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Differentiating Hypertrophic Cardiomyopathy from Athlete's Heart: A Diagnostic Challenge in Sports Cardiology - A Narrative Review

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Abstract

Background. Long-term athletic training induces structural, functional, and electrical cardiac adaptations known as athlete's heart. In some athletes, these physiological changes overlap with hypertrophic cardiomyopathy, creating a diagnostic grey zone.

Aim. The objective of this narrative review was to discuss current approaches to differentiating hypertrophic cardiomyopathy from athlete's heart in sports cardiology.

Materials and methods. Publications available in PubMed and Scopus from 2017–2026 were reviewed. The final set comprised 30 freely accessible full-text sources, including narrative reviews, systematic reviews, imaging studies, consensus statements, and clinical guidelines.

Results. Differentiation requires integration of clinical history, family history, ECG, echocardiography, cardiac magnetic resonance, rhythm monitoring, genetic testing, exercise assessment, and longitudinal observation. Athlete's heart is usually supported by proportional chamber enlargement, preserved function, high exercise capacity, and absence of myocardial fibrosis. Hypertrophic cardiomyopathy is suggested by disproportionate or focal hypertrophy, small left ventricular cavity, abnormal ECG findings, arrhythmias, fibrosis, symptoms, or positive family history.

Conclusions. A multimodal and individualized diagnostic strategy is essential to reduce sudden cardiac death risk while avoiding unnecessary restriction of healthy athletes from sport.

Keywords: hypertrophic cardiomyopathy, athlete's heart, sports cardiology, cardiac remodeling, echocardiography, cardiac magnetic resonance, sudden cardiac death

1. Introduction

Regular physical exercise is associated with major cardiovascular benefits, including improved aerobic capacity, autonomic regulation, endothelial function, and cardiometabolic profile. In athletes exposed to high training volumes over many years, the cardiovascular system may develop structural, functional, and electrical adaptations collectively referred to as athlete's heart. These changes may include resting sinus bradycardia, increased vagal tone, chamber enlargement, increased myocardial mass, enhanced stroke volume, and selected training-related electrocardiographic findings (Sharma et al. 2018; Palermi et al. 2023; Flanagan et al. 2023).

Athlete's heart is generally considered a physiological response to repeated hemodynamic loading. However, some features of athletic adaptation may overlap with early or mild inherited cardiomyopathies, particularly hypertrophic cardiomyopathy (HCM). HCM is a primary myocardial disease most commonly related to pathogenic variants in sarcomeric genes and characterized by unexplained left ventricular hypertrophy. It may remain asymptomatic or present with exertional dyspnea, chest pain, syncope, atrial fibrillation, ventricular arrhythmias, left ventricular outflow tract obstruction, heart failure, or sudden cardiac death (Marian and Braunwald 2017; Bonaventura et al. 2021; Ommen et al. 2024).

The differentiation between athlete's heart and HCM is especially challenging when left ventricular wall thickness is borderline. In this diagnostic grey zone, interpretation should not rely on wall thickness alone but should also consider sex, ethnicity, body size, sport discipline, training history, physical examination, symptoms, ECG assessment, and cardiovascular imaging findings (Malhotra and Sharma 2017; Lander et al. 2021; Pagourelias et al. 2025).

Accurate diagnosis is clinically important. Underdiagnosis of HCM may expose an athlete to preventable adverse events, whereas overdiagnosis may lead to unnecessary restriction from sport, psychological distress, and inappropriate medical labeling. Therefore, modern sports cardiology requires an individualized, multimodal approach that may include clinical assessment, ECG, echocardiography, cardiac magnetic resonance, exercise testing, rhythm monitoring, genetic testing, and longitudinal reassessment when uncertainty persists (Basu and Malhotra 2018; Maestrini et al. 2020; Kübler et al. 2021; Pelliccia et al. 2021; Ali et al. 2024; Kim et al. 2025).

Given this diagnostic overlap, the main research problem addressed in this narrative review is how clinicians can distinguish physiological cardiac remodeling from early or mild

HCM in athletes with borderline or ambiguous findings. Therefore, the objective of this review was to discuss the differentiation between hypertrophic cardiomyopathy and athlete's heart in competitive athletes and physically active individuals, with particular attention to diagnostic tools and return-to-play decision-making.

