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EARLY SIGNS OF CARDIAC REMODELING UNDER EXCESSIVE PHYSICAL LOAD

Bekmirzaev Ruzimukhammad Alimjon ugli
Vakhobov Bakhromjon Muminzhanovich
Department of Faculty Therapy,
Andijan State Medical Institute, Andijan, Uzbekistan

Abstract

The cardiovascular system undergoes significant morphological and functional adaptations in response to regular physical exertion, a phenomenon widely recognized as the athlete's heart. However, the boundary between physiological adaptation and early pathological remodeling remains challenging to delineate, particularly in individuals subjected to excessive or unmanaged physical loads. This study aims to investigate the early subclinical signs of maladaptive cardiac remodeling in athletes exposed to high-intensity training regimens exceeding standard recovery capacities. Utilizing a comprehensive diagnostic approach that includes advanced echocardiography with tissue Doppler imaging and the assessment of specific cardiac biomarkers, we evaluated a cohort of subjects engaged in varying intensities of physical activity. Our findings indicate that while moderate exercise induces harmonious eccentric hypertrophy with preserved or enhanced diastolic function, chronic excessive loading is associated with a subtle shift towards concentric geometric patterns, localized reduction in myocardial relaxation velocities, and subclinical elevations in N-terminal pro-b-type natriuretic peptide and high-sensitivity cardiac troponin. These alterations suggest an early fibrotic or inflammatory myocardial response preceding overt clinical symptoms. The identification of such transition points is critical for the prevention of exercise-induced arrhythmogenic substrates and long-term myocardial dysfunction. Continuous monitoring strategies incorporating both advanced imaging and molecular markers are essential to safeguard cardiovascular health in high-performance athletic populations.

Keywords

cardiac remodeling, excessive physical load, athlete heart, echocardiography, myocardial strain, biomarkers, subclinical dysfunction, sports cardiology.

РАННИЕ ПРИЗНАКИ РЕМОДЕЛИРОВАНИЯ МИОКАРДА ПРИ ЧРЕЗМЕРНОЙ ФИЗИЧЕСКОЙ НАГРУЗКЕ

Аннотация

Сердечно-сосудистая система претерпевает значительные морфологические и функциональные адаптации в ответ на регулярные физические нагрузки, что широко известно как феномен спортивного сердца. Однако граница между физиологической адаптацией и ранним патологическим ремоделированием остается сложной для определения, особенно у лиц, подвергающихся чрезмерным или неконтролируемым физическим нагрузкам. Данное исследование направлено на изучение ранних субклинических признаков дезадаптивного ремоделирования сердца у спортсменов, подвергающихся высокоинтенсивным тренировкам, превышающим стандартные возможности восстановления. Используя комплексный диагностический подход, включающий современную эхокардиографию с тканевой допплерографией и оценку специфических кардиальных биомаркеров, мы обследовали когорту лиц, занимающихся физической активностью различной интенсивности. Наши результаты показывают, что в то время как умеренные физические нагрузки вызывают гармоничную эксцентрическую гипертрофию с сохраненной или улучшенной диастолической функцией, хронические чрезмерные нагрузки связаны с незаметным сдвигом в сторону концентрических геометрических паттернов, локальным снижением скорости релаксации миокарда и субклиническим повышением уровня N-концевого про-мозгового натрийуретического пептида и высокочувствительного сердечного тропонина. Эти изменения указывают на ранний фиброзный или воспалительный ответ миокарда, предшествующий явным клиническим симптомам. Выявление таких переходных точек имеет решающее значение для профилактики индуцированных физической нагрузкой аритмогенных субстратов и долгосрочной дисфункции миокарда. Стратегии непрерывного мониторинга, включающие как передовые методы визуализации, так и молекулярные маркеры, необходимы для сохранения здоровья сердечно-сосудистой системы у спортсменов, занимающихся спортом высших достижений.

Ключевые слова

ремоделирование сердца, чрезмерная физическая нагрузка, спортивное сердце, эхокардиография, деформация миокарда, биомаркеры, субклиническая дисфункция, спортивная кардиология.

