Journal of Cardiology and Cardiovascular Sciences

Mini Review Open Access

Cardiac Morbidities in Elite Endurance Athletes: Rare but Potentially Serious

Ritesh Chandrasekaran[1]*, Amit Sagar[1], George R. Luck[1], Charles H. Hennekens[1]

[1]Florida Atlantic University, Schmidt College of Medicine.

Article Info

Article Notes
Received: February 04, 2026
Accepted: July 24, 2026

***Correspondence:**
Ritesh Chandrasekaran, Florida Atlantic University, Schmidt College of Medicine. Email: riteshchandra046@gmail.com.

© 2026 Chandrasekaran R. This article is distributed under the terms of the Creative Commons Attribution 4.0 International License.

Keywords:
Elite athletes
Cardiac morbidities
Endurance athletes
Masters athletes
Extreme exercise
Hypertension in athletes
Exercise-induced hypertension
Ventricular remodeling
Vascular remodeling
Sedentary behavior
Cardiac remodeling

Abstract

Elite endurance athletes (EEAs) have exceptional physical and mental capabilities. These allow EEAs to perform physical activities at extraordinary levels. Nonetheless, although any medical complications EEAs suffer are very rare, they may develop hypertension, atrial fibrillation, and even coronary heart disease (CHD). In our mini review we consider first the basic research and then the clinical relevance of these very rare but potentially serious complications. After completing our mini review, we conclude that the benefits accrued to EEAs participating in their extremely high levels of daily physical activities, far exceed their rare but potentially serious complications of hypertension, atrial fibrillation and CHD. These findings in EEAs are compatible with those in the general population that indicate the need to achieve higher daily levels of physical activities. In the United States (US), only 21% of individuals achieve even the minimally recommended daily levels of physical activity. These low levels can be achieved even among the oldest old. Thus, physical inactivity in the US seems to be the new "silent killer" replacing hypertension the old "silent killer."

Dedication

We dedicate this manuscript to the memory of our recently deceased valued colleague and dear friend, Professor Steven Lewis.

Introduction

Elite endurance athletes (EEAs) perform at extraordinary levels. They often possess a high VO2 max, the maximum rate of oxygen consumption during intense exercise, as well as an elevated lactate threshold which delays the onset of fatigue. These physiological adaptations enable them to achieve and sustain higher intensities of physical activities. These physiological adaptations are consistent with morphologic data of cardiac remodeling, including atrial dilatation and left ventricular hypertrophy, which increase cardiac output and oxygen delivery to muscles.

In some EEAs, cardiac remodeling and the attendant stressors may, at least in theory, increase the rate of morbidities such as atrial fibrillation (AF), hypertension, and coronary artery disease (CAD). In this mini review we consider the basic research and clinical relevance of these very rare but potentially serious complications.

Methodology

To identify the most relevant peer reviewed publications we used PubMed, Embase and the Cochrane Library. We included search terms of: elite athletes, cardiac morbidities, hypertension, atrial fibrillation, cardiac remodeling, and cardiac mortality.

Inclusion Criteria

All articles included were published in English from 1998 to 2025 and included men and women EEAs.

Coronary Artery Disease

EEAs tend to exhibit elevated coronary artery calcium (CAC) scores, a marker for atherosclerotic burden. Long-term marathon, ultramarathon, and extreme runners had higher CAC score than in sub marathon runners.[12] Moreover, athletes in the highest tier of exercise volume (>2,000 MET-min/week) exhibited a significantly higher prevalence of CAC scores (68%) compared to those exercising less (43%).[13] In some studies, masters athletes with low risks of CAD did not have significantly higher CAC scores than controls.[6,9] Male masters athletes were significantly more likely to have CAC scores > 300. In a recent study of 105 male EEAs with a mean age of 38, ¼ had high CAC scores.[3] Moreover, EEAs with a low risk of CAD had significantly higher percentage of having >1 coronary plaques than healthy, non-athletes.[8]

