Athletic Heart Syndrome: When, Why and How?

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Jacques’s Heart

- Jacques, 18, is a diver and competitive swimmer.
- Recently, he went for a physical examination for qualification for the Pan Am Games; the team doctor noticed a 2/6 systolic murmur at the left sternal border and a third and fourth heart sound.
- Jacques’s chest X-ray showed a cardiomegaly cardiothoracic ratio > 50%.
- Jacques’s electrocardiogram showed sinus arrhythmia with junctional rhythm at 45 beats per minute with a prominent U wave, peaked T waves and electrical criteria for left ventricular hypertrophy in the precordial leads.
- Jacques has never used performance-enhancing drugs or drugs of any kind.
- He declines symptoms of chest pain, dyspnea, presyncope or palpitations.

What is Jacques’s risk of sudden cardiac death? What should be done? For the answer, see page 30.

Q: What is athletic heart syndrome?

Athletic heart syndrome (AHS) is a common term for an enlarged heart associated with repeated strenuous exercise. As a result of the increased workload required of the heart, it will increase physiologically by enlarging chambers and muscle mass, or hypertrophy by enlarging the size of the chambers and increasing the volume of blood pumped per stroke. Consequently, the heart has to contract less frequently and, at rest, will beat as few as 45 times per minute as compared with an average number of 75 beats in a normal heart.

The athlete who needs a high capacity for oxygen transport benefits from a larger stroke volume, a low heart rate and a hypertrophied ventricular wall. But while the heart continues to function adequately as a pump by altering its rate and contractility when confronted with sudden demand, chronic demand causes dilation and hypertrophy of the cardiac muscle.

The changes in heart structure and function seen in AHS would suggest illness if seen in non-athletes. In athletes, however, these are normal physiologic adaptations to physically demanding lives. When abnormalities in heart structure or function are detected in an athlete, it is important to ensure the abnormalities are indeed due solely to exercise conditioning, and not to a cardiac disorder.
What other, more serious conditions appear similar to AHS?

Hypertrophic cardiomyopathy (HCM)

HCM is responsible for 50% of all sudden cardiac deaths (SCDs) in young athletes. It is an autosomal, dominant condition with variable penetration. The clinical criteria for diagnosis is a hypertrophic left ventricle without systemic or valvular obstruction, like hypertension or aortic stenosis.

HCM manifests only in entering adolescence. The first symptom of this condition can be syncope or thoracic pain, but sometimes SCD is the first symptom. The definitive diagnosis is made by echocardiography, but there are some clues in physical examination—like a systolic murmur potentiated by the Valsalva manoeuver and an S4, as well as with simple laboratory testing, like electrocardiogram modifications (noted in Jacques’s case) and cardiomegaly in chest X-rays—that increase the suspicion of HCM.

There are obviously some limitations to these tests; the gold standard is the echocardiogram, which often shows a hypertrophic left ventricle with asymmetric septal hypertrophy, outflow obstruction in Doppler or thickening of the anterior mitral valve leaflet.

Dilated cardiomyopathy (DCM)

Normally, DCM is caused by viral infections, like Coxsackie B. In most cases, it is asymptomatic until the signs and symptoms of congestive heart failure appear. Before these symptoms, DCM can cause ventricular arrhythmias and SCD, making the diagnosis difficult.

Marfan’s syndrome

This congenital syndrome is responsible for cardiovascular problems like aortic dissection, mitral valve prolapse, aortic insufficiency or arrhythmias.

More on Jacques

Jacques is at minimal risk for sudden cardiac death because he has an athletic heart, not cardiomyopathy. He should undergo a history and physical examination by a trained sports medicine doctor; if there is a concern, Jacques should have an echocardiogram.

Long QT syndrome

Long QT syndrome is a congenital disease where torsades de pointes syndrome may occur, and is thought to be caused by early contraction of cardiac tissue while most of the tissue is undergoing depolarization. As a result, polymorphic ventricular tachycardia may be present and lead to SCD.

Mitral valve prolapse and aortic stenosis

These conditions are also related to SCD, but they are less prevalent in provoking arrhythmias.

About the author...

Dr. Abdulla is pending Lecturer status at the University of Ottawa, and is the Medical Director of the Kingsway Health Centres and a Sports Medicine Consultant at the Nepean Sports Medicine Centre, Ottawa, Ontario.
How can one identify a young athlete at risk for SCD?

Until now, there have been no specific procedures to identify at-risk athletes and, more importantly, no definition of when an athlete should be disqualified or allowed to compete.

The most common screening practice is a series of questions based on pre-syncope symptoms, chest pain, palpitations, dyspnea and a family history of HCM, SCD, long QT syndrome or undiagnosed syncopal episodes. Also consider a clinical history of drug abuse, the use of anabolic steroids, recent viral infections and very tall athletes with arachnodactyly or an arm span greater than their height. Clinically suspicious athletes need to go for further testing.

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