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Hypertrophic Cardiomyopathy in Athletes: A Paradigm Shift in Exercise Safety, the Role of Comorbid Factors, and the Impact of Anabolic Steroids - A Literature Review

Authors:

Jakubowska Martyna
0009-0008-7234-5178
m.jakubowska2003@gmail.com
Medical University of Białystok, ul. Jana Kilińskiego 1, 15-089 Białystok, Poland

Jakubowska Paulina
0009-0007-6376-3135
paula.bialystok@wp.pl
Medical University of Białystok, ul. Jana Kilińskiego 1, 15-089 Białystok, Poland

Szaryński Mikołaj
0009-0001-7344-8020
mikolaj.szarynski@gmail.com
Medical University of Białystok, ul. Jana Kilińskiego 1, 15-089 Białystok, Poland

Gólcz Julia
0009-0009-3424-4082
golczjulia@gmail.com
Medical University of Białystok, ul. Jana Kilińskiego 1, 15-089 Białystok, Poland

Kadłubowska Karolina
0009-0006-5588-1329
karolina.kadlubowskaa@gmail.com
Medical University of Białystok, ul. Jana Kilińskiego 1, 15-089 Białystok, Poland

Bursztyn Michał
0009-0004-7566-6859
bursztyn82@gmail.com
Medical University of Białystok, ul. Jana Kilińskiego 1, 15-089 Białystok, Poland

Gólcz Adrian
0009-0009-8831-7398
adriangolcz@gmail.com
Medical University of Białystok, ul. Jana Kilińskiego 1, 15-089 Białystok, Poland

Chojnowska Natalia
0009-0004-8714-1726
nataliachojnowska@poczta.fm
Medical University of Białystok, ul. Jana Kilińskiego 1, 15-089 Białystok, Poland

Rytel Jan
0009-0006-0831-9990
janryt@poczta.fm
Medical University of Białystok, ul. Jana Kilińskiego 1, 15-089 Białystok, Poland

Bursztyn Tomasz
0009-0003-1862-1155
tomasz.bursztyn03@gmail.com
Medical University of Rzeszów, al. Tadeusza Rejtana 16c, 35-959 Rzeszów

Sikorski Adam
0009-0006-8463-3957
adam.sikorski515@gmail.com
Medical University of Gdańsk, M. Skłodowskiej-Curie Street 3a, 80-210 Gdańsk, Poland

Abstract

Introduction:

Hypertrophic cardiomyopathy (HCM) is the most common monogenic heart disease and one of the leading causes of sudden cardiac death in young athletes. Recent data have significantly changed the traditional approach to exercise safety in this population. At the same time, the emergence of targeted therapy such as mavacamten and the growing awareness of the cardiotoxic effects of anabolic-androgenic steroids (AAS) introduce new diagnostic and therapeutic challenges.

Materials and methods:

A narrative, critical literature review was conducted, covering high-methodological-quality publications from 2020–2024. The analysis was based on:

• the ESC cardiomyopathy guidelines (2023),

• meta-analyses and systematic reviews concerning the pathophysiology of exercise and new therapies,

• expert papers on the cardiotoxicity of AAS and their imaging manifestations.

From each source, key data were extracted and synthesized into a thematic summary.

Literature review:

Current evidence indicates that in appropriately selected patients with HCM, participation in moderate, and in some cases even intensive, physical activity may be safe provided that regular cardiological follow-up is maintained. Advanced cardiac magnetic resonance has become a key tool in differentiating physiological from pathological hypertrophy and in assessing the risk of sudden cardiac death. Mavacamten therapy significantly improves exercise capacity and reduces the left ventricular outflow tract gradient. At the same time, numerous studies confirm that the use of AAS increases myocardial fibrosis, raises arrhythmogenicity, and may mimic the HCM phenotype, complicating the diagnostic process.

Summary:

Contemporary data support moving away from a restrictive ban on physical activity in favor of individually tailored recommendations based on a multifaceted risk assessment. At the same time, the negative impact of AAS and the presence of comorbidities highlight the need for comprehensive, interdisciplinary care for patients with HCM. Advances in imaging diagnostics, pharmacotherapy, and long-term monitoring enable a safer and more flexible approach to physical activity in this patient group.

Keywords:

hypertrophic cardiomyopathy; athletes; physical exercise; sudden cardiac death; cardiac magnetic resonance; mavacamten; anabolic-androgenic steroids; athlete’s heart; risk stratification.

1. Introduction

Hypertrophic cardiomyopathy (HCM) remains one of the most common hereditary heart diseases and a leading cause of sudden cardiac death in young athletes. Advances in diagnostics, including more precise risk stratification models and new recommendations regarding physical activity, have significantly changed the way exercise safety is assessed in this population. The current ESC guidelines emphasize the need for an individualized approach to athletes with HCM, taking into account both the arrhythmic risk profile and therapeutic options. At the same time, epidemiological data indicate that appropriate qualification and monitoring may reduce the risk of sudden cardiac events in physically active individuals. In light of these changes, the analysis of clinical, behavioral, and environmental factors that influence the course of the disease and the long-term prognosis of athletes with HCM is becoming increasingly important [1,2].