Introduction

The profound impact of sustained physical activity on the structural and functional characteristics of the human heart has been the subject of intensive medical research for over a century. The physiological adaptations that occur in response to rigorous training regimes generally result in what is clinically referred to as the athlete's heart. This condition is typically characterized by resting bradycardia, increased left and right ventricular chamber volumes, and a proportional augmentation in myocardial wall thickness. Under normal circumstances these alterations represent a highly efficient physiological remodeling process designed to augment stroke volume and fulfill the escalated oxygen demands of working skeletal musculature during strenuous exertion. The medical community has long endorsed regular exercise as a cornerstone of cardiovascular disease prevention due to its well-documented capacity to improve endothelial function, lower systemic blood pressure, and optimize lipid profiles.

Despite the universally acknowledged benefits of regular exercise an emerging body of evidence suggests that the relationship between physical load and cardiovascular health may not be entirely linear. Recent epidemiological and clinical investigations have raised concerns regarding a potential dose-response reversal at the extreme upper limits of physical exertion. When training volumes and intensities surpass the physiological recuperative capacities of the individual the cardiovascular system may transition from a state of healthy adaptation to one of maladaptive remodeling. This grey zone between extreme physiological adaptation and the genesis of pathological cardiomyopathy represents one of the most complex diagnostic challenges in modern sports cardiology. The difficulty lies in the fact that the initial stages of excessive load-induced damage often mimic the benign characteristics of the athlete's heart rendering them undetectable through routine clinical examinations.

Prolonged and unremitting physical stress is hypothesized to induce transient states of acute volume and pressure overload. Over time these repetitive hemodynamic stressors

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combined with potential exercise-induced oxidative stress and systemic inflammatory responses may lead to cumulative micro-trauma within the myocardial architecture. The healing process subsequent to such micro-trauma often involves the deposition of extracellular matrix proteins leading to reactive interstitial fibrosis. Unlike the physiological hypertrophy which is characterized by the proportional growth of cardiomyocytes and the accompanying capillary network pathological remodeling is often accompanied by microvascular rarefaction and increased myocardial stiffness. Identifying the exact threshold where an individual crosses from healthy adaptation into the realm of excessive load-induced damage is exceedingly difficult as it is influenced by a myriad of factors including genetics, baseline fitness, nutritional status, and environmental stressors.

The primary objective of the present investigation is to elucidate the early non-invasive markers of maladaptive cardiac remodeling in individuals subjected to excessive physical loads. By utilizing a multi-parametric approach that extends beyond traditional echocardiography to include tissue deformation imaging and serological biomarker analysis this study seeks to identify subtle phenotypic shifts that precede overt functional decline. Understanding these early warning signs is imperative for sports physicians and cardiologists as it provides a critical window of opportunity for intervention. By modulating training intensity or implementing targeted recovery protocols during this early phase it may be possible to halt or even reverse the trajectory of maladaptive remodeling thereby preventing the long-term sequelae of exercise-induced cardiac dysfunction and minimizing the risk of adverse cardiovascular events during sporting activities.

Methods

The present research was designed as a cross-sectional observational study to evaluate the cardiovascular profiles of individuals exposed to varying degrees of physical exertion. The study population comprised male volunteers recruited from local sports clubs and athletic associations. To ensure a homogenous sample regarding baseline demographic characteristics strict inclusion criteria were applied. Participants were required to be between the ages of eighteen and thirty-five years, non-smokers, and free from any previously diagnosed congenital or acquired cardiovascular diseases, hypertension, metabolic disorders, or ongoing use of performance-enhancing pharmacological agents. Following the initial screening process the participants were categorized into distinct study groups based on detailed training logs and self-reported exercise histories covering the preceding two years. The categorization was designed to separate individuals with moderate health-oriented exercise routines from those engaging in high-volume intense training regimens that approach or exceed conventional professional limits.

The first group served as the active control and consisted of individuals engaging in moderate physical activity defined as three to four hours of aerobic and resistance training per week. The second group represented standard athletic adaptation comprising competitive athletes training between eight and twelve hours weekly. The third group which formed the focal point of this investigation consisted of ultra-endurance athletes and elite competitors whose weekly training volumes consistently exceeded eighteen hours with minimal allocated recovery periods. This final group was designated as the excessive load cohort. Prior to any clinical testing all participants provided written informed consent in accordance with the ethical principles outlined in the Declaration of Helsinki. The study protocol was reviewed and approved by the institutional ethics committee of the affiliated medical university.

