Layer-specific strain and the degree of left ventricular thickness in patients with hypertrophic cardiomyopathy

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Background: Left ventricular (LV) wall thickness is an important parameter for the diagnosis of hypertrophic cardiomyopathy (HCM) and is also associated with long-term clinical outcome in HCM patients. However, conventional tools have failed to analyze the mechanisms of structural and functional abnormalities that occur at the cellular level in hypertrophied myocardial tissue. Recently, technological progression of 2D-speckle tracking echocardiography (2D-STE) has enabled the estimation of layer-specific strain (LSS), such as epicardial, mid-myocardial, and endocardial longitudinal strain, respectively. LSS may have the potential to elucidate the detailed mechanisms of myocardial dysfunction.

Purpose: The aim of this study was (i) to clarify the detailed mechanisms of structural and functional abnormalities of myocardial tissue in HCM using LSS (ii) to investigate the diagnostic accuracy of LSS for HCM.

Methods: Forty-one patients with HCM and preserved LV ejection fraction (LVEF) (66% male, 52±18 years, LVEF 62.9±3.7%) and 41 controls matched for age and sex (66% male, 52±20 years, LVEF 63.5±8.2%) underwent 2D-STE (GE-Healthcare, Vivid-E9). Quantitative strain values of epicardial, mid-myocardial, and endocardial layers were measured.

Results: LV wall thickness including interventricular septum thickness (HCM vs. Controls; 18.9±5.0 vs. 9.1±1.8, $p<0.001$), posterior wall thickness (11.5±2.5 vs. 8.8±1.9, $p<0.001$), and maximum wall thickness (20.1±4.3 vs. 9.4±0.4, $p<0.001$) were significantly lower in HCM than in Controls. Absolute values of LSS for all layers were lower in HCM than in Controls (HCM vs. Controls; epicardial; −13.1±3.3 vs. −19.5±1.6, $p<0.001$; mid-myocardial; −15.8±3.3 vs. −21.4±1.7, $p<0.001$; endocardial; −18.9±3.9 vs. −23.6±1.9, $p<0.001$). End/Epi ratio was higher in HCM than in Controls (HCM vs. Controls; 1.5±0.2 vs. 1.2±0.0, $p<0.001$). Next, we investigated the echocardiographic parameters that correlated with LV maximal wall thickness (MWT). End/Epi ratio was an independent predictor of LV MWT (β=0.96, $p<0.001$). Receiver operating characteristic analysis revealed that a higher End/Epi ratio ($\geq$1.3) was the strongest predictor of diagnostic criteria for HCM (LV wall thickness $\geq$15 mm) (area under the curve 0.99, $p<0.001$, sensitivity 98%, specificity 97%).

Conclusions: In HCM patients with preserved LVEF, (i) LSS was lower and End/Epi ratio was higher than in controls. (ii) End/Epi ratio ($\geq$1.3) was the strongest predictor of abnormal wall thickness of HCM. The mechanism of higher End/Epi ratio in HCM might be attributable to the more common myofibrillar disarray in mid- and epicardial layers. Variations of LSS represented by End/Epi ratio might have the potential to accurately detect HCM and to elucidate the pathophysiology of impaired LV wall motion at cellular level in HCM.