Pathophysiology

The pathophysiology underlying accelerated coronary atherosclerosis is multifactorial but remains incompletely understood. With respect to EEAs, their marked increases in cardiac output and contractility during intense exercise may amplify the natural torsion and flexion of the coronary arteries, promoting shear stress and flow turbulence at arterial branch points.[1] Those who practice the higher intensity of endurance exercise have transient elevations in inflammatory cytokines.[2,3] Endurance exercise may increase parathyroid hormone levels, potentially disrupting calcium homeostasis and facilitating deposition within the arterial wall.[3] Testosterone may also be a contributor. If so, this might explain, at least in part, why predominantly male EEAs have higher CAC scores.[4]

Plaque Morphology

Elevated CAC scores in EEAs were initially interpreted as a benign consequence of having a higher proportion of calcified plaque compared with the mixed-composition or non-calcified plaques more commonly observed in sedentary controls.[5-7] Calcified plaques are denser and generally considered less prone to precipitate acute coronary events. It has been further hypothesized that endurance exercise may promote calcification of pre-existing plaques, suggesting that higher CAC scores in EEAs might even reflect a stabilizing, potentially protective mechanism.[1]

In a recent investigation, both athletes and nonathletes have similar calcified plaque. Lifelong athletes had a higher percentage of mixed and non-calcified plaques, as well as proximal plaques and lesions with significant stenosis, all of which are risk factors for CAD.[8] It is plausible that the potential plaque stabilizing effects of exercise diminish with age. In an 8-year follow-up of EEAs over age 35, the percentage of calcified plaque in athletes dropped from 80% at baseline to 43% at follow up.[9]

We believe that elevated CAC scores in EEAs should not prompt a reduction in exercise. This is especially true for female athletes, in whom elite endurance training does not appear to correlate with increased CAC or adverse plaque morphology. EEAs, particularly masters EEAs, should continue to undergo regular, personalized cardiovascular screening. Evidence based atherosclerotic therapy, particularly high potency statins, remains essential to reducing the risk of acute coronary events.[15]

Clinical Implications

In a study of 108 male marathon runners aged 50 and over, CAC scores were significantly higher than controls after matching for Framingham Risk Score.[11] Subsequent studies have shown this relationship to be related to the amount of exercise. Moreover, athletes in the highest tier of exercise volume (>2,000 MET-min/week) exhibited a significantly higher prevalence of CAC scores (68%) compared to those exercising less (43%).[13] In some studies, masters athletes with low risks of CHD did not have significantly higher CAC scores than in controls.[6,9] Male masters athletes were significantly more likely to have CAC scores > 300. In a recent study of 105 male EEAs with a mean age of 38, ¼ had high CAC scores.[3] Moreover, EEAs of low risk of CHD had significantly higher percentage of having >1 coronary plaques than healthy, non-athletes.[8]

The clinical significance of higher CAC scores in EEAs remains uncertain. These scores do not appear to predict subsequent risks of CHD. Among 798 EEAs followed over five years, major adverse cardiac events occurred at a low rate of 2.8 per 1,000-years.[14] Further, 90% of men in the general population who experienced an event had a Framingham Risk Score greater than 10%, underscoring that age and traditional cardiovascular risk factors are more powerful predictors of events than athletic participation itself.

Hypertension

Hypertension seems lower in EEAs regardless of activity level. In one study of master EEAs the prevalence of hypertension was 8.7%, or <1/3 that of the 27.8% in matched controls.[16] In this study, EEAs generally maintained lower body mass, and benefits of exercise persisted even after adjusting for body mass index, suggesting that the mechanisms of protection extend beyond weight management alone. Consistent findings were observed in a study of 2,793 participants in the World Masters Games. Hypertension prevalence was 8.1% among master EEAs compared to 17.2% in the general population.[17]

When stratifying the World Masters Games athletes by gender, only 19.9% of men and 49.7% of women were normotensive. Among 214 male masters athletes with low cardiovascular risk, 26% were hypertensive at rest.[18] Further, 41% showed ambulatory and 12% showed exercise hypertension. These findings may result, at least in part, from increased cardiac output and subsequent left ventricular hypertrophy (LVH). In a 50-year follow-up study of 764 Tokyo Olympian EEAs, athletes with baseline electrographic LVH had a significantly higher 60% incidence of hypertension when compared with those without.[19]

