Impact of Exercise Training on Arterial Wall Thickness and Distensibility in Young Competitive Athletes

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Objective: The thickening of the carotid artery wall has been described as a marker of early atherosclerosis. Exercise is associated with decreased CV risk and higher fitness confers cardioprotection. However, studies examining the impact of exercise training on arterial wall thickness in healthy young adults lead to contradictory results. Little is currently known regarding exercise and remodeling processes with regard to diameter (wall-to-lumen ratio (W-to-L-ratio)) and arterial stiffness of the carotid artery. The aim of this prospective study was to examine the carotid intima-media thickness (cIMT) in young competitive athletes compared to reference values. Further to analyze associations between cIMT, W-to-L ratio as well as intraventricular septal thickness (IVSd), leftventricular posterior wall dimensions (LVPWd) and exercise performance.

Methods: cIMT, W-to-L-ratio and arterial stiffness of the A. carotis communis were measured with ultrasonography in elite youth soccer players (n=34 boys, age 14-18 years, training duration 15-20 h/week). The athlete’s performance was quantified by cardiopulmonary exercise testing, measuring the percentage of oxygen consumption (VO2%) and the relative exercise performance (Watt/kg). Echocardiography was performed to evaluate myocardial wall thicknesses.

Results: cIMT (cIMT_right 0.531±0.03mm; cIMT_left 0.519±0.04mm) were above the 75th percentile compared to age-matched reference values. Multivariate regression analysis revealed a significant model on cIMT_right only (adjusted for age and blood pressure) and could explain 17.4% of the variance (VO2%: ß=0.000 p=0.312; relative performance (Watt/kg) ß=0.037, p=0.024, R2=0.174. Regarding cardiac structure (IVSd: 10.1 ±1.8 mm; IVSd z-score: 1.13±0.88; LVPWd: 8.6±1.57, LVPWd z-score: 0.80±0.85) and arterial stiffness no outstanding detection or significant correlations occurred.

Conclusion: An increased cIMT in young athletes might be considered as a vascular adaptation to exercise. Like adaptation mechanisms in athlete’s heart, the thickening could be interpreted as hypertrophy of smooth muscle cells, in order to economize the arteries work under elevated shear stress. The mechanisms explaining changes in the arterial wall by revealing the arterial elasticity as a result of exercise training are not fully understood. Identifying these stimuli in a larger cohort will help in the design and recommendation of optimal exercise training protocols to attenuate atherosclerosis burden and risk.