in age, left ventricular end diastolic dimension (LVED), maximum thickness, left atrial dimension, maximum dimension of LVAA and time of follow up. In nonviable group, mural thrombus presented in 3 patients and pericardiac effusion occurred in 2 patients. Transaortic septal myectomy and septal alcohol ablation were performed in 3 patients, respectively. In the 1.7 ± 0.9 years follow up, no adverse events occurred in viable group. In particular, a case of complete transition from viable to nonviable LVAA was recorded. In nonviable group (1.7 ± 0.7 years follow up), emergence or progression of congestive heart failure occurred in 5 patients, ventricular tachycardia in 3 patients.

Conclusions Viable LVAAAs existed in a small proportion of HCM patients, and might develop into nonviable LVAAAs. This finding warranted further investigations to reassess the mechanism, treatment considerations and prognosis of the disease.

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VIABLE APICAL ANEURYSMS OF LEFT VENTRICLE IN HYPERTROPHIC CARDIOMYOPATHY

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Background The formation of left ventricular apical aneurysm (LVAA) is a distinctive subset in hypertrophic cardiomyopathy (HCM), however, it is still unknown about myocardial viability of LVAAAs in these patients.

Objectives The study was carried out to assess the myocardial viability of LVAAAs in HCM patients.

Methods Of 510 HCM patients, 21 (4.1%, 17 M/4 F) were identified as HCM with LVAAAs. Coronary artery disease was ruled out by selective coronary angiography or coronary computed tomography angiography (CTA). Myocardial viability of LVAAAs was assessed by single photon emission computed tomography (SPECT), positron emission tomography (PET) and delayed-enhancement magnetic resonance imaging.

Results Viable LVAAAs were identified in 8 HCM patients and nonviable LVAAAs presented in the other 13 patients. Left ventricular obstruction presented in 15 patients, including 3 patients with mid-ventricular obstruction. In all LVAAAs, the aneurysmal wall was thin (<5.5 mm) and the maximum thickness of left ventricular segments was 20.5 ± 4.6 mm. The left ventricular ejection fraction (LVEF) was higher in viable group than that in nonviable group, (69.1 ± 6.8)% vs (58.4 ± 11.9)%, $p=0.03$. In the two groups, there were no significant differences