Cardiac MR Imaging of Apical Left Ventricular Aneurysm and Thinning Associated with Hypertrophic Cardiomyopathy

Y. Amano[1], M. Takayama[2], and S. Kumita[1]

[1]Radiology, Nippon Medical School, Tokyo, Tokyo, Japan, [2]Cardiology, Nippon Medical School, Tokyo, Tokyo, Japan

Introduction

Hypertrophic cardiomyopathy (HCM) is defined as a myocardial disorder that is characterized by hypertrophy of a nondilated left ventricular (LV) cavity in the absence of underlying diseases. Apical aneurysm is a rare complication of HCM, which induces the clinical symptoms, ECG abnormalities, and mural thrombosis. Cine and delayed contrast-enhanced MR imaging techniques have been reported to be useful for thin-walled akinetic LV aneurysm, the myocardium of which is replaced by collagen tissue. Due to the high spatial resolution and unlimited view, MR imaging can detect apical LV aneurysm as well as apical LV thinning, which is less than 5 mm, but not aneurismal. This apical LV thinning may be similar to the concealed apical asynergy that is associated with reduced myocardial perfusion. The cardiac MR images of apical LV thinning associated with HCM, however, have not been described so far. The purpose of this study was to delineate the cardiac MR imaging findings of apical LV aneurysm and thinning associated with HCM, and to clarify the relationship with other morphological findings and cardiac functional parameters.

Methods

Thirty-three consecutive patients with HCM were enrolled in this study. Cardiac MR imaging examinations were performed using 1.5-T MR imagers (Achieva and Signa CVi). Cine MR imaging in the long- and short-axis views was acquired using steady-state free precession imaging (TR, 2.8 ms; TE, 1.4 ms; flip angle, 60). Delayed contrast-enhanced MR imaging in the long- and short-axis planes (TR, 9.3 ms; TE, 4.6 ms; flip angle, 10) was obtained using inversion-recovery gradient-echo imaging.

The differences in the maximum myocardial wall thickness, LV ejection fraction, and LV myocardial mass among the three patient groups (i.e., apical aneurysm, apical thinning, hypertrophied apical myocardium) were statistically assessed. The differences in the patterns of HCM (asymmetrical septal hypertrophy [ASH] or non-ASH), myocardial hyperenhancement of apical region, and presence of midventricular obstruction (MVO) among the three patient groups were also assessed. The MVO in cine MR images was defined as an obstruction in the midventricular level at systole with hypointense turbulence.

Results

Two and 15 of the 33 patients with HCM had apical LV aneurysm and thinning, respectively. The remaining 16 patients had hypertrophied apical myocardial wall. Apical LV aneurysm showed circumferential, transmural hyperenhancement of the apical myocardium in these patients. Apical LV thinning was observed in the inferior wall in 14 patients, in the lateral wall in six, in the anterior wall in six, and in the septal wall in five of the 15 patients**.** Transmural hyperenhancement of the thin apical myocardium was observed in one of the 15 patients. The LV ejection fraction was lower in patients with apical LV aneurysm than in those with apical LV thinning ($P = 0.014$) and those with hypertrophied apical myocardium ($P = 0.002$). ASH was associated with the apical LV thinning more frequently than hypertrophied apical myocardium ($P = 0.0014$). There were no significant differences in the maximum myocardial wall thickness, LV myocardial mass, and presence of MVO among the three patient groups ($P > 0.28$).

Discussion

This study showed circumferential myocardial hyperenhancement of the apical LV aneurysm on delayed contrast-enhanced MR images, which may reflect collagen tissue in the region. This study indicated that akinesis and serious myocardial damage of the apical aneurysm might lead to lower values for the LV ejection fraction. Unexpectedly, apical LV thinning was a common MR imaging finding in HCM, which was frequently associated with ASH. The apical LV thinning commonly occurred in the inferior wall that protruded downward. The myocardial architecture and position of the apical inferior segment can result in the sensitivity of this segment to the increased intracavity pressure induced by ASH. However, an apical LV thinning is not necessarily a preceding condition of the apical LV aneurysm, because of the lack in myocardial hyperenhancement.

Neither apical LV aneurysm not thinning was related to maximum myocardial wall thickness, LV myocardial mass, and presence of MVO. These results suggested that the apical LV aneurysm and thinning might be associated with apical myocardial pathologies and patterns of hypertrophy rather than the degree of myocardial hypertrophy.

In conclusion, our findings suggest that apical LV aneurysm and thinning in HCM may be clearly visualized using cardiac MR imaging. Apical LV aneurysm resulting from serious myocardial fibrosis might lead to a lower LV ejection fraction. Apical LV thinning was a common complication of HCM with ASH, which showed no myocardial hyperenhancement in the majority of patients.

References

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