Your athlete-patient has a high coronary artery calcification score—‘Heart of Stone’. What should you advise? Is exercise safe?

Katharina Lechner, Bianca Spanier, Benjamin Lechner, Johannes Scherr

Coronary artery calcification (CAC) is a strong marker of subclinical coronary atherosclerosis and leading authorities recommend CAC scoring to help inform patient management decisions in cardiovascular disease (CVD) prevention. This will result in an increasing number of athlete-patients with subclinical coronary atherosclerosis presenting to sport and exercise medicine physicians, raising questions about exercise recommendations in this subgroup. With a specific focus on the recent outcomes data of DeFina and colleagues we extend our recent discussion of the topic by focusing on how to manage athlete-patients with elevated CAC in the sport and exercise medicine setting.

PHYSICAL ACTIVITY, CORONARY ARTERY CALCIUM AND CARDIOVASCULAR OUTCOMES—HIGHER PHYSICAL ACTIVITY PROTECTS AT EVERY LEVEL OF CAC

A breakthrough in reporting the association of CAC and mortality risk across different activity levels came from a recent study of 21758 healthy male participants without prevalent CVD. Higher levels of leisure-time physical activity were associated with a lower risk of mortality at any given level of CAC. The authors reported a higher risk metabolic profile (ie, higher baseline blood pressure, higher glucose concentrations and higher triglycerides) in the high volume exercise group with elevated CAC ≥100 AU compared with the high volume exercise group with CAC <100 AU. Preceding this observation, we reported that while training volume was not associated with impaired vascular function in marathon runners, higher blood glucose concentrations at baseline were associated with greater increases in carotid intima-media thickness over time.

EXERCISE RECOMMENDATIONS FOR ATHLETES WITH HIGH CAC

A zero CAC score denotes a very low risk profile, independent of the presence of traditional risk factors. Conversely, the presence of CAC in athletes deserves a thoughtful cardiovascular risk assessment. As outlined above, athletes are not immune to modifiable and non-modifiable cardiovascular risk factors and despite high activity levels, often exhibit cardiometabolic risk factors. See our related discussion for more detail. Note that exposure to risk factors at younger age may result in premature atherosclerotic plaque formation, an effect that may not be fully reversible by subsequent lifestyle changes. This raises the possibility of reverse causality in older athletes with elevated CAC—those older people with risk factors may start exercising because they are advised to by their physicians. The concept of cumulative burden as a major determinant of risk underpins the importance of taking into account lifetime exposure of risk factors.

General recommendations in athletes with CAC should promote healthy lifestyle choices like dietary patterns low in sugar, refined starches and trans fatty acids, and intermittent abstinence from nutrient exposure, avoiding cigarette smoke, regular circadian rhythms with a sleep duration of 6–8 hours per day and learning strategies to mitigate the physiological response to distress. If appropriate, pharmacotherapy should be initiated in athlete-patients at high risk for adverse cardiovascular events according to guidelines. Lipid modulating therapy with statins, despite the well-established effect on reducing cardiovascular end points, has been linked to increased CAC progression. One of the mechanisms underlying this observation may be that statin therapy promotes calcification of pre-existing plaque and might thus decrease the risk of acute plaque destabilisation.

DOES EXERCISE STABILISE CORONARY ARTERY PLAQUES?

It is tempting to speculate that physical activity may operate through similar plaque stabilising mechanisms. Physical activity promotes a more benign plaque phenotype with a higher collagen and elastin content, increased fibrous cap thickness, decreased necrotic lipid core and increased calcification. This renders plaque less prone to acute destabilisation and the associated clinical sequelae of arterial occlusion and tissue infarction, and provides plausibility for why higher levels of both physical activity and cardiorespiratory fitness are inversely related to all-cause mortality and cardiovascular mortality. As high level leisure-time physical activity is safe at every level of CAC it should be advised in the asymptomatic patient with CAC. Importantly, if typical (ie, chest pain on exertion) or atypical (ie, exercise intolerance or reduced exercise capacity) symptoms occur that suggest flow-limiting stenosis, the patient requires further diagnostic workup and referral to a sports cardiologist (diagnostic algorithms are reviewed by Borjesson and colleagues). Furthermore, athlete-patients with coronary artery disease (ie, CAC) who want to engage in competitive sports need to undergo individual evaluation by a sports cardiologist (reviewed by Borjesson and colleagues).

TAKE HOME MESSAGE

The presence of CAC is a strong predictor of CVD risk in the general population and also in highly active individuals. CAC in athletes is often associated with cardiovascular risk factors. These must be addressed by lifestyle modification and, if appropriate, pharmacotherapy. The collective evidence from analyses of plaque composition in athletes and from recent clinical outcomes data lends qualified support for the conclusion that—at any given level of CAC—long-term exposure to high leisure-time physical activity levels is safe, compatible with long-term health and confers prognostic benefit. We promote the advice ‘Keep Exercising’ as an important health message, even in individuals with—as sports cardiologist Dr Aaron Baggish cleverly termed it—‘Hearts of Stone’. 

1Department of Prevention and Sports Medicine, Technical University Munich, Munich, Germany
2Department of Internal Medicine V, Ludwig Maximillians University Munich, Munich, Germany
3University Center for Preventive and Sports Medicine, Balgrist University Hospital, University of Zurich, Zurich, Switzerland

Correspondence to Dr Katharina Lechner, Department of Cardiology, German Heart Center Technical University Munich, Lazarettstraße 36, 80636 München, Germany; contact@katharinalechner.net


The authors thank Jonathan A Drezner for his intellectual contribution during the revision process.

All authors contributed to this discussion. KL did the literature search, and drafted the manuscript. BS, BL and JS reviewed and edited the manuscript. All authors approved the final version of the manuscript.

The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

None declared.

Not required.

Not commissioned; externally peer reviewed.

© Author(s) (or their employer(s)) 2020. No commercial re-use. See rights and permissions. Published by BMJ.


