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CMR detects abnormal septal convexity into the left ventricle in preclinical hypertrophic cardiomyopathy

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Background

Sarcomeric gene mutations are responsible for hypertrophic cardiomyopathy (HCM). In gene mutation carriers without significant left ventricular (LV) hypertrophy (G+LVH-), subtle abnormalities can exist as mitral valve elongation, crypts, markers of elevated LV systolic function, and abnormal apical trabeculation. Reverse curvature of the interventricular septum into the LV is a characteristic of G+LVH+ patients. We aimed to assess LV septal convexity into the LV in G+LVH-.

Methods

Cardiovascular magnetic resonance was performed on 36 G+LVH- individuals (31±14 years, 33% men) with a pathogenic sarcomere mutation, and 36 sex and age-matched healthy volunteers (33±12 years, 33% men). Septal convexity (SCx) was measured in the apical four cavities view using a reference line joining the mid-septal wall at tricuspid valve insertion level and the apical right ventricular insertion. SCx was the maximal distance from this line to the LV endocardium border (A-B). The Figure depicts an example of SCx into LV in a G+LVH- (a) *vs.* matched control (b).

Results

Mean septal convexity into LV was 5.0±2.5mm in G+LVH- *vs.* 1.6±2.4mm in controls (p≤0.0001). Compared to controls, G+LVH- individuals also had longer anterior mitral valve leaflet (23.5±3.0mm *vs.* 19.9±3.1mm, p≤0.0001), higher relative wall thickness (0.31±0.05 *vs.* 0.29±0.04, p≤0.05), higher ejection fraction (70.8±4.3% *vs.* 68.3±4.4%, p≤0.05), and smaller LV end-systolic volume index (21.4±4.4ml/m2 *vs.* 23.7±5.8ml/m2, p≤0.05). Other morphologic measurements of LV angles, sphericity index, and excentricity index were not significantly different between G+LVH- individuals and controls.

Conclusions

Septal convexity into LV is an additional feature of preclinical HCM, occurring before the presence of any hypertrophy.

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