2. Materials and methods

A narrative literature review was conducted, covering publications from 2020–2024, focusing on studies with high methodological quality. The 2023 ESC guidelines, results of prospective studies, meta-analyses, and publications concerning the effects of physical exercise, modern imaging diagnostics, targeted therapy, and the consequences of AAS use were included. From each source, key results, methods, population characteristics, and authors' conclusions were extracted. They were then synthesized into coherent thematic areas, taking into account points where new data redefine previous recommendations.

3. Literature review

3.1. Diagnostics and SCD risk stratification in light of the 2023 ESC guidelines

The 2023 ESC guidelines assign particular importance to advanced imaging techniques, especially cardiac magnetic resonance (CMR), which is crucial in differentiating HCM from physiological

hypertrophy in athletes. It is recommended to assess not only the qualitative presence of LGE, but also its quantitative analysis (percentage of LV mass) as well as T1/T2 mapping and ECV measurements.

A key element remains the multiparametric stratification of SCD risk, in which the formal HCM Risk-SCD scale constitutes only one of the components. The guidelines emphasize the primary importance of the "major" risk factors, such as:

- extreme hypertrophy (≥30 mm),
- extensive LGE ≥15%,
- NSVT,
- EF <50%,
- apical aneurysm,
- positive family history of SCD.

The inclusion of a complete multimodal assessment is a necessary condition for any sports eligibility evaluation [1].

3.2. Evolution of the exercise-safety paradigm: from prohibition to individualized assessment

The revision of the traditionally restrictive approach to physical activity in patients with hypertrophic cardiomyopathy constitutes a key element of the latest guidelines of international cardiology societies. The 2024 AHA/ACC guidelines introduce a formal qualification pathway which—based on multiparametric risk assessment—allows consideration of participation in high-intensity sports even in stable patients with an implanted cardioverter-defibrillator (ICD), provided its function is normal and no other major risk factors are present [3].

In parallel, the 2023 ESC guidelines emphasize that any decision regarding activity must be preceded by a comprehensive, multimodal evaluation, taking into account especially the results of advanced cardiac magnetic resonance imaging to exclude subclinical myocardial injury [1].

Both documents are supported by the results of contemporary observational studies, such as LIVE-HCM and REST, which did not demonstrate an increased risk of adverse events associated with intense exercise in carefully selected patients under strict supervision.

This model finds additional pathophysiological justification in studies assessing exercise adaptation of the heart in HCM. A study using detailed echocardiographic analysis showed that in low-risk HCM patients, the hemodynamic response to maximal exercise is normal, and abnormalities in coronary flow reserve or diastolic function are not common and occur mainly in individuals with advanced disease [6]. This suggests that fundamental exercise physiology may be preserved in a significant proportion of patients with HCM, supporting the concept of individualized rather than universal disqualification.

An integrated approach that combines clinical assessment, imaging, and monitoring data thus enables a shift from a rigid prohibition model toward an individualized strategy in which safe activity becomes an achievable therapeutic goal.

3.3. The impact of targeted therapy and physical training on exercise capacity in HCM

Contemporary management of hypertrophic cardiomyopathy is evolving towards combining advanced pharmacotherapy with supervised physical activity, which is reflected in the latest studies. A breakthrough in the treatment of symptomatic, obstructive forms of the disease is the introduction of mavacamten—the first allosteric myosin inhibitor in its class. As indicated by the review of Braunwald and colleagues (2023), this drug, through direct action on the molecular mechanism of excessive sarcomere contractility, effectively reduces the left ventricular outflow tract (LVOT) gradient. This cardinal change in hemodynamics translates into a significant improvement in exercise tolerance and quality of life of patients, representing the first targeted therapeutic option with proven efficacy [7].

In parallel, the role of supervised physical training as a safe and effective adjunct to treatment is strongly supported by randomized clinical trials. Exercise programs for patients with HCM are safe and lead to significant improvements in physical fitness. Both moderate and high-intensity training in stable patients results in improved physical performance parameters, including peak oxygen uptake (VO_2peak), and favorably modifies the cardiovascular response to exercise. Collectively, these data support a holistic management model in which targeted therapy alleviating the underlying contractility defect (mavacamten) is synergistically complemented by personalized training programs aimed at improving exercise adaptation and functional capacity [8,9].

