



Review Article

Hypertrophic Cardiomyopathy Versus Athlete's Heart: An Updated Review - Implications of MRI

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Abstract

Hypertrophic Cardiomyopathy remains the leading cause of sudden cardiac death worldwide especially in young athletes with a prevalence of 1:500. The importance of screening of HCM, in families with a history of sudden cardiac death is particularly high, in order to avoid complications and ultimately a sudden death. Careful examination in young individuals with positive family history or suspicion of HCM based on the clinical image and clinical examination is highly recommended. With this review, we would like to provide a practical guide and a detailed screening for young athletes in order to exclude hypertrophic cardiomyopathy with Emphasis in MRI Screening.

Keywords: Athletes; Hypertrophic Cardiomyopathy; MRI; Heart.

Introduction

Hypertrophic cardiomyopathy (HCM) is the hypertrophy of the heart muscle of more than 15 mm, without an identifiable cause. It is the most recognized genetic cardiac disease and the most common inherited heart disease, with mutations occurring primarily in the β -myosin heavy chain gene and involve the cardiac myosin-binding protein [1]. The prevalence is 1 per 500 [1], and

remains the most common cause of sudden cardiac death in young population, especially in athletes under 35 years old.

On the other hand, Exercise-induced cardiac remodeling, often termed the "athlete's heart," is an adaptive increase in cardiac chamber size and wall thickness that is promoted by the volumes and pressure loads of exercise. These changes are accompanied by enhancements in lusitropic and contractile function that enable the heart to fill and eject larger volumes than nonathletes during exercise. Maximal heart rates in athletes are similar to nonathletes,

and thus, the greater exercise cardiac outputs observed in athletes are entirely caused by this ability to generate larger stroke volumes [2]. Under this appreciation, usually enlarged atria or enlarged left ventricle may be the primary finding. However, there is a tendency to use androgens and/ or testosterone and in that case, hypertrophy is the leading finding in imaging modality.

Methods that belong to the diagnostic armamentarium, in order to differentiate hypertrophic cardiomyopathy from athletes heart are a 12 canal ECG, echocardiography, magnetic resonance imaging, cardiopulmonary exercise test and last but not least, a careful clinical examination and detailed taking of family history. Upon diagnosis of hypertrophic cardiomyopathy, a genetic screening of first degree relatives is highly recommended.

Electrocardiogram

Normal ECG findings that belong to athletes heart include: 1. Increased QRS Voltage for LVH or RVH, 2. RBBB, 3. Early repolarization/ST Segment Elevation, 4. ST Segment Elevation followed by T Wave inversion in V1-V4 in black athletes, 5. T Wave inversion in V1-V3<16 years old.6. Sinus Bradycardia or sinus Arrhythmia, 7. Ectopic atrial or junctional rhythm, 8.1.degree AV Block, 9. Mobitz I 2.Degree AV-Block [3].

Typical ECG Findings that belong to hypertrophic cardiomyopathy include: 1. QTc Prolongation, 2. Pathological Q Waves, 3. Biphasic P Waves, 4. biphasic T Waves,5. Inverted T Waves, 6. prominent T Wave, 7. Fragmented R Wave, 8. Fragmented P wave, 9. Atrial fibrillation [4].

However, there are some borderline ecg finding that require careful clarification. These are: 1. Left axis deviation, 2. Left atrial enlargement, 3. Right axis deviation, 4. Right atrial enlargement, 5. Complete RBBB [5]

Echocardiography

Concerning athletes heart, due to intensive and prolonged training, especially under use of androgens and / or testosterone, the typical echocardiographic finding is an enlarged heart and hypertrophy. Usually, there is a reversal of the hypertrophy under cessation for 6 months. In any other case, always in combination with clinical and family history, further clarification is highly indicated. In hypertrophic cardiomyopathy, Maximal wall thickness > 15 mm in any myocardial segment is the criterion according to definition. In some cases, patients with positive genotype may have normal wall thickness.

All walls of the left ventricle should be assessed, from base to apex. Moreover, therapeutic strategies are largely dependent on the presence or absence of obstruction, as well as of symptoms. Almost 30% of patients will have rest obstruction (≥ 30 mm Hg).

Another 30% will have physiologically provoked gradients, e.g., after eating, and alcohol consumption [6] (< 30 mm Hg at rest and ≥ 30 mm Hg) [7] (Figure 1)

The final one-third will have the non-obstructive form of HCM. Gradients more than 50 mm Hg represent the threshold for intervention, in case of failing to ameliorate symptoms under medications.

During the last two decades, two-dimensional speckle tracking echocardiography, a new angle-independent imaging modality, has been employed for evaluating myocardial deformation indices in patients with HCM. A strong inverse correlation between left ventricular (LV) myocardial strain and the extent of myocardial fibrosis has been demonstrated [8]. Notably, the myocardial areas with the lowest regional strain values have been correlated with the greatest myocardial hypertrophy and fibrosis in HCM patients [9].

Furthermore, the systolic anterior motion aka SAM-Phenomenon is typically seen in obstructive HCM. SAM can be visualized with M-Mode or in 2-D Display. The Jet of secondary mitral regurgitation is posteriorly directed. The grade of SAM Phenomenon can be on a scale of 0-3. (Table 1) [10]

Finally, abnormalities in mitral valve (elongated mitral leaflets, displacement of papillary muscles, and SAM) may be the primary pathognomonic elements, even in the absence of hypertrophy [11].

