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Hypertrophic Cardiomyopathy Diagnosis and Evaluation

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Introduction

Hypertrophic cardiomyopathy (HCM or HOCM if obstructive) is a disorder in which the heart thickens for no apparent reason. The interventricular septum and the ventricles are the most usually afflicted sections of the heart. This causes the heart to be less effective at pumping blood and may also create electrical conduction difficulties. HCM patients may experience a variety of symptoms. People may have a variety of symptoms, including weariness, limb edoema, and shortness of breath. It can also cause chest pain or make you pass out. When a person is dehydrated, the symptoms may be exacerbated. Heart failure, an irregular heartbeat, and sudden cardiac death are all possible complications [1].

HCM is most usually inherited in an autosomal dominant manner from a person's parents. It is frequently caused by mutations in genes involved in the production of cardiac muscle proteins. Fabry disease, Friedreich's ataxia, and certain drugs like tacrolimus can all be genetic causes of left ventricular hypertrophy. Athlete's heart and hypertension are two more possible reasons of an enlarged heart (high blood pressure). A family history or pedigree, an ECG, an echocardiography, and stress testing are typically used to diagnose HCM. It is also possible to conduct genetic testing. The autosomal dominant type of HCM distinguishes it from other inherited causes of cardiomyopathy, whereas Fabry disease is X-linked and Friedreich's Ataxia is autosomal recessive.

Symptoms and other risk factors may influence treatment. Beta blockers and disopyramide are two medications that may be used. In patients with specific forms of irregular heartbeats, an implantable cardiac defibrillator may be advised. Those who do not improve with alternative treatments may need surgery, such as a septal myectomy or a heart transplant. The chance of dying from the condition is less than 1% each year with treatment [2].

Signs and Symptoms

HCM can be caused by a variety of factors. Many persons with HCM are asymptomatic or only slightly symptomatic, and many people with HCM disease genes do not have clinically evident disease. Shortness of breath due to stiffening and decreased blood filling of the ventricles, exertional chest pain (sometimes called angina) due to reduced blood flow to the coronary arteries, unpleasant awareness of the heart beat (palpitations), as well as disruption of the electrical system running through the abnormal heart muscle, lightheadedness, weakness, fainting, and sudden cardiac death are all symptoms of HCM.

Shortness of breath is caused primarily by stiffening of the left ventricle (LV), which not only hinders ventricular filling but also leads to raised pressure in the left ventricle and left atrium, resulting in back pressure and interstitial congestion in the lungs. The presence or severity of an outflow tract gradient has no effect on symptoms. Symptoms often resemble those of congestive heart failure (especially exercise intolerance and dyspnea), but therapy differs. In both situations, beta blockers are employed, but diuretics, which are a common treatment for CHF, increase symptoms in hypertrophic obstructive cardiomyopathy by lowering ventricular preload volume and thereby raising outflow resistance (less blood to push aside the thickened obstructing tissue) [2].

Diagnosis

A number of characteristics of the disease process are used to make a diagnosis of hypertrophic cardiomyopathy. While echocardiography, cardiac catheterization, and cardiac MRI are utilised in the identification of the illness, ECG, genetic testing (albeit not primarily used for diagnosis), and any family history of HCM or unexplained sudden death in apparently healthy persons are all essential considerations. The bottom section of the ventricular septum thickens more than 15 mm in 60 to 70% of instances, according to cardiac MRI. T1-weighted imaging can detect scarring in cardiac tissues, but T2-weighted imaging can detect oedema and inflammation in cardiac tissue, both of which are linked to acute symptoms including chest discomfort and fainting [3].

Screening

Although HCM can be asymptomatic, affected people can develop symptoms ranging from mild to severe heart failure and abrupt cardiac death at any age, from infancy to old age. HCM is the most frequent hereditary cardiovascular illness and the primary cause of sudden cardiac mortality in young athletes in the United States. According to one study, the rate of sudden cardiac mortality in young competitive athletes in the Veneto area of Italy has decreased by 89 percent since routine cardiac screening for athletes was introduced in 1982, from an abnormally high beginning rate. However, studies reveal that the rate of sudden cardiac death among all patients with HCM has decreased to less than one percent as of 2010. Individuals who have had a positive test and have been diagnosed with heart disease [4].