2. Research materials and methods

This narrative review was based on a literature search performed in the PubMed and Scopus databases. The search aimed to identify publications concerning the distinction between hypertrophic cardiomyopathy and athlete's heart, with emphasis on physiological cardiac remodeling, diagnostic grey zones, ECG interpretation, echocardiographic assessment, CMR, genetic evaluation, exercise testing, rhythm monitoring, detraining, and return-to-play decisions. Publications from September 2017 to January 2026 were considered.

The search strategy included the following terms used alone and in combination: "hypertrophic cardiomyopathy," "HCM," "athlete's heart," "athletic heart syndrome," "cardiac remodeling," "exercise-induced cardiac remodeling," "left ventricular hypertrophy," "sports cardiology," "diagnostic grey zone," "differential diagnosis," "ECG," "electrocardiography," "echocardiography," "cardiac magnetic resonance," "CMR," "late gadolinium enhancement," "genetic testing," "exercise testing," "Holter monitoring," "detraining," "sudden cardiac death," "sports eligibility," and "return to play."

Eligible sources included English-language original research articles, narrative reviews, systematic reviews, imaging studies, consensus documents, and clinical guidelines that discussed athlete's heart, HCM, cardiovascular screening in athletes, or diagnostic differentiation between physiological and pathological cardiac remodeling. Publications were excluded if they were not written in English, were published outside the selected time frame, were available only as conference abstracts, editorials, or opinion pieces, lacked full-text availability, or did not directly address the topic of this review.

3. Research results

3.1. Athlete's Heart as Physiological Cardiac Remodeling

Athlete's heart describes structural, functional, and electrical adaptations resulting from repeated long-term training. The magnitude and pattern of remodeling depend on training volume, sport discipline, sex, age, body size, ethnicity, and genetic background. Endurance

sports are usually associated with volume loading and chamber enlargement, whereas strength-oriented sports may add pressure-loading components. Because many disciplines combine dynamic and static exercise, interpretation should be based on the actual training profile rather than sport category alone (Flanagan et al. 2023; Palermi et al. 2023; Zholshybek et al. 2023).

Physiological remodeling is usually proportionate. Athletes may show enlarged left ventricular cavity size, increased myocardial mass, right ventricular and atrial enlargement, preserved systolic function, and normal or enhanced diastolic function. Mild wall thickening may be physiological when accompanied by large cavity size, balanced chamber enlargement, and absence of fibrosis or other pathological findings. Disproportionate hypertrophy, focal thickening, small cavity size, impaired relaxation, or myocardial fibrosis should prompt further evaluation (Malhotra and Sharma 2017; Kübler et al. 2021; Ali et al. 2024; Palmisano et al. 2021).

3.2. Hypertrophic Cardiomyopathy in Athletes

Hypertrophic cardiomyopathy is a primary myocardial disease commonly related to pathogenic variants in sarcomeric genes and characterized by unexplained left ventricular hypertrophy. Its pathophysiology may include myocyte hypertrophy and disarray, interstitial fibrosis, microvascular dysfunction, impaired relaxation, and arrhythmogenic substrate. Clinical expression is heterogeneous: some individuals remain asymptomatic, whereas others develop dyspnea, chest pain, palpitations, syncope, atrial fibrillation, ventricular arrhythmias, left ventricular outflow tract obstruction, heart failure, or sudden cardiac death (Marian and Braunwald 2017; Bonaventura et al. 2021; Ommen et al. 2024).

HCM is relevant in sports cardiology because mild disease may be compatible with high exercise capacity, while the condition remains associated with sudden cardiac death in young athletes. Morphology may include asymmetric septal, apical, concentric, or focal hypertrophy, with or without obstruction. Imaging may reveal mitral valve abnormalities, abnormal papillary muscles, left atrial enlargement, apical aneurysm, or fibrosis. These features explain why HCM cannot be assessed by wall thickness alone and often requires multimodality evaluation (Malhotra and Sharma 2017; Lander et al. 2021; Ghani et al. 2023; Kübler et al. 2021; Pagourelias et al. 2025).

