Non Invasive Imaging

Abstract:

**Background:** Hypertrophic Cardiomyopathy (HCM) is characterized by myocyte hypertrophy, disarray and fibrosis. We investigated the relationship between corrected QT interval dispersion (QTc dispersion), an index of the spatial dispersion of ventricular recovery times, and myocardial delayed enhancement (DE) as well as T1 relaxation times, indicators of replacement and interstitial fibrosis, respectively, in HCM.

**Methods:** EKG, echocardiography and CMR (cardiac magnetic resonance) were performed in 112 patients with a clinical diagnosis of HCM. EKG measurements were performed manually, using a standardized caliper program with further automatic computation of corrected QT intervals. DE was measured using a semi-automated threshold technique; TI scout images were used to compute T1 relaxation times after administration of Gadolinium.

**Results:** DE was evident in 62% of HCM patients. Inter-ventricular septal thickness was higher in patients with evidence of DE (p=0.0096), but left ventricular outflow tract gradients (LVOTG) at rest and following stress (exercise) were similar in patients with/without DE (rest/stress LVOTG=10+/-.8.8 and 39+/-.36.5 mmHg in patients with DE versus 11.5+/-.8.5 and 35+/-.32 mmHg in patients without DE). Majority (75%) of HCM patients had abnormal EKGs; patients with abnormal baseline EKGs had a 5 fold higher probability of having DE, when compared to patients with normal EKGs (OR 4.63 [1.726 12.438], p = 0.002). The median values of QTc dispersion tended to be higher when DE was present, in HCM patients with normal EKGs. However, in patients with abnormal EKGs, QTc dispersion was similar in patients with/without DE. The median for T1 relaxation times was 382.6ms. A strong inverse correlation was seen between QTc dispersion and T1 relaxation times, with lead group V1-V4 having the strongest correlation (p<0.001). There was no correlation between QTc dispersion and LV mass.

**Conclusion:** QTc dispersion is inversely correlated with interstitial fibrosis, reflected by lower T1 relaxation times, but not left ventricular mass in HCM. Shortened T1 relaxation times and increased QTc dispersion could be useful biomarkers for adverse electrophysiologic remodeling in HCM.