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Meeting abstract

2032 The relationship between electrocardiographic and cardiac magnetic resonance (CMR)-derived left ventricular parameters differs between physiologic and pathologic hypertrophy

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Background

Hypertensive left ventricular hypertrophy (LVH) and increased LV mass (LVM) compound its long-term cardiovascular risk, whereas in athletes LVH is innocuous. Electrocardiography (ECG) is commonly used to diagnose LVH, but has low sensitivity. CMR affords greater accuracy in assessing LVH and LVM. Few studies have looked at the relationship between ECG and CMR parameters and none in hypertensives or athletes. We therefore investigated the relationship between ECG markers of LVH and CMR-derived LV parameters in athletes and hypertensives with LVH based on ECG voltage criteria. We hypothesized that in these groups there should be similar correlation between ECG voltages/mass on the one hand, and CMR wall thickness/mass on the other.

Methods

Uncomplicated hypertensives (n = 16) without other cardiovascular risks and professional athletes (n = 11), both with ECG LVH were selected for this study. Controls (n = 18) were healthy volunteers. Blood pressure (BP) was measured in a sitting position using a mercury sphygmomanometer after a 10-minutes rest. 12 leads ECGs were obtained in supine position. QRS duration, Sokolow-Lyon (SL) voltage, Cornell (CL) product, the sum of QRS voltages and ECG derived LVM were measured. CMR was performed on a 1.5 Tesla scanner (Sonata; Siemens Medical Solutions, Erlangen, Germany) using validated cardiac protocols. Images were analysed using the Argus software (version 2002B; Siemens Medical Solutions). The following LV parameters were obtained; end-diastolic volume index (EDVI), mass and mass index (LVMI), end-diastolic (edwt) and end-systolic (eswt) wall thickness sums based on the AHA segment model. Continuous variables were compared using one way ANOVA. Pearson's correlation coefficient was used to establish relationship between CMR and ECG variables.

Results

Compared to controls (n = 18, age 39.2 ± 13.8 yrs, females = 5) and athletes (n = 11, age 24 ± 3 yrs, female = 1), hypertensives (n = 16, age 57.7 ± 13.9 yrs, 5 females) were older, $p < 0.001$. Body mass indices were similar amongst the groups and athletes (2.07 ± 0.24 m^2) had larger body surface areas than controls (1.88 ± 0.17 m^2), $p < 0.027$. Systolic and diastolic BP (mmHg) were higher in hypertensives (147 ± 16/83 ± 11) than in athletes (120 ± 9/65 ± 8) and controls (117 ± 12/72 ± 8), $p < 0.001$ for both BP. Hypertensives (3.14 ± 0.77) and athletes (3.68 ± 0.70) had comparable Sokolow-Lyon voltages (mV) that were significantly higher than those of controls (2.38 ± 5.1), $p < 0.001$).

LVEDVI (cm^3/m^2) was significantly larger in athletes (108.63 ± 12.44) than in both hypertensives (80.39 ± 38.9) and controls (77.72 ± 13.92), $p < 0.001$. Hypertensives (143.6 ± 29.0) and athletes (150.9 ± 21.9) had a similar sum of the end-diastolic wall thickness (edwt) (mm)

Table 1: Correlation coefficient between ECG and CMR parameters in hypertensives

	Sum of edwt	Sum of eswt	CMRLVM	CMRLVMI
QRS duration	0.78***	0.74***	0.85***	0.84***
SL voltage	0.78***	0.79***	0.73***	0.70***
Cornell product	0.79***	0.61*	0.88***	0.89***
ECGmass	036	069**	0.28	0.18
ECGmass index	039	0.72**	038	0.35

* denotes $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

that were significantly higher than in controls (111.4 ± 14.1), $p < 0.001$). However, hypertensives (201.2 ± 26.8) and controls (195.7 ± 28.9) had comparable sum of end-systolic wall thickness (eswt) (mm) that were significantly lower than that of athletes (262.1 ± 29.50), $p < 0.001$. Hypertensives (175.5 ± 110.8 and 92.2 ± 52.8) and athletes (209.5 ± 70.3 and 101.4 ± 22.7) had comparable CMR-derived LVM (g) and mass indices (g/m^2) that were significantly higher than those of controls (102.3 ± 27.5 and 54.01 ± 11.5), $p < 0.001$.

Table 1 shows correlation coefficient between ECG and CMR parameters in hypertensives. Hypertensives showed significant correlations between ECG variables and CMR parameters.

In contrast, athletes showed no significant correlation between CMR parameters and QRS duration, SL and Cornell product; only ECG-derived LVM showed significant correlation with all CMR parameters, i.e. sum of edwt (r = 0.72*), sum eswt (r = 0.70*), CMR derived LVM (r = 0.88**) and mass index (r = 0.73*). Similarly, in controls only SL voltage and ECG LVM correlated with CMR derived LVMI (r = 0.54* and 0.64* respectively).

Conclusion

Despite similar degree of LV hypertrophy, hypertensives and athletes show different correlations between ECG variables and CMR derived LV parameters.