Grade 0	Non existing
Grade 1	Deviation > 5 mm (M-mode)
Grade 2	Non holosystolic contact to ventricular septum
Grade 3	Holosystolic contact to ventricular septum
SAM :	Systolic Anterior Motion

Table 1: SAM-Grading and Echocardiographic Criteria.

Family Screening

First-degree relatives should get a screening with a detailed medical history, physical examination, electrocardiogram (ECG), and echocardiography. According to AHA guidelines, screening should start at age 12. If the child has a growth spurt or signs of puberty, is symptomatic, has activities in high-intensity sports, or there is a positive history of SCD (sudden cardiac death) in the family, screening should be performed earlier. The ESC recommends screening beginning at age 10. From age 12 to 18 years, a yearly screening with ECG and transthoracic echocardiography is recommended. After 18 years of age, screening can be spaced to every 5 years, in the absence of symptoms [12].

Cardiopulmonary exercise testing

The pathophysiological mechanisms undergirding the wide spectrum of HCM symptoms is complex, posing challenges in risk stratification and prognostic ascertainment. In this sense, cardiopulmonary exercise testing (CPET), a valuable noninvasive diagnostic modality, provides an integrated assessment of cardiac, pulmonary, hemodynamic, musculoskeletal, and cellular oxidative pathways. CPET has traditionally been used to enhance risk stratification and prognostication in patients with HF but is increasingly being used and has an expanding role in HCM with endorsements by both the American and European consensus guidelines for the management of HCM [13]. The most commonly used CPET metrics include peak O_2 up take (pVO_2), minute ventilation (VE) to CO_2 production (VCO_2) (VE/ VCO_2) slope, and abnormal blood pressure response to exercise.

Among them, and by far one of the most studied parameters for risk assessment and treatment strategy is maximal oxygen consumption aka VO_2 max. In case of ventricular hypertrophy, in athletes heart is usually more than 110 ml/min/kg, whereas in HCM is usually low [13].

Moreover, a recent study demonstrated, that EO (Exercise oscillatory ventilation) has a prevalence in HCM around 5% and is associated with enlarged left atrium and decreased VO_2 . EO is very well studied and is considered as an independent risk factor for sudden cardiac death, and so a potential new risk factor among the traditional risk factors for assessment, treatment strategy and for primary prophylaxis ICD-Implantation [14-16] (Figure 1).

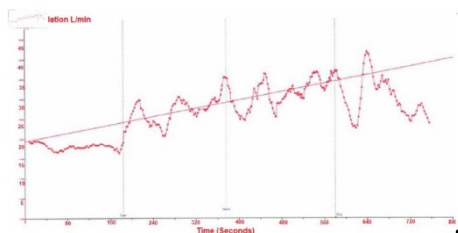


Figure 1. Exercise oscillatory ventilation

Atrial Fibrillation

Atrial fibrillation has an incidence of 2% per year and prevalence of 20% [17]. The risk factors/clinical perspectives for this atrial arrhythmia, which may lead to stroke are: 1) NYHA III/IV at initial evaluation, history of atrial fibrillation, an enlarged left atrium (≥ 45 mm), outflow obstruction (≥ 30 mm Hg) and age > 60 at initial evaluation. Exploratory analyses have shown evidence for a reduction of thromboembolism with vitamin K antagonist treatment. The CHA2DS2-VASc score should not be used to assess thromboembolism risk in HCM population [18].

Magnetic resonance imaging

As aforementioned, diagnosis of HCM will incorporate information obtained also from cardiac MRI. MRI provides useful means for evaluating cardiac components, cardiac structure assessment, cardiac function assessment, flow assessment, perfusion assessment, as also as highly important is the late gadolinium enhancement (LGE) [19].

Extensive assessment provides additional information for assessing SCD (sudden cardiac death) event risk among HCM patients. Furthermore, myocardial bridging, crypts and aneurysms that can be detected only by means of MRI provide an important risk assessment in case of HCM [19].

MRI in Physiological LVH (Athlete's Heart)

Wall Thickness: LV wall thickness is typically ≤ 13 mm. In rare cases (mostly highly trained or Black male athletes), it can measure 13 to 16 mm but is usually proportional to an enlarged left ventricular cavity [20].

Cavity Size: The LV end-diastolic dimension is usually enlarged (>55 mm), creating an "eccentric" pattern of hypertrophy [20].

Tissue Characterization, T1 Mapping & Extracellular Volume (ECV): There is typically no replacement fibrosis. Late gadolinium enhancement (LGE), which indicates scarring usually is absent. Myocardial T1 times and ECV remain normal, indicating healthy, non-fibrotic muscle [20].

Conclusion

Future Aspects

Hypertrophic Cardiomyopathy still remains the leading cause of sudden cardiac death in young athletes. From a cardiovascular challenge in the last century, in the meanwhile has become a contemporary treatable disease.

Nevertheless, in young patients, even in the presence of obvious reasons, aka intense training, use of androgens or testosterone, a finding of enlarged heart and hypertrophy should be considered a red flag as well as an indication for further clarification and screening.

Proper education of cardiopulmonary resuscitation, use of external defibrillator as well as frequent informative education material and / or seminars in general population is essential.

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