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Meeting abstract

2061 Delayed-enhancement in hypertrophic cardiomyopathy is linked to a low coronary sinus flow independently from the left ventricular mass

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Introduction

Previous study demonstrated that fibrosis in Hypertrophic cardiomyopathy (HCM) was not directly linked to the disarray phenomenon. Chronic ischemia due to the disproportion between mass and total coronary flow could be a decisive factor in the genesis of fibrosis in this cardiomyopathy. Cardiovascular Magnetic Resonance (CMR) allows to detect and quantify fibrosis in HCM as well as to quantify coronary flow by the measurement of coronary sinus flow (CSF).

Purpose

To determine if the relation between total coronary flow and left ventricular mass was different in in HCM and normal patients and its role in the development of fibrosis.

Methods

We enrolled thirty-one patients (20 male, mean age 39 year) with a confirmed diagnosis of HCM and 20 normal patients (11 male, mean age 35 years). All the patients underwent CMR examination by a 1.5 T GE Signa Cvi scanner with a 8 channel phased array cardiac coil. Study protocol included the evaluation of left ventricular (LV) mass by the acquisition of FIESTA images (30 phases for cardiac cycle, 8 mm slice thickness, no gap) in short axis views covering the entire LV mass. CSF was measured by a Phase-velocity encoded pulse sequence acquired orthogonally to the coronary sinus with a velocity encoded of 50 cm/sec. The cardiac cycle was subdivided in a number of frame in relation to the heart rate in order to obtain a time-gap between 10 and 15 milliseconds between every image. Fibrosis was evaluated by Delayed Enhancement (DE) technique obtained with an IR GRE pulse sequence with a TI prefixed to null normal myocardium acquired in short axis views. A software ad hoc was used to quantify delayed hyper-enhanced (DHE), mild-enhanced (MDE) and non-enhanced regions.

Results

CSF was not significantly lower in HCM patients than in normal patients (2.17 ± 1 vs 2.29 ± 1.2 ml), but the ratio CSF/mass was lower in HCM than in normal patients (12.4 ± 4 vs 22 ± 10 × 10-3 ml/grams, $p < 0.0001$). Twenty-two HCM patients (73.3%) had a positive DE (DE-HCM), 8 patients (26.7%) showed a negative DE (No-DE-HCM). No differences were found in mass and mass index between DE-HCM and no-DE-HCM groups (189 ± 33 vs 196 ± 33 grams and 97 ± 24 vs 106 ± 18 grams/m2 respectively). CSF in DE-HCM group was lower than in No-DE-HCM (1.93 ± 0.5 vs 3.13 ± 0.6 ml, $p < 0.0001$). Yet, the ratio CSF/mass was lower in DE-HCM patients (11 ± 4 vs 16 ± 3 × 10-3 ml/grams, $p < 0.003$). As evidenced in Figure 1D, the extent of HDE and MD E significantly correlated with CSF and CSF/mass.