Regular physical activity is a cornerstone of blood pressure control and is consistently associated with meaningful reductions in resting systolic and diastolic blood pressure.[20,21]

Atrial Fibrillation

EEAs tend to have higher risks of atrial fibrillation correlated with their lifetime exercise exposure. In a study of over 200,000 participants in the Vasaloppet ski race, men with the highest number of completed races and the fastest finishing times had the highest risks of AF.[9] In another study, athletes with a lifetime accumulation of 1,500 hours of vigorous sport exercise had a nearly threefold increased risk of AF compared to controls with less training.[23] In several studies of over 600 participants, EEAs had a five-fold increase in risk of AF.[26,27] In a meta-analysis of observational studies of nearly 100,000 participants, athletes engaged in vigorous exercise had a nearly threefold higher risk of developing AF.[25]

Most of these studies diagnosed lifelong EEAs with atrial fibrillation around age 50 or above. In a retrospective observational cohort study, 6,813 young EEAs had an exceptionally low incidence of AF of 0.3%.[22] These observations contributed to the formulation of a hypothesis that any arrhythmogenic substrate is a chronic maladaptation requiring decades of hemodynamic overload to manifest. In one study, those who had a lifetime accumulation of 1,500 hours of vigorous sport practice had a nearly threefold increased risk of AF.[23]

In a prospective study, moderately active individuals had a 32% lower risk of AF than inactive individuals.[24] In a meta-analysis of observational studies of nearly 100,000 participants, those with sedentary lifestyles had higher risks of developing AF.[25] In several studies of over 600 participants, EEAs had a five-fold increase in risk of AF.[26,27] EEAs also have increased left atrial diameter and volume.[28] The pathophysiology of AF in athletes has been described as Coumel's Triangle, where endurance training uniquely impacts all three corners of arrhythmogenesis: the substrate, the trigger, and the modulator.

Clinical Implications

EEAs should be closely monitored for AF. Detraining (reducing exercise intensity/duration) can reduce arrhythmia risk.[33] If detraining fails, rate control should be achieved and, if necessary, rhythm control attempted for symptomatic athletes. If medical management fails, catheter ablation may be a reasonable option, but data are sparse in EEAs. Despite increased risk of developing AF, EEAs with AF have lower risks of stroke and death compared to non-athletes.[31]

Coronary Heart Disease

The benefits of increased physical activity on risks of CHD begin immediately and are sustained.[34] In a study of 108 male marathon runners aged 50 years or greater, CAC scores were significantly higher than controls when matched for Framingham Risk Score.[35] Another more recent study showed that long term marathon and ultramarathon EEAs had significantly higher rates of CAC than sub marathon runners.[12] Moreover, athletes in the highest tier of exercise volume of >2,000 MET-min/week had a significantly higher prevalence of CAC of 68% compared to 43%.[5] In other studies, EEAs did not have significantly higher CAC scores than controls.[6,9] In subgroup analyses, male athletes were significantly more likely to have a CAC > 300. In a recent study of 105 male EEAs with a mean age of 38 years, 1 in 4 had elevated CACs despite most having only low-to-moderate risks of CHD.[3] Moreover, among low-risk individuals, lifelong EEAs were significantly more likely to have >1 coronary plaques than healthy, non-athletes.[8]

Discussion

Although they possess extreme physical fitness, EEAs can develop hypertension, AF and CHD. The large benefits of exercise, however, far exceed these small risks. Male EEAs appear to have a higher prevalence of elevated CAC scores, though the clinical significance of this finding remains uncertain. They also demonstrate an increased risk of AF, with both CAC burden and AF risk appearing to rise in a dose-dependent manner with cumulative exercise exposure. Female EEAs seem to derive cardiovascular benefit without a comparable increase in risk. With regards to hypertension, high-volume training appears to confer a protective effect in both sexes.

