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Cardiac Hypertrophy: Athlete's Heart or Heart Disease?

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Abstract

Hypertrophic heart or cardiac hypertrophy is a medical condition involving a thickening of the heart muscle which can make it more difficult for the heart to pump blood. The causes can be varied and can lead to serious heart problems and in extreme cases to death. The important thing is to distinguish a condition of cardiac hypertrophy which turns out to be a real condition of heart disease compared to a condition of cardiac hypertrophy from "athlete's heart".

A hypertrophic heart refers to a series of heart problems in which the tissue of the muscle walls thickens. The causes can be varied and can lead to serious heart problems and in extreme cases to death. Hypertrophic heart or cardiac hypertrophy is a medical condition involving a thickening of the heart muscle which can make it more difficult for the heart to pump blood. Heart hypertrophy often goes undiagnosed because many people have no symptoms. Conversely, in people in whom they occur, it causes shortness of breath, chest pain and alterations in the electrical system of the heart; these lead to life-threatening irregular heart rhythms or sudden death. As already mentioned, some people with myocardial hypertrophy have no symptoms while others can feel them with exercise or exertion. Still others may not have them in the initial stages but develop them later. Knowing the symptoms is important as it can help in getting an earlier diagnosis and therefore better results with treatment. Symptoms are chest pain, especially with physical exertion, shortness of breath, fatigue, arrhythmias, dizziness, fainting, and ankle swelling. This is a chronic disease and can get worse over time. For this reason, those who are subject to it must pay attention to their lifestyle.

The important thing, however, remains to distinguish a condition of cardiac hypertrophy which turns out to be a real condition of heart disease compared to a condition of cardiac hypertrophy from "athlete's heart".

Hypertrophic Cardiomyopathy is a condition in which the heart muscle thickens, becoming hypertrophic, in the absence of dilatation of the ventricles. Research shows that this condition is much more common than previously thought: estimates speak of 1 in 500-1,000 people affected by hypertrophic cardiomyopathy, men and women equally, which is present in all parts of the world. Often it is not even diagnosed due to the absence of symptoms and in many cases, it allows you to lead a normal life. Possible manifestations are: arrhythmias (which can cause sudden death), symptoms of obstruction to the outflow of blood from the left ventricle (such as dizziness and fainting), heart failure and myocardial ischemia. In hypertrophic cardiomyopathies, the left ventricle becomes less elastic and therefore has a reduced capacity to receive blood from the lungs. The result is a reduction in the amount

of blood pumped by the heart ("diastolic" or "preserved ejection fraction" heart failure): hence the symptoms of heart failure. There is also a microvascular dysfunction that causes myocardial ischemia, which can lead to micro-infarctions; the chest pains often present in this pathology are perhaps partly attributable to this. There are several possible developments of a hypertrophic cardiomyopathy that has not been promptly treated. Arrhythmias with consequent possibility of fatal outcome and in general the most common are brief or non-sustained ventricular tachycardia and extrasystole. The onset of atrial fibrillation which may be tolerated by some patients while causing severe symptoms in others. The atria of the heart stiffen and remain still, while the ventricles contract abnormally, which can also cause blood clots to form, which can lead to stroke. Heart failure, which involves the inability of the heart to pump the blood needed to nourish the body. Cardiac ischemia, in which the reduction of blood flow to the tissues can cause death by necrosis of cells, with the possibility of myocardial infarction. According to estimates, the risk of sudden death is not obvious, but seems above all to be linked to some risk factors: severe hypertrophy (3 centimeters or even more), previous cardiac arrest, syncope not connected to the function of the vagus nerve and family history of sudden death especially in young relatives.