3.3. The Diagnostic Grey Zone

The diagnostic grey zone refers to overlap between physiological athletic remodeling and mild or early HCM, particularly in athletes with borderline left ventricular wall thickness. In athlete's heart, hypertrophy is usually accompanied by proportionate chamber enlargement, preserved or enhanced diastolic function, high exercise capacity, and remodeling consistent with training background. HCM becomes more likely when hypertrophy is focal, asymmetric, disproportionate, associated with small cavity size, impaired diastolic function, or myocardial fibrosis on Cardiac Magnetic Resonance (CMR) (Malhotra and Sharma 2017; Lander et al. 2021; Flanagan et al. 2023; Ali et al. 2024; Pagourelias et al. 2025).

The interpretation of grey-zone findings must account for sex, ethnicity, body size, and sport discipline. Athletes of African or Afro-Caribbean ancestry may show greater physiological hypertrophy and more frequent repolarization changes, whereas female athletes generally have lower absolute wall thickness values. Standard population thresholds may therefore increase both overdiagnosis and underdiagnosis if applied without athlete-specific context (Ozo and Sharma 2020; Davis et al. 2022; Palermi et al. 2023).

3.4. Clinical History, Family History, and Physical Examination

Clinical evaluation remains essential. Exertional syncope, unexplained presyncope, chest pain, disproportionate dyspnea, palpitations, or reduced exercise tolerance should not be attributed automatically to training, particularly when combined with abnormal ECG or imaging findings. Symptoms during exertion are generally more concerning than post-exertional collapse, although both require clinical judgment (Pelliccia et al. 2021; Lampert et al. 2024; Ommen et al. 2024).

Family history of HCM, unexplained sudden death, premature cardiovascular death, implantable cardioverter-defibrillator implantation, or inherited arrhythmia syndromes increases suspicion of genetic cardiovascular disease. Physical examination may be normal, but a dynamic systolic murmur may suggest left ventricular outflow tract obstruction. Blood pressure measurement, training history, sport discipline, cumulative load, supplement use, possible anabolic-androgenic steroid exposure, and previous cardiac testing should be included in the assessment (Gray and Semsarian 2020; Bonaventura et al. 2021; Malhotra and Sharma 2017; Pelliccia et al. 2021; Ommen et al. 2024).

3.5. Electrocardiography in Differentiation

Resting 12-lead ECG is a central tool in athlete evaluation. Training-related findings may include sinus bradycardia, sinus arrhythmia, first-degree atrioventricular block, early repolarization, and isolated voltage criteria for left ventricular hypertrophy. In contrast, deep T-wave inversion, ST-segment depression, pathological Q waves, left bundle branch block, ventricular pre-excitation, prolonged QT interval, Brugada-like patterns, or frequent ventricular ectopy require further evaluation (Sharma et al. 2018; Basu and Malhotra 2018; Fanale et al. 2024; Finocchiaro et al. 2026).

Athlete-specific ECG interpretation criteria reduce false-positive findings while maintaining sensitivity for relevant disease. Repolarization abnormalities, especially lateral or inferolateral T-wave inversion, may precede clear structural expression of cardiomyopathy and should prompt further evaluation even if echocardiography is normal. This may include CMR, Holter monitoring, exercise testing, genetic evaluation, and follow-up (Sheikh et al. 2018; Bernardini et al. 2023; Caramoci et al. 2025; Finocchiaro et al. 2026).

3.6. Echocardiography and Functional Assessment

Transthoracic echocardiography is usually the first-line imaging test in athletes with suspected structural heart disease. It allows assessment of wall thickness, distribution of hypertrophy, cavity size, systolic and diastolic function, atrial and right ventricular size, valve morphology, and left ventricular outflow tract gradients. A large cavity with proportionate wall thickening favors athlete's heart, whereas increased wall thickness with a small or non-dilated cavity raises suspicion of HCM (Malhotra and Sharma 2017; Flanagan et al. 2023; Ali et al. 2024).

Echocardiographic interpretation should integrate maximal wall thickness, relative wall thickness, left ventricular end-diastolic diameter, left atrial size, Doppler and tissue Doppler indices, and strain. HCM may show impaired relaxation or abnormal strain despite preserved ejection fraction, while athlete's heart usually shows preserved or enhanced function. Speckle-tracking echocardiography may add value but should remain an adjunct rather than a stand-alone diagnostic criterion (Flanagan et al. 2023; Ali et al. 2024; Ommen et al. 2024).