EEAs experience repetitive coronary shear stress, chronic inflammatory activation, and hormonal changes, which may promote coronary artery calcification. EEAs also experience sustained cardiac volume overload, which may increase their risk of atrial fibrillation through atrial dilation, alterations in autonomic tone, and increased ectopic activity. EEAs who develop hypertension may be predisposed by LVH secondary to increased cardiac output.

Cardiopulmonary exercise testing (CPET) has emerged as a valuable screening tool for EEAs. CPET provides an integrated assessment of cardiovascular, pulmonary, and metabolic function under physiologic stress. Key parameters including VO2 max, ventilatory efficiency, oxygen pulse, and heart rate recovery can identify subtle functional impairments that may not be apparent at rest or with standard exercise testing.[38]

Despite their risk of cardiac morbidities, EEAs consistently demonstrate very low absolute event rates. The major risk factors of overweight and obesity, inactivity, and smoking are the strongest predictors of CHD. We believe the totality of evidence favors regular physical activity, particularly in the US where only 21% of individuals achieve even the minimally recommended daily levels of physical activity.[21] They also reinforce the importance of individualized risk assessment, aggressive management of traditional cardiovascular risk factors, and continued promotion of physical activity.

Gender Differences

Based on a preponderance of evidence in men, female EEAs tend to have lower risks. Several mechanisms have been proposed including the smaller heart sizes and blood vessels of women. When women experience menopause their risks of CHD increase, due, at least in part, to declines in estrogen levels.[36] Men also typically present with heavily calcified plaques that can cause blockages but are less likely to rupture.[37]

Declarations and Statements

Conflicts of Interest

The authors declare no conflicts of interest. Professor Hennekens also declares that he serves as an independent scientist in an advisory role to investigators and sponsors as Chair of two Data Monitoring Committees for Amgen (erenumab and evolocumab); to the United States Food and Drug Administration as a Special Government Employee (SGE) and UpToDate; receives royalties for authorship or editorship of 3 textbooks; has an investment management relationship with the West-Bacon Group within Truist Investment Services, which has discretionary investment authority; does not own any common or preferred stock in any pharmaceutical or medical device company.

Acknowledgment

We are indebted to Gina Seits, BS, for her expert technical assistance.