Hypertrophic cardiomyopathy is therefore the condition in which the walls of the heart muscle become markedly thicker, especially in the left ventricle. In this area of the heart, the thickening can be slight or even reach a level three times greater than the standard: under normal conditions, the left ventricle has a rather uniform thickness of about 10 millimeters, usually 3 times greater than the right. In the case of hypertrophic cardiomyopathy, the diagnosis becomes certain when the left ventricle has even only one wall whose thickness corresponds to 15 millimeters or more. In the most serious cases, hypertrophy can even reach 4 centimeters in thickness: unfortunately, the risk of sudden death is greater in these subjects. If, on the other hand, the increase in thickness is less, up to 13 or 14 millimeters, we speak of hypertrophic cardiomyopathy only if there are also serious anomalies detected with an electrocardiogram, genetic mutations linked to hypertrophy and familiarity. The hypertrophy of the muscle can also be distributed in a non-homogeneous way in the wall, while the cavity of the left ventricle is of the standard size or reduced. Precisely depending on the severity of this increase in thickness and its effects on the heart, it is possible to distinguish two types of hypertrophic cardiomyopathy. Hypertrophic obstructive cardiomyopathy, or with obstruction, which is present in about 25% of patients. With "obstruction" we mean the one to the efflux of blood, that is to say its exit from the left ventricle. It is caused by hypertrophy of the interventricular septum and by an abnormality of the mitral valve, between the ventricle and the left atrium. This valve is larger in size and moves like a flag, thus preventing the blood from flowing regularly. Generally, the cardiologist can easily

recognize it through auscultation of a characteristic heart murmur, linked to the turbulent motion of the blood. In some patients it is latent, that is it occurs only after and during an effort or a very substantial meal. It is the most serious type of hypertrophic cardiomyopathy, as it can have an unfavorable course if left untreated. In about 3% of patients, hypertrophic cardiomyopathy evolves into a dilated form with refractory heart failure and poor prognosis. Non-obstructive hypertrophic cardiomyopathy in which the reduction in blood flow from the left ventricle is only modest. The localization of the thickening is also useful in identifying the type of this condition. In fact, we recognize asymmetric hypertrophic cardiomyopathy, which implies hypertrophy in the interventricular septum alone, that is, in that muscular area that divides the right and left ventricle. If all the ventricular walls are thickened, we speak of concentric hypertrophy. And finally, apical hypertrophic cardiomyopathy, in which hypertrophy mainly affects the upper part (apex) of the heart.

Generally, at the basis of hypertrophic cardiomyopathy there is a genetic mutation, which determines not only myocardial hypertrophy, but also an abnormal arrangement of the cardiac muscle fibers. As it is a hereditary disease, there are no methods to prevent hypertrophic cardiomyopathy. The risk that the child of an individual affected by the problem inherits the underlying genetic mutation is 50%, as the pathology is transmitted in an autosomal dominant manner. The mutation, that is the alteration of the DNA, occurs in the genes linked to the coding of the contractile proteins of the myocardium: in particular, 70% of subjects with hypertrophic heart have mutations in the genes MYH7 (of the heavy chains of Beta-myosin), MYBPC3 (myosin binding protein C), TNNT2 (cardiac troponin T) and TNNI3 (cardiac troponin I).

A completely different condition is the one that develops in the heart of subjects who perform regular, amateur, or professional physical activity; in this case the heart undergoes a series of morphological and functional adaptations known as "athlete's heart".

"Athlete's heart", or athlete's heart syndrome, is an expression that in the medical field not only metaphorically indicates the excellent state of cardiovascular health of those who train constantly but refers to specific and measurable changes that involve the anatomy and functioning of the heart. The same term is used both for the adaptations that those who practice aerobic exercise (for example running or cycling at high intensity) undergo, as well as for those who instead dedicate themselves to weight lifting, which in reality are at least partially different. Athlete's heart is a constellation of structural and functional changes found in the heart of subjects who train for prolonged periods (> 1 hour almost every day) and/or frequently at high intensity. The changes are asymptomatic; signs include bradycardia, a systolic murmur, and added tones. Intense and prolonged resistance and strength

