

Hinge point fibrosis in athletes is not associated with structural, functional or electrical consequences: a comparison between young and middle-aged elite endurance athletes

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Background: The health benefits of extensive endurance training have been debated due to the report of myocardial fibrosis (MF), arrhythmias and temporary post-race cardiac impairment in middle-aged and veteran athletes. The extent of these changes is unknown in elite young athletes.

Purpose: To assess the prevalence of MF and its structural, functional and electrical impact in highly trained young endurance athletes (YA, 15–23 years) as compared to middle-aged athletes (MA, 30–50 years). We hypothesised that MF would be more frequent in MA and associated with more structural, functional and electrical abnormalities.

Methods: We prospectively assessed 197 YA and 34 MA. All had ECG, maximal oxygen consumption (VO₂max) testing, cardiac magnetic resonance imaging (CMR), echocardiography and 24h-holter. Indexed left ventricular and right ventricular end diastolic volume (LVEDVi, RVEDVi), ejection fraction (LVEF, RVEF), left ventricular mass (LVMi), and MF defined as delayed gadolinium enhancement were assessed by CMR. LV and RV free wall strain (LVSL, RVfWSL) were assessed by 2D speckle tracking echocardiography. Ventricular premature beats (VPB) and non-sustained ventricular tachycardia (nsVT) were assessed by 24h-holter.

Results: YA and MA (18±2 vs 38±5 years [$p<0.01$]; 78% vs 80% male [$p=0.99$]) with an elite level of fitness (VO₂max 61±8 vs 54±10 mL/min/kg [$p<0.01$]; % predicted VO₂max 150±20 vs 158±30 [$p=0.02$]) had a large variance in LV and RV remodelling (Figure 1). MF was seen in 28 athletes

(12.5%) and more prevalent in MA than in YA (23.5 vs 10.5%, $p=0.048$). MF was limited to the hinge points in all 8 MA with MF and 17 YA. 3 YA had LV lateral wall subepicardial MF. 27 of 187 (14.4%) male athletes had MF compared to 1 of 50 (2%) female athletes ($p=0.01$).

MF+ MA(A) and YA(B) as well as MF– MA(C) and YA(D) had similar structural remodelling (LVEDVi 110±14 vs 118±14 vs 113±19 vs 110±16 mL/m²; RVEDVi 120±14 vs 128±17 vs 117±19 vs 125±23 mL/m²; LVMi 77±11 vs 83±14 vs 81±14 vs 77±15 g/m², $p>0.05$). LVEF, LVSL and RVSL were similar (59±3 vs 58±5 vs 61±6 vs 58±6%; –18.8±2 vs –18.8±2 vs –19.8±2 vs –19.3±2%; –26.3±2.4 vs –24.4±2.4; –26.3±3 vs –25.8±3.5% respectively, $p>0.05$). LVEF <50% was seen in 19 (8.2%) athletes (0 [0%] vs [5%] 1 vs 1 [3.8%] vs 17 [9.6%]; $p=0.51$). RVEF was higher in D compared to C without further differences between groups (54±4 vs 54±6 vs 53±6 vs 57±5, $p=0.005$). RVEF <45% was seen 21 (9.1%) athletes (0 [0%] vs 1 [5%] vs 0 [0%] vs 20 [11.3%]; $p=0.14$). Abnormal T-wave inversion was similar (12.5 vs 5 vs 7.4 vs 6.2%, $p=0.93$) as was the prevalence of >100VPB/24h (12.5 vs 5 vs 11.1 vs 5.1%, $p=0.42$). 2 athletes had nsVT, both in D. All had similar exercise capacity (% predicted VO₂max 157±26 vs 152±15 vs 147±24 vs 158±32%; $p=0.11$).

Conclusion: Hinge-point fibrosis was more prevalent in MA, possibly due to repeated hemodynamic stress during exercise, but is not associated with structural, functional or electrical consequences.

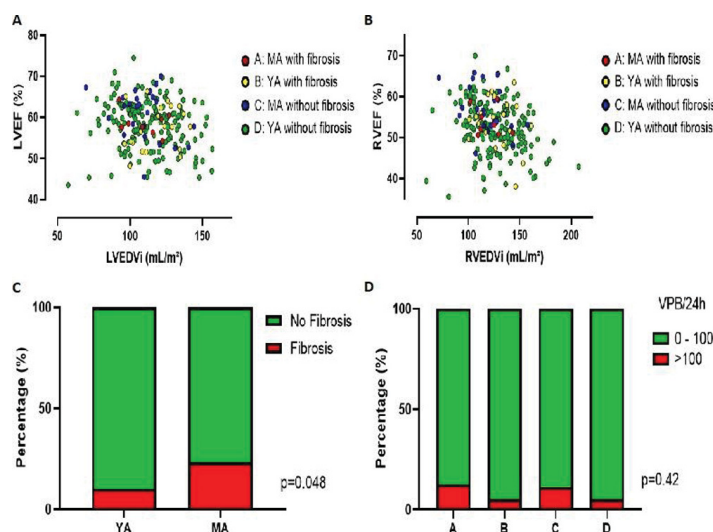


Figure 1. Cardiac remodelling in elite athletes