References

1. Fernandez AB, Chaudhry W, Thompson PD. Coronary atherosclerosis in masters athletes: mechanisms and implications for cardiovascular disease risk. Curr Treat Options Cardiovasc Med. 2019;21(12):87. doi:10.1007/s11936-019-0798-0
2. Celeski M, Di Gioia G, Nusca A, Segreti A, Squeo MR, Lemme E, et al. The spectrum of coronary artery disease in elite endurance athletes—a long-standing debate: state-of-the-art review. J Clin Med. 2024;13(17):5144. doi:10.3390/jcm13175144
3. Certo Pereira J, Santos R, Moscoso Costa F, Monge J, de Araújo Gonçalves P, Dores H. Coronary atherosclerotic burden in veteran athletes: the relationship between cardiovascular risk and volume of exercise. Rev Port Cardiol. 2024;43(7):377–384. doi:10.1016/j.repc.2024.03.001
4. Zambrano A, Tintut Y, Demer LL, Hsu JJ. Potential mechanisms linking high-volume exercise with coronary artery calcification. Heart. 2023;109(15):1139–1145. doi:10.1136/heartjnl-2022-321986
5. Aengevaeren VL, Mosterd A, Braber TL, et al. Relationship between lifelong exercise volume and coronary atherosclerosis in athletes. Circulation. 2017;136(2):138–148. doi:10.1161/CIRCULATIONAHA.117.027834
6. Merghani A, Maestrini V, Rosmini S, et al. Prevalence of subclinical coronary artery disease in masters endurance athletes with a low atherosclerotic risk profile. Circulation. 2017;136(2):126–137. doi:10.1161/CIRCULATIONAHA.116.026964
7. Feuchtner G, Langer C, Barbieri F, et al. Relationship of exercise to coronary artery disease extent, severity and plaque type: a coronary computed tomography angiography study. J Cardiovasc Comput Tomogr. 2019;13(3):34–40. doi:10.1016/j.jcct.2019.02.001
8. De Bosscher R, Dausin C, Claus P, Bogaert J, Dymarkowski S, Goetschalckx K, et al. Lifelong endurance exercise and its relation with coronary atherosclerosis. Eur Heart J. 2023;44(26):2388–2399. doi:10.1093/eurheartj/ehad152
9. Fyyaz S, Merghani A, Al-Turaihi S, Chatrath N, Bhatia R, Maclachlan H, et al. Longitudinal insights into coronary plaque amongst master athletes. Eur J Prev Cardiol. 2024;31(Supplement_1):zwae175-263.
10. Papatheodorou E, Aengevaeren VL, Eijsvogels TMH, et al. Prevalence of coronary atherosclerosis in female masters endurance athletes. Circulation. 2024;150(18):1478–1480. doi:10.1161/CIRCULATIONAHA.124.069484
11. Möhlenkamp S, Lehmann N, Breuckmann F, Bröcker-Preuss M, Nassenstein K, Halle M, et al. Running: the risk of coronary events: prevalence and prognostic relevance of coronary atherosclerosis in marathon runners. Eur Heart J. 2008;29(15):1903–1910. doi:10.1093/eurheartj/ehn163
12. Jafar O, Friedman J, Bogdanowicz I, et al. Assessment of coronary atherosclerosis using calcium scores in short- and long-distance runners. Mayo Clin Proc Innov Qual Outcomes. 2019;3(2):76–77. doi:10.1016/j.mayocpiqo.2019.03.007
13. Aengevaeren VL, Mosterd A, Braber TL, et al. Relationship between lifelong exercise volume and coronary atherosclerosis in athletes. Circulation. 2017;136(2):138–148. doi:10.1161/CIRCULATIONAHA.117.027834
14. Morrison BN, Isserow S, Taunton J, et al. Masters athlete screening study (MASS): incidence of cardiovascular disease and major adverse cardiac events and efficacy of screening over five years. Eur J Prev Cardiol. 2023;30(9):887–899. doi:10.1093/eurjpc/zwad090
15. Dunn J, Hennekens CH. Editorial commentary: low density lipoprotein (LDL) and beyond in the treatment and prevention of cardiovascular disease. Trends Cardiovasc Med. 2025;35(8):539–540. doi:10.1016/j.tcm.2025.07.005
16. Hernelahti M, Kujala UM, Kaprio J, Karjalainen J, Sarna S. Hypertension in master endurance athletes. J Hypertens. 1998;16(11):1573–1577. doi:10.1097/00004872-199816110-00002
17. Climstein M, Walsh J, DeBeliso M, Heazlewood T, Sevene T, Del Vecchio L, et al. Resting blood pressure in master athletes: immune from hypertension? Sports (Basel). 2023;11(4):85. doi:10.3390/sports11040085

18. Parry-Williams G, Obaid D, Moser J, Vlahos I, Bulleros P, Fanton Z, et al. 183 Low-grade hypertension and coronary atherosclerosis in male master endurance athletes. 2021.

19. Ashikaga K, Yoneyama K, Musha H, Akashi YJ, Kawahara T. Association of electrocardiographic left ventricular hypertrophy with incident hypertension among the Tokyo 1964 Olympic athletes: a 50-year follow-up study. Sport Sci Health. 2017;8:517–527.

20. Lewis S, Hennekens CH. Regular physical activity: forgotten benefits. Am J Med. 2016;129(2):137–138.

21. Davis BR, Hennekens CH, Greenwald J, Dunn J, Matarazzo A, Benson K, et al. New clinical challenges in hypertension management: the 'old silent killer' is alive and well. Am J Med. 2024;137(12):1154–1156. doi:10.1016/j.amjmed.2024.07.005

22. Boraita A, Santos-Lozano A, Heras ME, et al. Incidence of atrial fibrillation in elite athletes. JAMA Cardiol. 2018;3(12):1200–1205. doi:10.1001/jamacardio.2018.3482

