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Non Invasive Imaging

NOT MYOCARDIAL PERFUSION STATUS ITSELF, BUT ITS FINAL HEMODYNAMIC CONSEQUENCE IS RELATED TO MYOCARDIAL SYSTOLIC FUNCTION IN HYPERTROPHIC CARDIOMYOPATHY: INTEGRATED APPROACH WITH ECHOCARDIOGRAPHY AND CARDIAC MAGNETIC RESONANCE IMAGING

Poster Contributions
Hall C
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Background: Microvascular dysfunction is considered to play a crucial role in decreased coronary flow reserve (CFR), and to be related to myocardial functional changes in patients with hypertrophic cardiomyopathy (HCM) having normal coronary arteries. In this study, we aimed to determine in HCM 1) myocardial perfusion abnormality pattern on cardiac magnetic resonance (CMR) and 2) the relation between myocardial perfusion status on CMR and myocardial function assessed by speckle tracking-determined myocardial strain.

Methods: 54 HCM patients who underwent both echocardiography and CMR were consecutively enrolled. With speckle tracking technique, LV systolic function was assessed by LV global longitudinal (LV-GLS), circumferential (LV-GCS) and radial (LV-GRS) strains. CFR was assessed by Doppler echocardiography in all HCM patients and age-matched healthy controls (n=27). CMR was performed in all subjects to calculate LV mass index (LVMI) and to assess myocardial perfusion abnormality under pharmacologic stress.

Results: CFR was significantly lower in HCM than in controls (2.25±0.89 vs. 3.03±0.39, p<0.05). While LV-GLS was associated with decreased CFR in HCM (r2=0.175, p=0.037), LV-GCS (p=0.062) and LV-GRS (p=0.985) were not. Myocardial perfusion abnormality on CMR was present in 28 patients (51.9%), all of whom showed “subendocardial” perfusion defect observed only under adenosine stress. LV-GLS, LV-GCS and LV-GRS were not associated with the presence or absence of myocardial perfusion abnormality on CMR, but CFR was significantly lower in patients with myocardial perfusion abnormality (1.96±0.57 vs. 2.35±0.87, p=0.038). Mean LVMI on CMR was 122.9±38.5g/m2, which was comparable between in patients with and without perfusion abnormality (124.3±29.1 vs. 121.2±41.2, p=0.546).

Conclusions: Subendocardial, not transmural, perfusion defect is a consistent abnormal perfusion pattern in HCM. Although CFR was related to LV-GLS, any LV strain was not different according to myocardial perfusion status. Thus, not a decrease in myocardial perfusion itself under stress, but its hemodynamic consequence reflected by CFR exerts a significant influence on myocardial function.