3.4. Anabolic-androgenic steroid (AAS)-induced cardiomyopathy as a clinical challenge

Chronic use of anabolic-androgenic steroids (AAS) constitutes a significant, non-genetic risk factor for the development of cardiomyopathy with a phenotype resembling hypertrophic cardiomyopathy (HCM), referred to as AAS-induced cardiomyopathy (AASIC). This pathology, being a separate clinical entity, poses a serious diagnostic challenge due to its morphological similarity to other forms of left ventricular hypertrophy, including HCM and physiological athlete's heart. Key pathophysiological mechanisms of

AASIC include the direct, receptor-mediated effect of androgens on cardiomyocytes, leading to their hypertrophy, as well as induction of myocardial fibrosis, endothelial dysfunction, and autonomic disturbances, which ultimately result in impaired diastolic function and create a substrate for arrhythmias [5,10].

Experimental data provide insight into the long-term consequences of AAS exposure, indicating that administration already during adolescence can induce permanent, adverse cardiac remodeling, manifested as hypertrophy and increased myocardial susceptibility to ischemia-reperfusion injury in adulthood [11].

Clinically, the potential interaction of AAS with subclinical or overt HCM is particularly dangerous. In individuals with a genetic predisposition, AAS may act as a trigger or accelerate the manifestation and progression of the disease, leading to the creation of a high-risk phenotype combining features of both pathologies. Therefore, in the differential diagnosis of unexplained LV hypertrophy in athletes or physically active individuals, especially young men, it is imperative to include a detailed history of performance-enhancing drug use and the application of advanced imaging techniques, such as cardiac magnetic resonance with fibrosis assessment (LGE), to distinguish AASIC from other causes [5].

3.5: Comorbidities, complications, and long-term perspective in HCM

Hypertrophic cardiomyopathy (HCM) is associated with the risk of developing significant, prognostically burdensome complications. A key challenge is atrial fibrillation (AF), which affects a substantial proportion of patients and constitutes an independent risk factor for increased all-cause mortality and ischemic stroke [12].

Furthermore, the natural progression in some patients involves the development of heart failure with preserved ejection fraction (HFpEF), which is associated with poorer hemodynamic parameters, advanced myocardial fibrosis, and increased hospitalization rates [13].

The most serious, albeit rarer, complication remains sudden cardiac death (SCD), which in athletes with HCM is most often caused by rapid ventricular arrhythmia on the background of structural and electrical disease substrate, such as significant hypertrophy, extensive scarring (LGE), or latent obstruction [2].

The presence of comorbidities, such as arterial hypertension, obesity, or coronary artery disease, accelerates this adverse cardiac remodeling. They exacerbate diastolic dysfunction, impair microcirculatory perfusion, and act additively on arrhythmic risk, creating a high-risk phenotype. From a long-term perspective, the prognosis of a patient with HCM therefore depends on the synergistic influence of the underlying myocardial pathology, the presence and control of complications (AF, HFpEF), and modifiable cardiovascular risk factors.

4. Summary

Hypertrophic cardiomyopathy in athletes remains a complex and multidimensional clinical problem, requiring individual risk assessment and an interdisciplinary approach. With careful and multi-step risk stratification, participation in moderate, and in selected cases even intensive, physical activity can be safe. Thus, the traditionally restrictive model is being replaced by an approach based on evidence and precise personalization of recommendations.

The introduction of new diagnostic tools, particularly advanced cardiac magnetic resonance imaging, enables precise differentiation of HCM from physiological hypertrophy in athletes and more accurate assessment of the risk associated with arrhythmias and sudden cardiac death. Equally significant progress concerns treatment, where the emergence of targeted therapy—especially the myosin inhibitor mavacamten—has markedly improved quality of life and physical performance in patients with obstructive forms of the disease.

An important risk factor in athletes is the use of anabolic-androgenic steroids. These substances can lead to severe myocardial fibrosis, increased arrhythmogenicity, and accelerated progression of heart failure. Therefore, any decisions regarding physical activity in athletes with HCM must consider a reliable assessment of exposure to AAS.

From a long-term perspective, comprehensive patient management is crucial, including control of comorbidities such as arterial hypertension, metabolic disorders, or obesity, which can exacerbate disease progression and increase the risk of complications. Regular, multidisciplinary care—especially in centers specializing in cardiomyopathies—remains the foundation for safe functioning of patients and allows for better prediction of their prognosis.

Disclosure
Author's contribution
Conceptualization: Martyna Jakubowska, Paulina Jakubowska,

Methodology: Michał Bursztyn, Tomasz Bursztyn
Formal analysis: Natalia Chojnowska, Paulina Jakubowska, Karolina Kadłubowska
Investigation: Jan Rytel , Karolina Kadłubowska, Adam Sikorski
Writing-rough preparation: Mikołaj Szaryński, Natalia Chojnowska, Jan Rytel
Writing-review and editing: Mikołaj Szaryński, Michał Bursztyn
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In preparing this work, the authors used ChatGPT for the purpose of checking language accuracy. After using this tool/service, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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