Elite Olympic Calibre High-Endurance Athletes Have Evidence For Myocardial Fibrosis : A Cardiovascular Magnetic Resonance Study

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Background:

Athletes are at almost a 3-fold greater risk of developing sudden cardiac death than healthy individuals, and an increased number of sudden deaths occur in relation to physical exercise. In the field of cardiology, the concept of the 'athlete's heart' has emerged, where abnormal conduction of cardiac rhythm is considered to be 'normal' for athletes. Long-term investigation of athletes presenting with severe conduction abnormalities, found that these individuals were more likely to develop adverse cardiac events. Moreover, myocardial fibrosis has shown to result in an increased propensity to develop arrhythmias, conduction blocks, which can lead to terminal cardiac events. Case reports of autopsy findings in athletes have revealed that athletes possess myocardial injury and fibrosis. It is also well documented that during competitive events, serological markers for cardiac injury such as troponin, are elevated.

Cardiovascular magnetic resonance (CMR) has emerged as an important diagnostic tool for the evaluation of tissue characteristics. T1-weighted late enhancement can non-invasively identify the presence and extent of myocardial fibrosis in various cardiomyopathies. We undertook this study to assess whether elite Olympic caliber athletes present with evidence for myocardial injury.

Hypothesis:

We hypothesized that fibrosis, as demonstrated by CMR-based late enhancement imaging, is a prevalent finding in elite athletes, and is associated with reduced cardiac function.

Methods:

We prospectively recruited 48 elite high-endurance athletes (25 males, 32±13 years), as well as 8 healthy volunteers to serve as a control group (4 males, 31±9 years). All images were acquired with a 1.5 T scanner (Avanto, Siemens Medical Solutions, Erlangen, Germany). Using standard cine-SSFP, left ventricular function was acquired in short axis with complete coverage of the left ventricle (10mm slices, 0 gap). Following Gadolinium-DTPA injection of a 0.20 mmol/kg dose, T1-weighted late enhancement images were acquired using a phase sensitive inversion recovery sequence. Late enhancement images were acquired in the same slice positions as function images, with 10mm slices and 0 gap, as well as in long axis orientations (4-, 3-, and 2-chamber views).

Standard approach to quantifying left ventricular function was applied. Two experienced independent observers assessed for the presence of myocardial fibrosis visually. Late enhancement was considered to be present if it was reproducible in 2 or more slice orientations.

Results:

37 of 48 athletes (77%) had evidence for myocardial fibrosis, while only 1 of 8 healthy control subjects did (13% incidence) (Figures 1 and 2). Athletes who had cardiac fibrosis had larger left-ventricular end-diastolic volume indexed-to-height (117±19 vs. 98±15 ml/m, $p<0.05$), a larger end-systolic volume indexed-to-height (44±11 vs. 34±10 ml/m, $p<0.05$) and a higher stroke volume indexed-to-height (73±11 vs. 64±7 ml/m, $p<0.05$), while ejection fraction (63±5 vs. 66±6%, $p>0.05$) did not differ, when compared to those who did not have fibrosis.

Conclusion:

Our results demonstrate that elite high-endurance athletes have a high incidence of myocardial injury and fibrosis. Fibrosis in these athletes also comes with reduced contractile function and larger hearts, while overall cardiac function remains unchanged. Further evaluation of the long-term consequences of the presence of myocardial fibrosis in elite athletes is required.

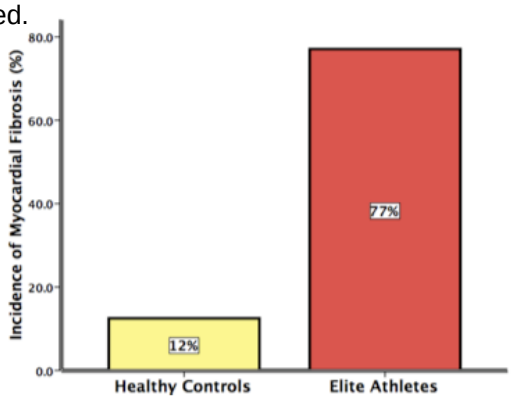

Figure 1. Elite Olympic calibre high-endurance athletes have a very high incidence of myocardial fibrosis (77%), compared to healthy individuals from the general public (13%).

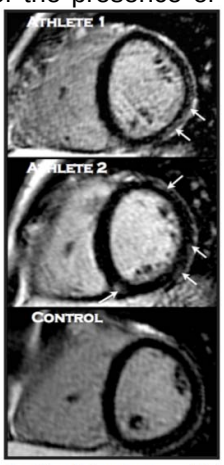

Figure 2. Elite high-endurance athletes with evidence for myocardial fibrosis (white arrows), while there is no evidence for fibrosis in a healthy control.