All clinical evaluations were conducted in a standardized environment. Participants were instructed to abstain from any strenuous physical activity, caffeine, and alcohol consumption for a minimum of forty-eight hours prior to the assessment to eliminate the confounding effects of

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acute exercise fatigue. The cardiovascular assessment commenced with a comprehensive twelve-lead resting electrocardiogram to screen for pathological arrhythmias, repolarization abnormalities, and voltage criteria for ventricular hypertrophy. Following the electrocardiogram resting blood pressure was measured utilizing an automated sphygmomanometer after the participant had been seated quietly for ten minutes.

The core diagnostic modality of the study was comprehensive transthoracic echocardiography performed by a single experienced sonographer to eliminate inter-operator variability. The echocardiographic examinations were conducted utilizing a high-resolution ultrasound system equipped with a phased-array transducer. Two-dimensional imaging was used to obtain linear dimensions, volumes, and calculated variables according to the current guidelines of the American Society of Echocardiography. Left ventricular mass was calculated using the area-length method and indexed to body surface area. Relative wall thickness was determined to categorize the geometric pattern of the left ventricle as either normal, concentric remodeling, concentric hypertrophy, or eccentric hypertrophy. Systolic function was evaluated through the calculation of the left ventricular ejection fraction using the biplane Simpson's method.

To detect subtle alterations in myocardial mechanics that are not apparent on standard two-dimensional imaging the protocol incorporated tissue Doppler imaging and two-dimensional speckle tracking echocardiography. Tissue Doppler was applied at the septal and lateral aspects of the mitral annulus to record peak systolic and early and late diastolic myocardial velocities. The ratio of early transmitral flow velocity to early diastolic annular velocity was calculated as an estimate of left ventricular filling pressure. Speckle tracking echocardiography was utilized to assess global longitudinal strain which provides a highly sensitive measure of myocardial deformation and contractile efficiency. A reduction in global longitudinal strain absolute values is increasingly recognized as a harbinger of subclinical myocardial impairment.

In conjunction with the imaging protocol venous blood samples were obtained from the antecubital vein of each participant. The samples were collected in standard serum separator tubes and ethylenediaminetetraacetic acid tubes depending on the specific assay requirements. The blood was centrifuged immediately and the serum and plasma aliquots were stored at negative eighty degrees Celsius until analysis. The biochemical evaluation focused on markers of myocardial wall stress and cardiomyocyte injury. Specifically the levels of N-terminal pro-b-type natriuretic peptide and high-sensitivity cardiac troponin were quantified using commercially available electrochemiluminescence immunoassays. The assays were performed by laboratory technicians who were completely blinded to the athletic status and group allocation of the participants to prevent any observational bias.

The data acquired from the clinical, imaging, and biochemical assessments were subjected to rigorous statistical analysis. Continuous variables were expressed as means accompanied by standard deviations while categorical variables were presented as absolute numbers and percentages. The normal distribution of the data was verified using the Shapiro-Wilk test. To determine the statistical significance of differences across the three study groups a one-way analysis of variance was employed for normally distributed variables followed by post-hoc testing for multiple comparisons. For data that did not exhibit a normal distribution the Kruskal-Wallis non-parametric test was utilized. Correlations between training volume, echocardiographic parameters, and biomarker levels were explored using Pearson or Spearman correlation coefficients as appropriate. A probability value of less than zero point zero five was established as the threshold for statistical significance across all analyses.

Results

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The demographic analysis of the study population revealed no significant differences in age, height, or baseline body mass index among the three groups indicating a well-matched cohort. Resting heart rates demonstrated a predictable training-related gradient with the lowest values observed in the excessive load group reflecting enhanced vagal tone characteristic of extensive endurance training. Systolic and diastolic blood pressure readings remained within normal physiological limits across all groups and did not exhibit any statistically significant variances. The standard twelve-lead electrocardiograms predominantly displayed patterns consistent with training-related vagotonia such as sinus bradycardia and early repolarization changes in the athletic groups. No pathological arrhythmias or distinct patterns indicative of inherited channelopathies or structural heart disease were identified during the initial screening phase.

The standard two-dimensional echocardiographic evaluation demonstrated expected morphological adaptations in both the standard athletic group and the excessive load group compared to the moderate activity controls. Both athletic cohorts exhibited significantly increased left ventricular end-diastolic volumes and left ventricular mass indices indicative of exercise-induced cardiac enlargement. However a critical divergence was observed when analyzing the specific geometry of the ventricular remodeling. The standard athletic group predominantly displayed a classic eccentric hypertrophy pattern characterized by a proportional increase in chamber size and wall thickness. In contrast a notable subset of individuals within the excessive load group exhibited a disproportionate increase in wall thickness relative to chamber dimension resulting in an elevated relative wall thickness. This subtle shift towards a concentric remodeling geometry in the highest training volume group suggests a deviation from harmonious physiological growth.

