Journal of Cardiovascular Magnetic Resonance

Technologist presentation

The concordance rates between left ventricular hypertrophy and right ventricular hypertrophy in patients with hypertrophic cardiomyopathy as diagnosed by CMR with fibrosis imaging

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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):T10 doi:10.1186/1532-429X-12-S1-T10

This abstract is available from: http://jcmr-online.com/content/12/S1/T10

Introduction

CMR has become the leading technique to define the clinical impact of hypertrophic cardiomyopathy (HCM) providing complete coverage of both ventricles with high spatial resolution. Gadolinium delayed-hyperenhancement (DHE) accurately identifies regions of myocardial fibrosis. Via CMR, innumerable studies have established that LVH is the predominant phenotypic expression of HCM. It is well known that myocardial fibrosis can occur in HCM patients and is independently linked to a poorer prognosis than those without fibrosis by CMR. The genotypic expressions would appear to affect the entire heart yet, classically, investigations have been limited to the LV, in some part due to inability to image the smaller, thinner RV. Thus, little information is available about the RV in HCM.

Purpose

We hypothesize there is significant RV involvement in HCM when incorporating a CMR analysis for RV hypertrophy and fibrosis.

Methods

Review of all patients referred for HCM was performed. SSFP/DHE was used to diagnose patients with HCM, using gadolinium administration (0.15mmol/kg, MultiHance, Bracco Diagnostics, Princeton,NJ). Post-injection (2/10 minutes) DHE images were obtained using manual T1-weighted, inversion recovery preparations. Regions of myocardium with abnormally high signals were designated as fibrotic. LV/RV mass indices (LVMI/RVMI) were calculated. Fibrosis was semi-quantitatively assessed in both ventricles.

Results

Via 44 patients referred for HCM from 2006-2010, 15 (34%) were CMR confirmed. Image quality was judged excellent in14/15 (93%). The mean LVMI was 86±26gm^2 (>2SD >normal) while the mean RVMI was 23±6gm^2 (1SD >normal). All patients met formal LVH criteria while 6/15 (40%) met RVH criteria. 12/15 (80%) had evidence of LV fibrosis while 9/15 (60%) had evidence for RV fibrosis (Figure 1). No patient with RV fibrosis had absent LV fibrosis. In neither LV nor RV was there a difference in the degree of hypertrophy as to prediction of likelihood for fibrosis (88±28 vs.83±25g/m^2 and 21±5 vs.23±6g/m^2, respectively) but on average, DHE+ patients had more, not less hypertrophy.

Conclusions

The frequency of RVH/fibrosis in the setting of HCM is surprising in that this phenomenon is rarely, if ever, described. However, given the genetic abnormalities, there is no reason to expect phenotypic expression should

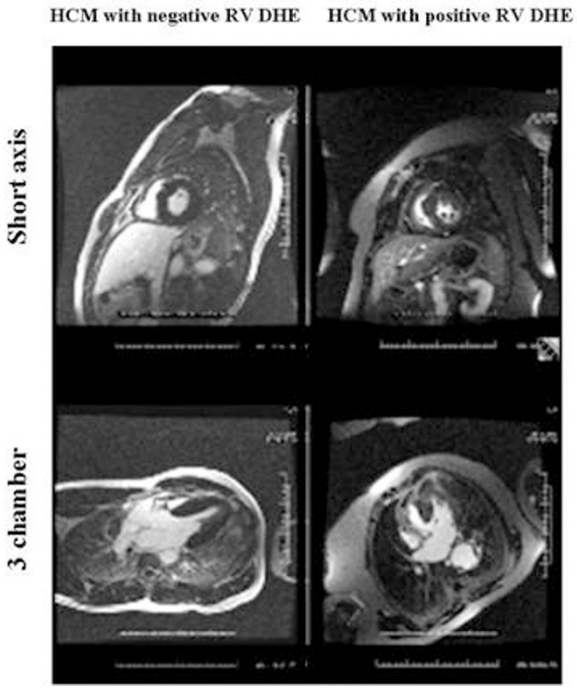

Figure 1

be limited to the LV. Interestingly, similar to the LV, the degree of RVH had little predictive power towards the finding of DHE-defined RV fibrosis. When considering HCM, it is important to recognize that the pathology is not limited to the LV and CMR resolution helps remind us of the concordance between the two ventricles.