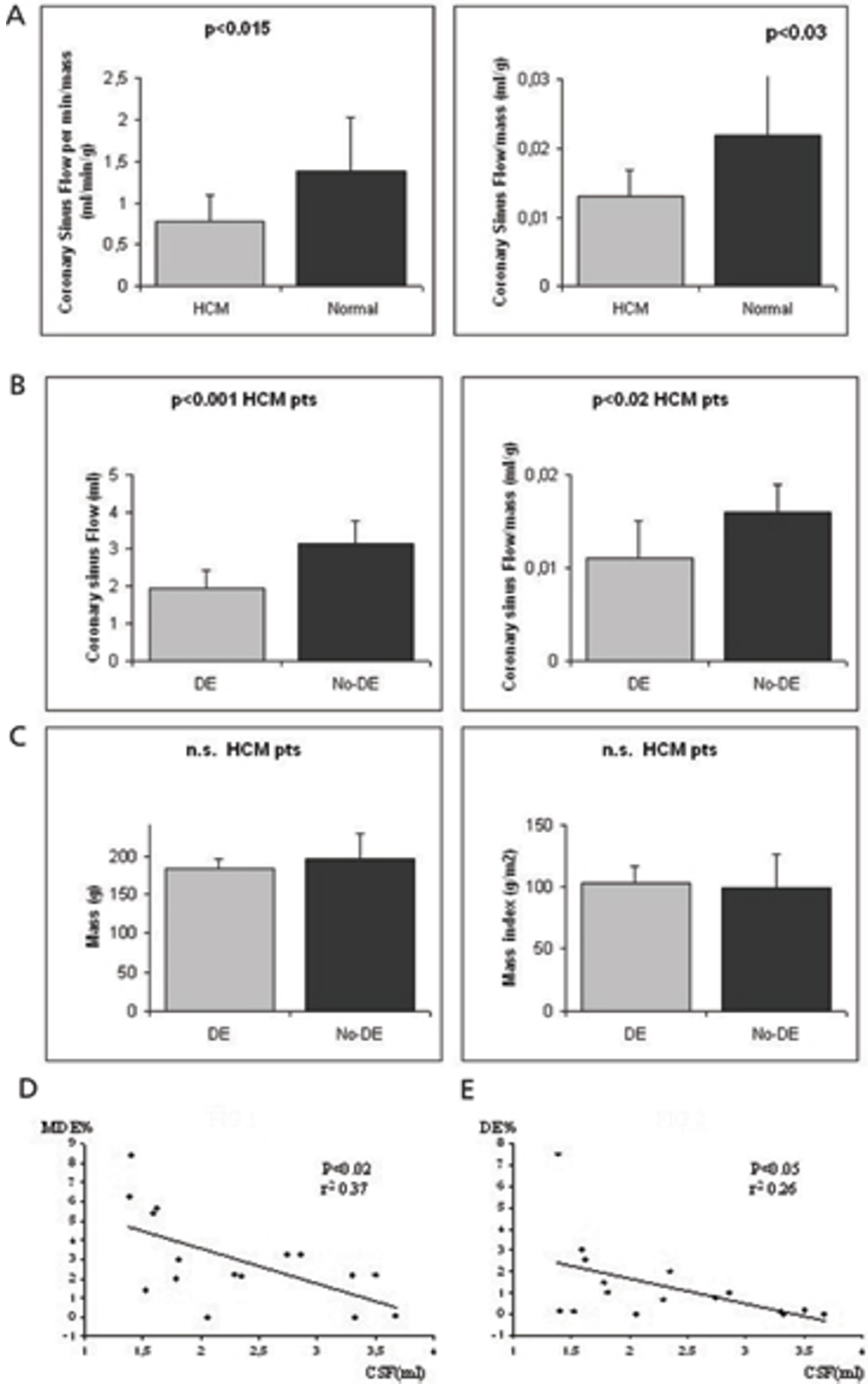
A
p<0.015
Coronary Sinus Flow per min/mass (ml/min/g)
2,5
2
1,5
1
0,5
0
HCM
Normal
p<0.03
Coronary Sinus Flow/mass (ml/g)
0,03
0,02
0,01
0
HCM
Normal
B
p<0.001 HCM pts
Coronary sinus Flow (ml)
5
4
3
2
1
0
DE
No-DE
p<0.02 HCM pts
Coronary sinus Flow/mass (ml/g)
0,02
0,01
0
DE
No-DE
C
n.s. HCM pts
Mass (g)
200
150
100
50
0
DE
No-DE
n.s. HCM pts
Mass index (g/m2)
150
100
50
0
DE
No-DE
D
MD E%
P<0.02
r² 0.37
CSF(ml)
E
DE%
P<0.05
r² 0.26
CSF(ml)

Figure 1

Conclusion

This study confirmed that in HCM an inappropriate hypertrophy is not adequately compensated by an increase in total coronary flow. The signficant difference in CSF between HCM patients with and without DE suggests that fibrosis could probably be linked to this disproportion between coronary flow and LV mass. Furthermore in HCM patient the extent of HDE and MDE were inversely related to the coronary flow.