Despite these morphological differences global systolic function as measured by the left ventricular ejection fraction remained robust and indistinguishable across all three groups. All participants maintained an ejection fraction well above the established lower limits of normal. The necessity for more sophisticated imaging techniques became apparent when standard systolic parameters failed to differentiate the groups. The application of speckle tracking echocardiography revealed crucial insights into myocardial mechanics. While the global longitudinal strain values for the moderate activity and standard athletic groups were well within the robust normal range and nearly identical a statistically significant attenuation in the absolute values of global longitudinal strain was recorded in the excessive load group. This microscopic reduction in longitudinal shortening occurs before any discernible drop in overall ejection fraction indicating early subclinical impairment in the longitudinal myofibers located in the subendocardium.

The assessment of diastolic function through tissue Doppler imaging further illuminated the emerging differences between the cohorts. The standard athletic group demonstrated supra-normal diastolic function evidenced by high early diastolic annular velocities and very low filling pressure estimates which is the hallmark of a highly compliant and well-conditioned ventricle. Conversely the excessive load group did not exhibit this supra-normal relaxation. Instead their early diastolic velocities were lower than those of the standard athletes and closely resembled or fell slightly below those of the moderate activity group. Furthermore the ratio estimating left ventricular filling pressures was marginally but significantly elevated in the excessive load cohort compared to the standard athletes. While these values did not reach the threshold for clinical diastolic dysfunction the trend suggests a relative stiffening of the myocardium potentially related to early interstitial changes.

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The biochemical analysis provided an additional layer of evidence supporting the imaging findings. Baseline levels of both N-terminal pro-b-type natriuretic peptide and high-sensitivity cardiac troponin were detectable across all groups. However the quantitative analysis revealed a distinct upward trajectory correlated with training volume. The excessive load group demonstrated significantly higher resting levels of both biomarkers compared to both the moderate activity and standard athletic groups. It is important to contextualize that these elevated levels generally remained below the acute diagnostic cut-offs used in emergency settings for acute coronary syndromes or heart failure. Nevertheless the chronic baseline elevation of these proteins in asymptomatic young individuals at rest strongly suggests a state of continuous low-grade myocardial wall stress and subtle ongoing cardiomyocyte membrane permeability or microscopic injury associated with extreme physical demands.

Discussion

The boundary distinguishing the supreme physiological conditioning of the athlete's heart from the incipient stages of pathological remodeling is increasingly recognized not as a sharp dividing line but as a complex and dynamic continuum. The findings of the current investigation provide compelling evidence that while standard training induces highly beneficial morphological and functional adaptations pushing physical exertion beyond individual recuperative thresholds can initiate a cascade of subclinical maladaptive changes. Our observation of a subtle transition towards concentric remodeling patterns coupled with microscopic functional decrements in the excessive load cohort aligns with the emerging hypothesis of exercise-induced cardiac fatigue and cumulative structural wear.

Historically the enlargement of the heart in athletes has been viewed universally as a positive adaptation. The classic Morganroth hypothesis postulated a dichotomous response where endurance exercise led to eccentric hypertrophy due to volume overload while resistance training resulted in concentric hypertrophy due to pressure overload. However contemporary sports cardiology acknowledges that modern training regimens usually involve mixed physiological stressors resulting in overlapping morphological phenotypes. Our results indicate that in extreme endurance and high-volume training the heart may experience prolonged periods of extreme pressure work during maximal efforts. Over years of sustained training this repeated pressure stress might override the volume-induced eccentric growth promoting a relative thickening of the walls. This concentric shift is clinically relevant because unlike eccentric hypertrophy which enhances compliance concentric geometry is often a precursor to myocardial stiffness and reduced end-diastolic volumes.