training cause various physiological adaptations. Volume and pressure load increase in the left ventricle, which, over time, lead to an increase in left ventricular muscle mass, wall thickening, and cavity size. They increase maximal stroke volume and cardiac output, contributing to a lower resting heart rate and prolongation of diastolic filling time. The slow heart rate is primarily caused by increased vagal tone but decreased sympathetic tone and other non-autonomic factors that reduce intrinsic sinus node activity may also play a role. Bradycardia reduces myocardial oxygen demand; at the same time, increases in total hemoglobin and blood volume increase oxygen delivery. Despite these changes, systolic and diastolic function remain normal. Structural changes in women are typically less marked than in men of the same age, body size, and fitness level. Thus, cardiac adaptations to training are characterized by a symmetrical increase in intracavitary diameters and atrial and ventricular chamber wall thicknesses. The morphology of the athlete's heart often presents intermediate characteristics between a concentric and eccentric pattern of left ventricular hypertrophy (LVH), in view of the large prevalence of sports activities with mixed type characteristics and training protocols, in which a balanced aerobic and anaerobic activity is usually programmed. With ultrasound it is possible to distinguish the different models of adaptation of the heart in athletes who practice different sports. As far as the left ventricle is concerned, two adaptation models have been identified: eccentric hypertrophy which concerns aerobic and resistance athletes, in which the left ventricle increases its internal volume and the thickness of its walls, assuming a rounded shape and the concentric hypertrophy which concerns athletes who perform static, power sports, in which the left ventricle increases the thickness of the walls without increasing the internal volume, maintaining its original shape, ovoid, or assuming a more elongated shape.

In its most marked expressions, athlete's heart can resemble the heart of some cardiac diseases (hypertrophic, dilated, arrhythmogenic cardiomyopathy, non-compact myocardium). In these cases, it becomes important, even if not always simple, to distinguish between the natural remodeling of the heart and the disease, and to establish whether pathological conditions are hidden behind the athlete's heart. A healthy athlete is usually asymptomatic. During the visit, there are some clinical conditions that must prompt the doctor to request more in-depth investigations (second level). The main conditions include family history of sudden cardiac death or cardiomyopathy, palpitations (feeling of rapid heartbeat in the chest), syncope (fainting, loss of consciousness), chest pain, heart murmurs or other heart sounds. The standard ECG of athletes often shows "alterations" which are the result of the natural adaptation of the heart to physical training. The most frequent alterations (up to 80% of cases), which should not cause concern, are sinus bradycardia, first degree atrioventricular block, signs of early repolarization and signs of left ventricular hypertrophy. Abnormalities that, on the other hand,

necessarily deserve further diagnostic evaluations, since they could reflect the presence of an underlying cardiac pathology, are the following: ST segment depression and/or T wave inversion, pathological Q waves, intraventricular conduction delays (complete left bundle branch block, bifascicular block, nonspecific intraventricular conduction delays), signs of ventricular pre-excitation, long or short QT interval, and ventricular arrhythmias. Finally, there are ECG changes of uncertain significance in athletes such as atrial enlargements, axis deviations, and right ventricular hypertrophy.

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Echocardiography can generally distinguish athlete's heart from cardiomyopathies, but the distinction is not always clear because there is a continuum from physiological to pathological to regard cardiac enlargement. The zone of overlap between the athlete's heart and cardiomyopathy is represented by the thickness of the septum: 13-15 mm in men and 11-13 mm in women. In this overlapping zone, the presence of strong systolic anterior mitral valve motion strongly suggests hypertrophic cardiomyopathy. Also, diastolic indices may be abnormal in cardiomyopathy but are usually normal in athlete's heart. In general, echocardiographic changes correlate poorly with fitness level and cardiovascular performance. Minimal mitral and tricuspid regurgitation are usually noted. Of note, the reduction in physical training will result in a regression of cardiac dilatation in patients with athlete's heart, but not in those with cardiomyopathy.

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