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Derangement of cardiac energy metabolism is acutely exacerbated during exercise in hypertrophic cardiomyopathy, independent of hypertrophy or late gadolinium burden

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Summary

This work demonstrates that cardiac energetcics is further impaired during exercise in hypertrophic cardiomyopathy. This may be a possible reason for exercise related death in HCM.

Background

In hypertrophic cardiomyopathy (HCM), sarcomere mutations increase the energy cost of contraction. Impaired resting cardiac energetics as measured by phosphocreatine/adenosine triphosphate (PCr/ATP) using 31Phosphorus MR Spectroscopy(31P MRS) has been documented in animal models and patients.

We hypothesize that: 1.Cardiac energetics are further impaired acutely during exercise in HCM, which does not occur in normals or athletes (physiological hypertrophy); 2. This impairment is not related to the degree of hypertrophy or late gadolinium enhancement (LGE) burden.

Methods

Cardiac 31P MRS (3T) was performed in 35 HCM patients, 12 athletes and 20 normal controls (all age- and gender-matched) at rest and during 8 minutes of prone leg exercise with 2.5 kg weights attached to both legs. Cine and LGE images were also acquired.

Results

Increases in rate pressure product with exercise were similar: normal 72±44%; HCM 73±38%; Athlete 75±47%.

There was no difference in resting PCr/ATP between normals (2.14±0.36) and athletes (2.04±0.32, P=0.36). Resting PCr/ATP was significantly reduced in HCM, (1.71±0.35, P<0.01 compared to normal and athletes, figure 1).

During exercise, there was a further reduction in PCr/ATP in HCM (1.56±0.31, P<0.05), but no significant change in normals (2.13±0.34, P=0.98), and athletes (2.09±0.50, P=0.63, figure 1). The change of PCr/ATP during exercise in HCM (-0.14±0.34) was significantly different (P<0.05) from the change in normals (+0.05 ±0.27).

LV mass index was higher in HCM (90±14g/m2) and athletes (92±19g/m2) compared to normal (65±10g/m2) P >0.05.There was no correlation between cardiac mass index and rest PCr/ATP ratios (HCM: R=0.01, P=NS; athletes: R=0.22 P=NS) or change in PCr/ATP with exercise.

In HCM, average wall thickness at voxel placement for PCr/ATP was 18±6mm (range 7.8-28.8 mm). This did not correlate with resting PCr/ ATP or change in energetics with exercise. Wall thicknesses were normal in the athlete group, 9±2mm.

Normals and athletes had no LGE. In HCM, the average LGE >2SD in the mid ventricular septum was 24 ±15%. LGE correlated weakly with resting PCr/ATP ratio, (R=- 0.35, P=0.04), and did not correlate with absolute exercise or change in PCr/ATP with exercise.

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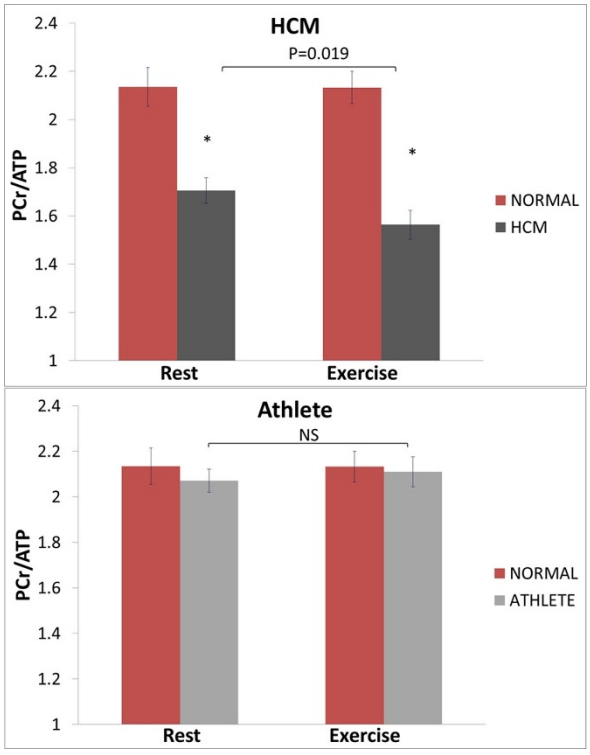

Figure 1 The effect of exercise on PCr/ATP in normal controls, HCM, and Athletes, showing a reduction in the HCM group, but no change in athletes or normal.*p<0.05 vs normal.

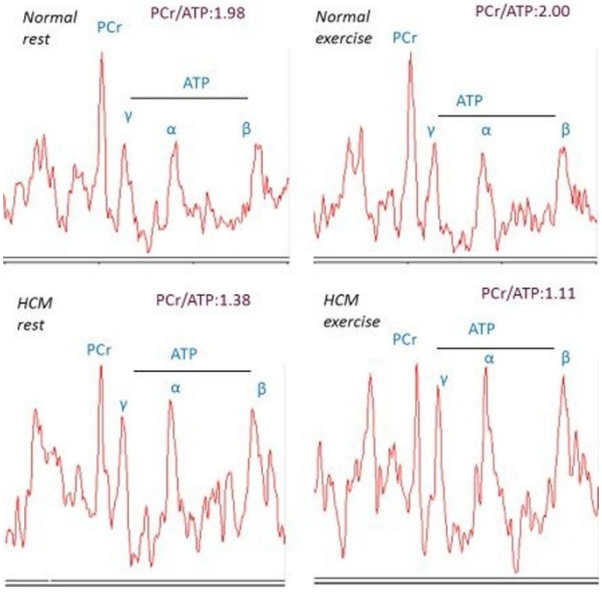

Figure 2 Examples of rest and exercise energetics in normal and HCM.

Conclusions

During exercise, the pre-existing energetic deficit in HCM is further exacerbated and is not influenced by the degree of hypertrophy or scar burden. Acute derangement of energy-dependent ion homeostasis, triggering Ca++ overload and ventricular arrhythmias, may be a possible explanation for the high incidence of exercise-related death in HCM. Treatments that optimize energetics may be protective.

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