The standard echocardiographic measurement of ejection fraction proved insufficiently sensitive to detect the early consequences of this extreme remodeling. Ejection fraction relies heavily on radial contraction which often remains preserved or even hyperdynamic to compensate for failing longitudinal mechanics. The integration of speckle tracking echocardiography in our study was crucial as it exposed a significant reduction in global longitudinal strain among the athletes subjected to the highest training loads. The longitudinal myocardial fibers located in the subendocardial layer are particularly vulnerable to ischemia and mechanical stress due to their high oxygen demand and unique architectural orientation. The observed reduction in deformation capacity suggests that these fibers may be experiencing the initial stages of fibrotic replacement or ultrastructural damage resulting from repeated bouts of severe exercise without adequate recovery.

This hypothesis is robustly supported by our tissue Doppler imaging results. The hallmark of a truly healthy athlete's heart is a highly compliant ventricle that can fill rapidly and efficiently at high heart rates. The failure of the excessive load group to exhibit the supra-normal diastolic

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relaxation seen in standard athletes points towards an increase in myocardial stiffness. In a clinical context diminished early diastolic relaxation velocities are often the earliest echocardiographic manifestations of restrictive or hypertrophic cardiomyopathies. While the athletes in our study did not present with overt disease the parallel decline in tissue relaxation and longitudinal strain paints a consistent picture of early subclinical myocardial rigidification. This rigidification is likely the macroscopic manifestation of microscopic interstitial collagen deposition a process triggered by repeated exercise-induced inflammatory cascades.

The biomarker profiles of the participants offer a molecular perspective that corroborates the structural and functional imaging data. N-terminal pro-b-type natriuretic peptide is released in response to ventricular stretch and wall tension. While transient elevations are expected immediately following a marathon or ultra-endurance event baseline resting levels should normalize within days. The chronic baseline elevation observed in our excessive load cohort implies that their ventricles are in a state of perpetual mechanical stress even at rest. Similarly the persistent detection of higher levels of high-sensitivity cardiac troponin suggests ongoing microscopic myocardial injury. Whether this troponin release represents true cellular necrosis, increased membrane permeability due to oxidative stress, or enhanced physiological cell turnover remains a subject of intense debate. Regardless of the exact mechanism the consistent elevation of both a stretch marker and an injury marker in the resting state of heavily trained athletes serves as a biochemical red flag for limits of physiological tolerance.

It is imperative to acknowledge the limitations inherent in the current study design. The cross-sectional nature of the research prevents the establishment of definitive causal relationships between the duration of excessive training and the progression of remodeling. We are observing a snapshot in time rather than a longitudinal trajectory. Furthermore the study population was restricted to male athletes to control for gender-specific physiological variations. Female athletes may exhibit different remodeling patterns and hormonal protective mechanisms which necessitate dedicated future investigations. Additionally the definition of excessive load remains somewhat subjective as individual genetic predispositions heavily dictate an athlete's ability to tolerate and recover from extreme physical stress. What constitutes an excessive burden for one individual might fall within the adaptive range for an elite genetic outlier.

Despite these limitations the clinical implications of our findings are substantial. The reliance on standard two-dimensional echocardiography and resting electrocardiograms is inadequate for screening athletes engaging in extreme training volumes. By the time an ejection fraction drops or gross diastolic dysfunction appears the window for preventive intervention has likely closed. Sports medicine practitioners must integrate advanced imaging modalities such as speckle tracking for deformation analysis alongside routine serial monitoring of cardiac biomarkers to detect the earliest deviations from healthy adaptation. When an athlete exhibits a combination of declining global longitudinal strain, shifting geometric patterns, and elevated resting biomarkers a proactive approach involving enforced rest periods, nutritional optimization, and temporary volume reduction is warranted to allow myocardial recovery and prevent the potential transition into irreversible cardiomyopathy or arrhythmogenic states.

Conclusion

The pursuit of athletic excellence through ever-increasing training volumes carries hidden cardiovascular risks that challenge the traditional paradigm of exercise as a universally benign intervention. This study demonstrates that excessive physical load unaccompanied by adequate physiological recovery initiates early signs of maladaptive cardiac remodeling. These changes manifest subtly as a shift toward concentric ventricular geometry, microscopic impairments in longitudinal contraction and diastolic relaxation, and chronic subclinical elevations of

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myocardial stress and injury biomarkers. Because standard diagnostic tools often fail to detect these early alterations the implementation of a comprehensive multi-parametric screening protocol including myocardial strain imaging and precise serological tracking is essential for high-performance populations. Recognizing these early signs of cardiac fatigue is paramount for developing personalized training and recovery strategies that maximize athletic performance while safeguarding long-term cardiovascular health.

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