Journal of Cardiovascular Magnetic Resonance

Poster presentation

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Mitral valve abnormalities identified by cardiovascular magnetic resonance represent a primary phenotypic expression of hypertrophic cardiomyopathy

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from 13th Annual SCMR Scientific Sessions
Phoenix, AZ, USA. 21-24 January 2010

Published: 21 January 2010

Journal of Cardiovascular Magnetic Resonance 2010, **12**(Suppl 1):P187 doi:10.1186/1532-429X-12-S1-P187

This abstract is available from: http://jcmr-online.com/content/12/S1/P187

Introduction

Hypertrophic cardiomyopathy (HCM) is a genetic heart disease characterized by left ventricular (LV) hypertrophy due to mutations in 11 genes encoding proteins of the sarcomere. Whether a primary abnormality of the mitral valve is part of this disease process is unresolved.

Purpose

We have applied cardiovascular magnetic resonance (CMR) with its high spatial and temporal resolution, to characterize mitral valve morphology in a large HCM population.

Methods

Cine CMR images were obtained in 224 HCM subjects (42 ± 18 years; 71% male) and 70 normal control subjects (41 ± 17 years; 69% male). ECG-gated, breath-hold cines were obtained in the 3-chamber cine view (3CV) and in contiguous 10 mm thick short-axis slices achieving complete coverage of the LV. Anterior mitral leaflet (AML) and posterior mitral leaflet (PML) lengths were manually measured at end-diastole in the 3CV when the leaflets were fully extended parallel to the anterior septum and anterolateral wall.

Results

The AML and PML lengths were greater in HCM patients compared to controls (26 ± 4 mm vs. 20 ± 4 mm; $P < 0.001$ and 14 ± 4 mm vs. 11 ± 3 mm; $P < 0.001$, respectively), including 24 (10%) with substantially increased AML length of ≥32 mm (≥3 SD above control). AML and PML lengths measured *in vivo* by CMR did not differ significantly from that previously reported valves removed at surgery or postmortem (p = NS).

Although AML length was longer in male patients (26 ± 4 mm vs. 25 ± 4 mm in females, p = 0.04) there was no difference with respect to age, so that HCM patients ≤16 years of age had similar leaflet lengths to those ≥65 years (23 ± 5 mm vs. 26 ± 3 mm; p = 0.05). In addition, there was no difference in AML length in HCM patients with or without LV outflow obstruction (27 ± 4 vs. 26 ± 5 mm; p = 0.10), nor between AML length and maximum LV wall thickness (R = 0.11; p = 0.09) or total LV mass (R = 0.72; p = 0.3). Specifically, in HCM patients with only limited hypertrophy (13-19 mm), 31 patients (14%) had AML lengths which were greatly increased (≥2 SD above controls).

Conclusion

CMR provides an accurate *in vivo* assessment of mitral valve leaflets lengths, which are increased in HCM patients, unrelated to other clinical and demographic variables, including age and magnitude of LV hypertrophy. These findings suggest that in addition to LV hypertrophy, enlargement of the mitral valve is likely part of the primary phenotypic expression of HCM.