

# Hypertrophic Cardiomyopathy in a Heart-Related Patient

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## Introduction

The heart thickens without apparent cause in hypertrophic cardiomyopathy (HCM) or obstructive hypertrophic cardiomyopathy (HOCM). The interventricular septum and the ventricles of the heart are the parts of the heart that are most frequently affected. This makes it harder for the heart to pump blood and may also hinder electrical conduction. A wide range of symptoms may be experienced by HCM patients. People may experience shortness of breath, limb edoema, weariness, and other symptoms. It also has the potential to cause chest pain or pass out. If someone is dehydrated, the symptoms may get worse. Problems like heart failure, irregular heartbeats, and sudden cardiac death are all possible [1].

## Description

HCM is frequently inherited autosomally dominantly from parents. Most of the time, the problem is caused by mutations in genes that make proteins for the heart muscle. Left ventricular hypertrophy can be caused by Fabry disease, Friedreich's ataxia, or certain drugs like tacrolimus. Athlete's heart or hypertension (high blood pressure) could also cause an enlarged heart. An ECG, an echocardiogram, stress testing, and a family history or pedigree are used to diagnose HCM. Genetic testing is yet another option. Unlike Fabry disease, which is X-linked, and Friedreich's Ataxia, which is autosomal recessive, HCM is an autosomal dominant form of inherited cardiomyopathy.

Symptoms and other risk factors may influence treatment. Beta blockers and disopyramide are two possible medications. Patients with certain kinds of irregular heartbeats may benefit from the use of an implantable cardiac defibrillator. Surgery, such as a septal myectomy or a heart transplant, may be necessary if other treatments fail. The disease's annual mortality rate is less than 1% with treatment [2].

## Signs and symptoms

There are a variety of factors that can cause HCM. A lot of people with HCM have no symptoms or only minor symptoms, and many people with HCM genes don't have a disease that can be seen in the clinic. Symptoms of HCM include shortness of breath due to stiffening and decreased blood filling of the ventricles, exertional chest pain (also known as angina) due to reduced blood flow to the coronary arteries, unpleasant awareness of the heartbeat (palpitations), disruption of the electrical system that runs through the abnormal heart muscle, lightheadedness, weakness, fainting, and sudden cardiac death.

The stiffness of the left ventricle (LV) is the primary cause of shortness of breath. This not only prevents the ventricular chambers from filling up, but it also increases pressure in the left ventricle and left atrium, causing back pressure

and interstitial congestion in the lungs. The presence or severity of an outflow tract gradient has no effect on symptoms. Although the treatment is distinct, the symptoms, particularly exercise intolerance and dyspnea, are comparable to those of congestive heart failure. While diuretics, a common CHF treatment, exacerbate symptoms in hypertrophic obstructive cardiomyopathy by lowering ventricular preload volume and thereby increasing outflow resistance (less blood to push aside the thickened obstructing tissue), beta blockers are used in both cases.

## Diagnosis

Multiple aspects of the disease process are taken into consideration when making the diagnosis of hypertrophic cardiomyopathy. ECG, genetic testing (albeit not primarily for diagnosis), and any family history of HCM or unexplained sudden death in otherwise healthy people are also important factors to examine. While cardiac MRI, cardiac catheterization, and echocardiography are used to diagnose the disease, the ECG is not the only important factor. In 60 to 70% of cases, cardiac MRI shows that the bottom of the ventricular septum thickens by more than 15 millimeters. Scarring in cardiac tissues can be seen with T1-weighted imaging, while oedema and inflammation, both of which are linked to acute symptoms like chest pain and fainting, can be seen with T2-weighted imaging.

## Screening

Despite the fact that HCM might be asymptomatic, those who are affected can develop symptoms ranging from mild to severe heart failure and sudden cardiac death at any age, from infancy to old age. In the United States, HCM is the most common genetic cardiovascular disease and the leading cause of sudden cardiac death in young athletes. According to one study, since 1982, when routine cardiac screening for athletes was started, the rate of sudden cardiac mortality in young competitive athletes in the Veneto area of Italy has fallen by 89 percent, from an abnormally high beginning rate. However, studies show that, as of 2010, the rate of sudden cardiac death among all HCM patients had dropped to less than 1%. Those who have received a positive test.

An echocardiogram (ECHO), which can be followed by an electrocardiogram (ECG) to look for heart abnormalities, can diagnose HCM with an accuracy of 80% or higher. Cardiac Magnetic Resonance Imaging (CMR), the gold standard for analysing the physical features of the left ventricle wall, can be employed as an alternative screening method when an echocardiogram provides unclear data. For example, segmental lateral ventricular hypertrophy cannot be detected just using echocardiography. In children under the age of thirteen, left ventricular hypertrophy may or may not exist. This puts doubt on pre-adolescent echocardiography results [3].

Even though HCM may not cause any symptoms, those with it can experience mild to severe heart failure and sudden cardiac death at any age, from infancy to old age. HCM is the most prevalent genetic cardiovascular disease and the leading cause of sudden cardiac death in young athletes in the United States. One study found that the rate of sudden cardiac death among young competitive athletes in the Veneto region of Italy has decreased by 89% from an abnormally high beginning rate since the introduction of routine cardiac screening for athletes in 1982. However, research indicates that the overall rate of sudden cardiac death among HCM patients had decreased to less than 1% by 2010. Individuals who have tested positive.

HCM can be diagnosed with an accuracy of at least 80% using an echocardiogram (ECHO), which can be followed by an electrocardiogram (ECG) to look for heart abnormalities. When an echocardiogram provides unclear data, cardiac magnetic resonance imaging (CMR), the gold standard

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for analyzing the physical features of the left ventricle wall, can be used as an alternative screening method. For instance, echocardiography alone cannot detect segmental lateral ventricular hypertrophy. There may or may not be left ventricular hypertrophy in children under the age of thirteen. This casts doubt on the findings of the pre-adolescent echocardiogram.

Crypts in the interventricular septal tissue were discovered when CMR was used to investigate asymptomatic carriers of an HCM-causing mutation. These crypts have been interpreted as a sign of myocyte disorganization and altered vessel walls, both of which could one day result in the clinical manifestation of HCM. The fact that the majority of family history research focuses primarily on whether or not there was a sudden death is one possible explanation. It doesn't take into account how often relatives died of sudden cardiac death or how old they were.

## Treatment

Although strenuous activities and competitive sports should be avoided, many people with hypertrophic cardiomyopathy do not exhibit symptoms and go about their daily lives normally. People who are asymptomatic should have their risk factors for sudden cardiac death checked. Situations that result in dehydration or vasodilation (such as the use of vasodilator or diuretic blood pressure medications) should be avoided in patients with resting or inducible outflow obstructions. Septal reduction therapy should not be used on people who don't have any symptoms [4].

Medication's primary objective is to alleviate symptoms like palpitations, shortness of breath, and chest pain. First-line medications are beta blockers

because they can slow the heart rate and reduce the risk of ectopic heartbeats. Patients who are unable to take beta blockers may be treated with nondihydropyridine calcium channel blockers, such as verapamil, but these medications can be harmful if you suffer from severe shortness of breath at rest or low blood pressure. These medications also slow the heart rate, but people with significant outflow obstruction, high pulmonary artery wedge pressure, or low blood pressure should use them with caution. People who have signs of a blocked calcium channel should not take dihydropyridine calcium channel blockers [5].

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