23. Elosua R, Arquer A, Mont L, Sambola A, Molina L, García-Morán E, et al. Sport practice and the risk of lone atrial fibrillation: a case-control study. Int J Cardiol. 2006;108(3):332–337. doi:10.1016/j.ijcard.2005.05.020

24. Heitmann KA, Løchen ML, Stylidis M, Hopstock LA, Schirmer H, Morseth B. Associations between physical activity, left atrial size and incident atrial fibrillation: the Tromsø Study 1994–2016. Open Heart. 2022;9(1):e001823. doi:10.1136/openhrt-2021-001823

25. Mohanty S, Mohanty P, Tamaki M, Natale V, Gianni C, Trivedi C, et al. Differential association of exercise intensity with risk of atrial fibrillation in men and women: evidence from a meta-analysis. J Cardiovasc Electrophysiol. 2016;27(9):1021–1029. doi:10.1111/jce.13023

26. Abdulla J, Nielsen JR. Is the risk of atrial fibrillation higher in athletes than in the general population? A systematic review and meta-analysis. Europace. 2009;11(9):1156–1159. doi:10.1093/europace/eup197

27. Karjalainen J, Kujala UM, Kaprio J, Sarna S, Viitasalo M. Lone atrial fibrillation in vigorously exercising middle-aged men: case-control study. BMJ. 1998;316(7147):1784–1785. doi:10.1136/bmj.316.7147.1784

28. Molina L, Mont L, Marrugat J, et al. Long-term endurance sport practice increases the incidence of lone atrial fibrillation in men: a follow-up study. Europace. 2008;10(5):618–623. doi:10.1093/europace/eun071

29. Ostojic M, Ostojic M, Petrovic O, Nedeljkovic-Arsenovic O, Perone F, Banovic M, et al. Endurance sports and atrial fibrillation: a puzzling conundrum. J Clin Med. 2024;13(24):7691. doi:10.3390/jcm13247691

30. Lobo HM, Naves ÍG, Marçal SB, Canzi CC, Rodrigues ABS, Menezes AS Jr. Atrial fibrillation in endurance training athletes: scoping review. Rev Cardiovasc Med. 2023;24(6):155. doi:10.31083/j.rcm2406155

31. Svedberg N, Sundström J, James S, et al. Long-term incidence of atrial fibrillation and stroke among cross-country skiers. Circulation. 2019;140(11):910–920.doi:10.1161/CIRCULATIONAHA.118.039461

32. Drca N, Larsson SC, Grannas D, Jensen-Urstad M. Elite female endurance athletes are at increased risk of atrial fibrillation compared with the general population: a matched cohort study. Br J Sports Med. 2023;57(18):1175–1179. doi:10.1136/bjsports-2022-106035

33. Estes NAM III, Madias C. Atrial fibrillation in athletes: a lesson in the virtue of moderation. JACC Clin Electrophysiol. 2017;3(9):921–928. doi:10.1016/j.jacep.2017.03.019

34. Kitsantas P, Caceres J, Price-Wise G, Hennekens CH. Pocketbook and Manual of Physical Activity to Combat the "New Silent Killer." 1st ed. Amazon; 2025.

35. Möhlenkamp S, Lehmann N, Breuckmann F, Bröcker-Preuss M, Nassenstein K, Halle M, et al. Running: the risk of coronary events: prevalence and prognostic relevance of coronary atherosclerosis in marathon runners. Eur Heart J. 2008;29(15):1903–1910. doi:10.1093/eurheartj/ehn163

36. Campbell KR. Women and Cardiovascular Disease: Addressing Disparities in Care. World Scientific; 2023.

37. Betai D, Ahmed AS, Saxena P, Rashid H, Patel H, Shahzadi A, et al. Gender disparities in cardiovascular disease and their management: a review. Cureus. 2024;16(5):e59663. doi:10.7759/cureus.59663

38. Kowalski T, Kasiak P, Chomiuk T, Mamcarz A, śliż D. Optimizing the interpretation of cardiopulmonary exercise testing in endurance athletes: precision approach for health and performance. Transl Sports Med. 2025;2025:5904935. doi:10.1155/tsm2/5904935