3.7. Cardiac Magnetic Resonance Imaging

CMR is particularly useful when echocardiography is inconclusive, image quality is limited, or focal, apical, or fibrotic disease is suspected. It accurately quantifies ventricular volumes, wall thickness, myocardial mass, ejection fraction, and right ventricular morphology. In the grey zone, CMR helps assess whether myocardial mass and chamber volumes are proportionate and whether focal hypertrophy, apical disease, or tissue abnormalities are present (Maestrini et al. 2020; Czimbalmos et al. 2019; Kübler et al. 2021; Zholshybek et al. 2023).

In athlete's heart, CMR typically shows balanced chamber enlargement, proportionate myocardial mass, and absence of pathological fibrosis. In HCM, it may reveal asymmetric or focal hypertrophy, apical hypertrophy, myocardial crypts, abnormal papillary muscles, apical aneurysm, or replacement fibrosis. LGE supports pathological remodeling in the appropriate context, and T1 mapping or extracellular volume quantification may add information about diffuse fibrosis (Maestrini et al. 2020; Małek et al. 2022; Grech et al. 2025; Pagourelias et al. 2025).

3.8. Genetic Testing and Family Screening

Genetic testing can support diagnosis in athletes with suspected HCM, especially when borderline imaging findings, abnormal ECG, or positive family history are present. A pathogenic or likely pathogenic sarcomeric variant may confirm a genetic substrate and enable cascade screening in relatives. However, a negative test does not exclude HCM, and variants of uncertain significance should not be used alone to restrict sports participation (Gray and Semsarian 2020; Bonaventura et al. 2021; Ommen et al. 2024).

Genotype-positive but phenotype-negative athletes require individualized assessment rather than automatic exclusion from sport. Risk assessment should consider phenotype expression, symptoms, arrhythmia burden, family history, imaging findings, sport demands, and athlete preferences. Genetic testing therefore complements but does not replace clinical and imaging-based evaluation (Pelliccia et al. 2021; Kim et al. 2025; Ommen et al. 2024).

3.9. Exercise Testing, Rhythm Monitoring, and Detraining

Exercise testing provides functional information beyond resting imaging. In athlete's heart, exercise capacity is usually high, blood pressure response is appropriate, and significant arrhythmias are absent. In HCM, abnormal blood pressure response, exercise-induced symptoms, reduced functional capacity, or ventricular arrhythmias may support pathological

interpretation. Cardiopulmonary exercise testing may be useful, although high peak oxygen uptake does not exclude mild HCM (Malhotra and Sharma 2017; Pelliccia et al. 2021; Ommen et al. 2024; Gray et al. 2025).

Ambulatory ECG monitoring helps detect intermittent arrhythmias such as non-sustained ventricular tachycardia, frequent ventricular ectopy, complex ventricular arrhythmias, or atrial fibrillation. Detraining may be considered when diagnostic uncertainty persists, because physiological remodeling may regress after reduced training load, whereas HCM is less likely to regress substantially. However, detraining should be interpreted together with ECG, echocardiography, CMR, clinical history, and family history (Lampert et al. 2024; Lander et al. 2021; Palermi et al. 2023; Ommen et al. 2024).

3.10. Return-to-Play and Shared Decision-Making

Return-to-play decisions in athletes with suspected or confirmed HCM should be individualized. Contemporary recommendations have shifted away from automatic restriction toward risk assessment, shared decision-making, and longitudinal follow-up. Confirmed athlete's heart should not lead to unnecessary restriction, whereas definite HCM requires education, risk stratification, emergency planning, family screening, and regular follow-up. Borderline cases may require temporary restriction until uncertainty is resolved (Pelliccia et al. 2021; Ommen et al. 2024; Lampert et al. 2024; Kim et al. 2025).

Shared decision-making should include explanation of diagnosis, uncertainty, potential consequences, warning symptoms, emergency planning, follow-up requirements, and sport-specific demands. The athlete's values are considered, but medical teams remain responsible for risk communication, documentation, and appropriate referral when specialist sports cardiology evaluation is needed (Lampert et al. 2024; Kim et al. 2025).

