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Meeting abstract

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# 233 Evidence that post-contrast delayed enhancement identified by cardiovascular magnetic resonance is an arrhythmogenic substrate in hypertrophic cardiomyopathy

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### Introduction

Delayed enhancement (DE) on contrast-enhanced cardiovascular magnetic resonance (CMR), representative of myocardial fibrosis is relatively common in the left ventricle (LV) of patients with hypertrophic cardiomyopathy (HCM). However, the clinical significance and arrhythmogenic potential of this finding remains unresolved.

## **Purpose**

Therefore, the objective of the present study was to assess the prevalence and frequency of spontaneous tachyarrhythmias in HCM with regard to DE on contrastenhanced CMR.

### **Methods**

A total of 177 HCM patients (age  $41 \pm 16$  years; 95% asymptomatic or mildly symptomatic) were evaluated by 24-hour ambulatory Holter ECG and contrast-enhanced CMR.

### Results

A total of 72 patients (41%) had DE (transmural in 39/72), occupying  $8.5 \pm 7.8$  percent of the LV wall. Presence of premature ventricular contractions (PVCs), couplets

and nonsustained ventricular tachycardia (NSVT) were more common in patients with DE than those without DE (PVCs: 89% vs. 72%; couplets: 40% vs. 17%; NSVT: 28% vs. 4%; p < 0.0001–0.007). Patients with DE also had greater numbers of PVCs (202 ± 655 vs. 116 ± 435), couplets (1.9 ± 5 vs. 1.2 ± 10), and NSVT runs (0.4 ± 0.8 vs. 0.06 ± 0.4) than did non-DE patients (all p < 0.0001); DE was an independent predictor of NSVT (relative risk 7.3, 95% CI 2.6 – 20.4; p < 0.0001). Of note, however, extent (%) of DE was similar in patients with and without PVCs (8.2% vs. 9.1%; p = 0.93), couplets (8.5% vs. 8.4%; p = 0.99) and NSVT (8.3% vs. 8.5%; p = 0.35).

# Conclusion

In this large HCM cohort with no or only mild symptoms, myocardial fibrosis detected by CMR was associated with greater likelihood and increased frequency of ventricular tachyarrhythmias (including NSVT) on ambulatory Holter ECG. Therefore, contrast-enhanced CMR identifies HCM patients having increased susceptibility to ventricular tachyarrhythmias with implications for sudden death risk stratification.

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