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Cardiac damage induced by long term and intensive endurance exercise training in senior athletes: a cardiac magnetic resonance and exercise echocardiography analysis

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Background: Moderate intensity regular physical activity is a mainstay of cardiovascular prevention. However, it is under debate whether the cumulative effects of intensive endurance exercise induce chronic cardiac damage. It has been proposed that long-term endurance exercise may be associated with myocardial fibrosis detected by late gadolinium-enhancement (LGE) cardiac magnetic resonance (CMR), and/or with right ventricle contractile dysfunction, creating a potential substrate for ventricular arrhythmia, but these aspects are not constantly found across studies. Extracellular volume (ECV) determined by CMR, circulating cardiac biomarkers, and longitudinal global 2D strain (LGS) assessed by cardiac echography are relevant tools to explore these potential adverse effects of exercise in senior athletes.

Purpose: To assess the presence of myocardial fibrosis detected by CMR (LGE and ECV) and biomarkers, and to evaluate ventricles function with LGS, in endurance senior competitive athletes.

Methods: Thirty-three asymptomatic endurance senior athletes (47 ± 6 years old, 9,6 ± 1,7 h of training per week for 26 ± 6 years), were compared to 18 sedentary controls (49 ± 7 years old). They underwent a CMR protocol including morphological and late gadolinium-enhancement (LGE) analysis, T1 mapping and calculation of ECV using a 1.5 T MRI scanner. A maximal exercise transthoracic echography with LGS analysis was performed. Cardiac biomarkers (N terminal pro brain natriuretic peptide, high-sensitivity troponin T and plasma markers of collagen biosynthesis and degradation procollagen type I N terminal propeptide and procollagen type III NT propeptide) were also analysed.

Results: Athletes had higher left (84,0 ± 20,8 vs 67,62 ± 12,4 ml/m²; p=0,00593) and right (41,3 ± 9,5 vs 32,9 ± 6,0 ml/m²; p=0,00228) ventricular volumes, higher total biventricular mass (109,61 ± 14,18 vs 98,66 ± 16,25 g/m²; p=0,02038), and higher stroke volume (50,7 ± 12,6 vs 40,1 ± 9,2 ml/m²; p=0,00441) assessed by CMR versus controls. Native T1 (left ventricle: 938 ± 21 vs 940 ± 34 ms; p=0,8098 - right ventricle: 1027 ± 53 vs 1026 ± 114 ms; p=0,92983) and ECV (21,5 ± 1,6 vs 22,0 ± 2,2 %; p=0,40868) were not significantly different in athletes compared with controls. LGE was not found in athletes. Peak exercise LGS values were higher in athletes for left (23,19 ± 2,86 vs 20,82 ± 2,05 %; p=0,00335) and right (29,25 ± 3,47 vs 26,48 ± 3,22 %; p=0,00777) ventricles, compared with control. The levels of cardiac biomarkers were normal in all subjects.

Conclusion: Athletes showed signs of physiological cardiac remodelling, consistent with previous descriptions of athlete’s heart. Despite this important remodelling, there was no evidence of myocardial fibrosis or exercise left or right ventricular dysfunction in athletes. Our results are not supporting the hypothesis of deleterious cardiac effects induced by long term and intensive endurance exercise training.