3.11. Practical Diagnostic Pathway

A practical diagnostic pathway should begin with symptoms, family history, physical findings, previous testing, and training history. ECG and echocardiography are usually the next steps. If findings are consistent with physiological remodeling and there are no symptoms or concerning family history, routine follow-up may be sufficient. If ECG is abnormal, wall thickness is borderline, cavity size is small, diastolic function is impaired, or symptoms are present, further evaluation may include CMR, exercise testing, ambulatory rhythm monitoring, and genetic

counseling (Sharma et al. 2018; Basu and Malhotra 2018; Flanagan et al. 2023; Pelliccia et al. 2021; Palermi et al. 2023; Ommen et al. 2024).

CMR should be considered when echocardiography is inconclusive or when focal, apical, or fibrotic disease is suspected. Genetic testing should be reserved for athletes with clinical suspicion of inherited disease. If uncertainty persists after complete evaluation, longitudinal follow-up helps determine whether the phenotype is stable, progressive, or reversible (Maestrini et al. 2020; Kübler et al. 2021; Małek et al. 2022; Gray and Semsarian 2020; Pagourelias et al. 2025).

3.12. Clinical and Psychological Implications

A diagnosis of HCM or prolonged diagnostic uncertainty may affect an athlete's identity, career, education, emotional well-being, and motivation. Clinicians should communicate clearly, avoid unnecessary alarm, and explain why further testing or temporary restriction may be needed. Reassurance must be evidence-based, especially when athletes face pressure from competition, coaches, clubs, or organizations (Pelliccia et al. 2021; Ommen et al. 2024; Kim et al. 2025).

Shared decision-making is essential when evidence is limited or risk is not zero but may be acceptable after counseling. It should remain a guided clinical process rather than a transfer of responsibility to the athlete. Transparent communication, documentation, and follow-up planning help balance safety with preservation of athletic participation (Pelliccia et al. 2021; Lampert et al. 2024; Kim et al. 2025).

3.13. Broader Differential Diagnosis in Athletes with Left Ventricular Hypertrophy

Although this review focuses on HCM and athlete's heart, left ventricular hypertrophy in athletes has a broader differential diagnosis. It may result from physiological adaptation, HCM, hypertension, aortic valve disease, infiltrative or storage disorders, myocarditis-related remodeling, or performance-enhancing substances. Masters athletes may also have age-related cardiovascular risk factors such as hypertension or coronary artery disease (Pelliccia et al. 2021; Ommen et al. 2024; Pagourelias et al. 2025).

Hypertension may coexist with athletic training and produce mixed remodeling. Aortic valve disease should be considered when auscultation or echocardiography is abnormal. Myocarditis and post-myocarditis scarring may produce ECG abnormalities, arrhythmias,

reduced performance, or LGE on CMR. Anabolic-androgenic steroids and other performance-enhancing substances may influence blood pressure, myocardial mass, lipid profile, thrombosis risk, and arrhythmia susceptibility (Maestrini et al. 2020; Grech et al. 2025; Pelliccia et al. 2021; Lampert et al. 2024).

3.14. Follow-Up Strategy and Longitudinal Reassessment

Longitudinal reassessment is essential when initial findings do not clearly support either athlete's heart or HCM. Physiological adaptation is usually stable or partially reversible with reduced training load, whereas HCM may persist, progress, or show emerging abnormalities. Follow-up should be individualized and may include repeat ECG, echocardiography, ambulatory rhythm monitoring, exercise testing, CMR, or genetic reassessment depending on the level of suspicion (Pelliccia et al. 2021; Lander et al. 2021; Palermi et al. 2023; Lampert et al. 2024; Ommen et al. 2024; Pagourelias et al. 2025).

A structured follow-up plan should define what will be monitored, when reassessment will occur, and which changes would alter management. Documentation should include symptoms, family history, training load, ECG interpretation, imaging measurements, rhythm findings, and the reasoning behind sport eligibility decisions. Athletes should be educated to report exertional syncope, new palpitations, unexplained decline in performance, chest pain, or disproportionate dyspnea (Gray and Semsarian 2020; Ommen et al. 2024; Kim et al. 2025).

