMR enables assessment of infarction size and location.10,11 In the present case, visualization of LGE in the targeted area, reflected the infarction effect of ethanol. CMR may be an excellent modality to follow up biventricular HCM after ASA.

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