

Increased left ventricular torsion in hypertrophic cardiomyopathy mutation carriers with normal wall thickness

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Background:

Hypertrophic cardiomyopathy (HCM) is characterized by asymmetrical (septal) hypertrophy in the absence of increased external load and is caused by mutations in mainly sarcomeric genes (1,2). Increased left ventricular (LV) torsion has been observed in patients with manifest hypertrophy. Since LV torsion is generated by counteracting contractions of obliquely oriented myofibers in favour of the subepicardial fibers, an increase in torsion is thought to be caused by subendocardial dysfunction. We hypothesize that abnormalities in myocardial contractility might already be present in HCM mutation carriers with normal wall thickness. Therefore, the purpose of our study was to determine LV torsion in HCM mutation carriers with normal wall thickness.

Methods:

Seventeen carriers with an LV wall thickness <10mm, and seventeen age and gender matched controls underwent MRI cine imaging and tissue tagging during single or multiple, respectively, breath hold acquisitions in mild expiration. All subjects were imaged on a 1.5T whole body scanner (Magnetom Sonata, Siemens, Erlangen, Germany), using a six-channel phased array body coil.

LV volumes and mass were calculated from retro-triggered, balanced SSFP short-axis cine imaging with full coverage of the LV. Image parameters were: slice thickness 5mm, slice gap mm, temporal resolution <50ms, echo time 1.54ms, repetition time 3.2ms, flip angle 60°, image resolution 1.3x1.6mm². Dedicated software was used for post-processing (Mass, Medis, Leiden, the Netherlands).

LV torsion, torsion rate and endocardial circumferential strain, were determined using retro-triggered, sinusoidal SSFP CSPAMM tagging (3), on 3 short-axis slices evenly distributed over an end-systolic four-chamber view. Image parameters were: slice thickness 6mm, FoV 300x300mm², matrix size 256x78, temporal resolution 15ms, echo time 1.8ms, repetition time 3.6ms, flip angle 20°, tag-line distance 7mm. The HARP method was used for post-processing, as described before (4-6).

Furthermore, as there normally exists a constant ratio between LV torsion and ejection (which is directly dependent on subendocardial circumferential strain) (7), the ratio of torsion to subendocardial circumferential strain (TECS) was calculated.

Comparisons were made using Student's T-test or Mann-Whitney U test, where appropriate. Differences were considered to be statistically significant when p<0.05.

Results:

LV volumes, mass and circumferential strain were comparable between groups, whereas LV ejection fraction, torsion (Fig. 1a) and TECS-ratio (Fig. 1b) were increased in carriers compared to controls (see Table 1).

Table 1. Results.

	Carriers (n=17)	Controls (n=17)	p-value
End-diastolic volume (ml)	177 ± 29	180 ± 35	Not significant
End-systolic volume (ml)	67 ± 12	72 ± 16	Not significant
Ejection fraction (%)	63 ± 3	60 ± 3	0.04
Mass (g)	104 ± 26	104 ± 30	Not significant
Torsion (°)	10.1 ± 2.5	7.7 ± 1.2	0.001
Subendocardial circumferential strain (%)	19.9 ± 2.8	18.9 ± 2.6	Not significant
TECS ratio (°/%)	0.52 ± 0.14	0.42 ± 0.10	0.02

Discussion and Conclusion:

HCM mutation carriers with normal wall thickness display increased LV torsion and TECS-ratio with respect to controls. The observed difference might be due to HCM-related subendocardial myocardial dysfunction. Future studies are needed to establish the role of LV torsion as a diagnostic tool to identify carriers.

References:

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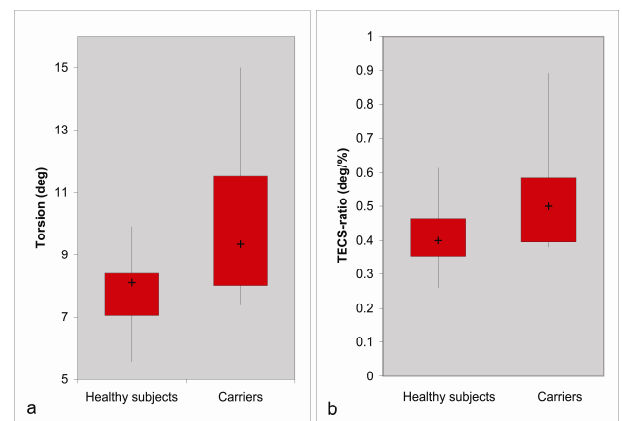


Figure 1. Boxplots of LV torsion (a) and TECS-ratio (b) in healthy subjects and carriers.