4. Discussion

The reviewed literature demonstrates that differentiating HCM from athlete's heart remains one of the most demanding tasks in sports cardiology. The problem is not limited to measuring left ventricular wall thickness. Diagnosis requires interpretation of morphology, function, electrical findings, tissue characterization, genetic background, symptoms, family history, and longitudinal change.

Wall thickness is an important starting point but not a stand-alone diagnostic marker. The same measurement may have different implications depending on body size, sex, ethnicity, training history, sport type, ECG pattern, cavity size, and CMR findings. A mildly increased wall thickness in a large endurance athlete with enlarged ventricular cavity, normal diastolic function, normal ECG, no symptoms, and no family history is more likely to reflect physiological remodeling. The same measurement with small cavity size, inferolateral T-wave

inversion, family history of sudden death, or LGE on CMR should raise concern for HCM (Malhotra and Sharma 2017; Kübler et al. 2021; Flanagan et al. 2023).

ECG and imaging are complementary. Isolated voltage criteria for left ventricular hypertrophy are common and usually benign in trained athletes, whereas pathological Q waves, ST-segment depression, deep T-wave inversion, and complex ventricular ectopy require further evaluation. Echocardiography remains the first-line imaging method, while CMR is especially useful in the diagnostic grey zone because it can identify focal or apical hypertrophy, quantify ventricular volumes, assess the right ventricle, and detect myocardial fibrosis (Sharma et al. 2018; Basu and Malhotra 2018; Maestrini et al. 2020; Małek et al. 2022; Finocchiaro et al. 2026).

Genetic testing may support diagnosis and family screening in selected athletes, particularly when clinical findings, family history, ECG, or imaging raise suspicion of inherited disease. However, a negative result does not exclude HCM, and variants of uncertain significance should not be used as definitive evidence of disease or as the sole basis for sports restriction (Gray and Semsarian 2020; Bonaventura et al. 2021; Ommen et al. 2024).

The practical challenge is to avoid both underdiagnosis and overdiagnosis. Underdiagnosis may expose athletes with HCM to avoidable risk, whereas overdiagnosis may unnecessarily remove healthy athletes from sport. Contemporary sports cardiology has therefore shifted toward individualized, multimodal assessment and shared decision-making rather than rigid classification based on a single measurement (Pelliccia et al. 2021; Lampert et al. 2024; Kim et al. 2025).

Overall, differentiation between HCM and athlete's heart should be viewed as a dynamic process rather than a single diagnostic event. The athlete's phenotype may change with age, training load, disease expression, and clinical circumstances. A multimodal, individualized, and longitudinal strategy provides the best balance between safety and preservation of athletic participation.

5. Conclusions

Differentiating hypertrophic cardiomyopathy from athlete's heart is a clinically important challenge in sports cardiology, particularly in athletes with borderline left ventricular hypertrophy. Athlete's heart is usually characterized by proportionate chamber enlargement, preserved or enhanced diastolic function, high exercise capacity, absence of pathological

myocardial fibrosis, and partial reversibility after reduction of training load. HCM should be suspected when left ventricular hypertrophy is disproportionate, asymmetric, associated with small cavity size, impaired diastolic function, abnormal ECG findings, myocardial fibrosis, symptoms, arrhythmias, or positive family history.

Echocardiography remains the first-line imaging tool, but CMR is essential when echocardiographic findings are inconclusive or myocardial tissue characterization is required. ECG, echocardiography, CMR, exercise testing, Holter monitoring, genetic testing, family history, and longitudinal observation should be integrated rather than interpreted separately. Return-to-play decisions should be individualized and based on shared decision-making, specialist evaluation, risk stratification, athlete preferences, and regular follow-up.

A multimodal diagnostic strategy is necessary to reduce both underdiagnosis of potentially life-threatening HCM and unnecessary exclusion of healthy athletes from sport. In athletes with ambiguous findings, the diagnostic process should remain longitudinal and context-specific, rather than based on a single threshold or isolated investigation.

Disclosure

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In preparing this work, the author(s) used ChatGPT for language improvement and grammatical correction. After using this tool/service, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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