Myocardial extracellular space expansion is related to burden of premature ventricular contractions in patients with hypertrophic cardiomyopathy without non-sustained ventricular tachycardia

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Background: Current guidelines suggest the presence of non-sustained ventricular tachycardia (NSVT) as a risk factor of sudden cardiac death in patients with hypertrophic cardiomyopathy (HCM). However, high burden of premature ventricular contraction (PVC) may reflect myocardial fibrosis although the absence of NSVT.

Purpose: We investigated the association between PVC burden and myocardial extracellular space expansion in HCM patients without NSVT.

Methods: Of the 212 patients prospectively enrolled to the HCM registry of genetics, 84 patients were evaluated with both cardiac magnetic resonance and 24hr holter. Among them, 71 patients (58 males, mean age: 71 ± 13 years) have not been diagnosed with NSVT.

Results: Patients with NSVT (n = 13) showed more impaired LA functional indices and higher myocardial fibrosis burden compared with patients without NSVT (n = 71). Among patients who have not been diagnosed with NSVT, patients with late gadolinium enhancement (LGE, n = 46) had a higher total beats (109 ± 332 vs. 7 ± 13 beats per a day, p = 0.003) and burden (0.114 ± 0.225 vs. 0.008 ± 0.014 %, p = 0.003) of PVC during 24-hour compared with patients without LGE (n = 25). %LGE was correlated with total beats of PVC (r = 0.358, p = 0.002) and PVC burden (r = 0.377, p = 0.001). ECV also correlated with total beats of PVC (r = 0.387, p = 0.001) and PVC burden (r = 0.401, p = 0.001). The optimal cutoff value for PVC number was 45 (37.0% of sensitivity and 100% of specificity) with 0.733 of the area under the ROC curve (p < 0.001). Pathogenic or likely pathogenic sarcomere mutation was higher in NSVT group than no NSVT group (p < 0.05), and had a higher tendency in higher PVC burden group (0.05 < p < 0.1) than lower PVC burden group.

Conclusions: Total beats and burden of PVC are significantly related to increase in myocardial fibrosis in HCM patients without NSVT.

Abstract Figure. Mechanism of ventricular arrhythmia