An echocardiogram (ECHO) can diagnose HCM with an accuracy of 80% or higher, which can be preceded by an electrocardiogram (ECG) to check for heart irregularities. When an echocardiogram yields ambiguous results, cardiac magnetic resonance imaging (CMR), the gold standard for assessing the physical features of the left ventricular wall, can be used as an alternate screening tool. The detection of segmental lateral ventricular hypertrophy, for example, cannot be done just by echocardiography. Left ventricular hypertrophy may sometimes be absent in children under the age of thirteen. This casts doubt on the echocardiography results of pre-adolescents. Researchers have used CMR to study asymptomatic carriers of an HCM-causing mutation and were able to find crypts in the interventricular septal tissue in these persons. It's been suggested that the creation of these crypts is a sign of myocyte disorganisation and changed vessel walls, which could lead to the clinical manifestation of HCM later on. One probable explanation is that most family history gathering focuses solely on whether or not a sudden death occurred. It ignores the age at which relatives died of sudden cardiac death, as well as the frequency with which they died [5].

Treatment

Asymptomatic

Many persons with hypertrophic cardiomyopathy have no symptoms and

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live regular lives, though they should avoid particularly rigorous activities or competitive athletics. People who are asymptomatic should be tested for risk factors for sudden cardiac death. Situations that cause dehydration or vasodilation (such as the use of vasodilatory or diuretic blood pressure drugs) should be avoided in patients with resting or inducible outflow blockages. Asymptomatic persons should avoid septal reduction therapy.

Medications

Medication's main objective is to alleviate symptoms like chest discomfort, shortness of breath, and palpitations. Beta blockers are first-line medications because they can lower heart rate and reduce the risk of ectopic beats. Nondihydropyridine calcium channel blockers, such as verapamil, can be used in persons who cannot tolerate beta blockers, but they are potentially dangerous in people who also have low blood pressure or severe shortness of breath at rest. These drugs also lower heart rates, albeit they should be used with caution in those who have severe outflow blockage, increased pulmonary artery wedge pressure, or low blood pressure. In those who have indications of blockage, dihydropyridine calcium channel blockers should be avoided.

Surgical septal myectomy

Surgical septal myectomy is an open-heart procedure used to ease symptoms in persons who have not responded to medicinal treatment. It has been effectively conducted since the early 1960s. Surgical septal myectomy reduces left ventricular outflow tract obstruction and improves symptoms uniformly, with a surgical mortality rate of less than 1% and an 85 percent success rate at experienced facilities. It entails removing a part of the interventricular septum and performing a median sternotomy (general anaesthesia, opening the chest, and cardiopulmonary bypass) [6].

Conclusion

Identification of the HCM phenotype during cascade family screening relies initially on echocardiography or CMR imaging but can be supplemented with genetic testing to more conclusively determine the affected status of preclinical phenotype-negative relatives. However, routine diagnostic echocardiographic imaging in very young (<10-12 years of age) family members can be fraught with uncertainty or false positive diagnoses that create anxiety and may lead to unnecessary clinical recommendations, even though therapeutic interventions are seldom indicated for asymptomatic patients during the first decade. The genetics of HCM is complicated by heterogeneity, incomplete penetrance, variable expression and phenocopies, as well as the difficulty in reliably establishing pathogenicity with testing. Furthermore, increasingly, it is evident that the 30-year single-gene (monogenic) causation hypothesis for HCM lacks robust evidence in many respects, for example, a firm genetic etiology can be identified in only a minority (i.e., 30%) of clinically diagnosed patients. Alternatively, it is possible that the genesis of HCM may be multifactorial and involve nongenetic or environmental factors.

Conflict of Interest

None.

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