Exercise Prescription for the Athlete with Cardiomyopathy

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KEYWORDS

• Cardiomyopathy • Sports participation • Exercise • Sudden cardiac death • Bethesda conference

KEY POINTS

- Current international guidelines recommend that athletes with cardiomyopathies refrain from all but low-intensity competitive sports independent of the presence or absence of high-risk features for sudden cardiac death (SCD).
- The incidence of SCD among athletes is low and not different than age-matched nonathlete populations. Among studies of SCD in athletes, the causes of death are widely discrepant. Hypertrophic cardiomyopathy is not uniformly the single most common cause of SCD in young athletes.
- Risk of SCD with sports participation is likely not equivalent for all athletes and many may be able to continue to compete safely. At present, risk stratification for SCD in athletes with cardiomyopathy is challenging and more data are needed before existing guidelines can be modified.

A question that care providers for patients with cardiomyopathies are often asked is "Can I exercise?" This question is not a simple one deserving of a "yes" or "no" answer. It becomes even more complicated when the patients posing the question are athletes, some of whom depend on their physical capabilities to make a living and almost all of whom depend on them to feel "alive" every day. So, how can one balance the patient's desire to exercise, train, and compete against a real or perceived risk of causing harm? Do these recommendations differ depending on the type of cardiomyopathy? What data inform existing guidelines? How should clinicians guide the new cohort of individuals who carry a gene mutation yet lack any evidence of clinical disease (genotype positive-phenotype negative)?

INHERITED CARDIOMYOPATHIES PRIMER

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is the most common genetic cardiovascular disease, affecting 1 in 500 people and characterized by ventricular hypertrophy and relaxation abnormalities.[1] It is inherited in an autosomal-dominant fashion and caused by mutations in cardiac sarcomere protein genes.[2] Despite being considered a monogenic disease, HCM is very heterogeneous with respect to clinical disease severity, even within a single family sharing an identical mutation. Diagnosis of HCM relies primarily on imaging informed by history and physical examination. Echocardiogram typically demonstrates a nondilated, hypertrophied, hyperdynamic left ventricle (LV) in the absence

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of any other disease that may otherwise explain the hypertrophy (Fig. 1). HCM can be complicated by sudden cardiac death (SCD), heart failure (HF), and/or cardioembolic stroke resulting from atrial fibrillation. Treatment options for HCM include implantable cardioverter-defibrillator (ICD) therapy to prevent SCD and medications, percutaneous alcohol septal ablation, or surgical myectomy to reduce symptomatic outflow tract obstruction. Many patients have a normal life expectancy, as the average mortality rate for individuals afflicted with HCM roughly equals that of the US population.[2]

Dilated Cardiomyopathy

Dilated cardiomyopathy (DCM) is characterized by dilation and impaired systolic function of the left or both ventricles (Fig. 2). DCM can present with symptomatic HF, arrhythmias, thromboembolic complications, or SCD. It may also be detected incidentally in asymptomatic individuals. Idiopathic DCM accounts for about 50% of patients with initially unexplained cardiomyopathy.[3] It is estimated that 20% to 35% of patients with idiopathic DCM have familial disease (FDC).[4–6] Most FDC is transmitted in an autosomal-dominant inheritance pattern. Several major societies and organizations have published guidelines for the treatment of HF with reduced ejection fraction that share many recommendations.[7–9]

Left Ventricle Noncompaction

LV noncompaction (LVNC) is newly recognized in its isolated form as a distinct cardiomyopathy. It is characterized by prominent LV trabeculae and deep intertrabecular recesses resulting in a double-layered myocardial structure consisting of an outer, compacted layer and inner, trabeculated or noncompacted layer (Fig. 3). Like HCM and DCM, autosomal-dominant inheritance is most common with reports of 12% to 50% having a family history of LVNC.[10–12] Its true incidence and prevalence are not known because of the lack of agreement on diagnostic criteria and the difficulty in its identification, and its heterogeneous clinical spectrum.

Its clinical expression is highly variable. Affected individuals may or may not have any symptoms, impaired systolic function and HF, arrhythmias, thromboembolic events, or sudden death.[11,13–16] The largest study on the natural history of children with LVNC highlights an overall mortality rate of nearly 13%, with systolic dysfunction and arrhythmias portending a worse prognosis.[14] Those with isolated hypertrabeculation and no associated systolic dysfunction, chamber dilation, or arrhythmias had a significantly decreased risk of mortality. A systematic review including five studies with a total of 241 adults with isolated LVNC also revealed an overall mortality rate of 14% over 39 months.[17]

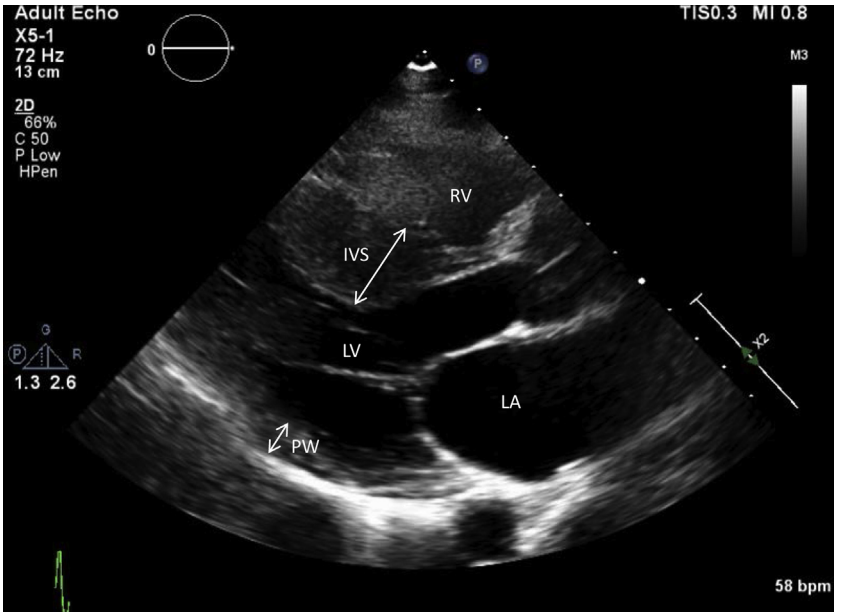

Fig. 1. Hypertrophic cardiomyopathy. Echocardiographic image of hypertrophic cardiomyopathy showing asymmetric hypertrophy of the interventricular septum (IVS) relative to the posterior wall (PW) (*double-headed arrows*). LA, left atrium; RV, right ventricle.