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Hypertrophic cardiomyopathy:

New hope for an old disease

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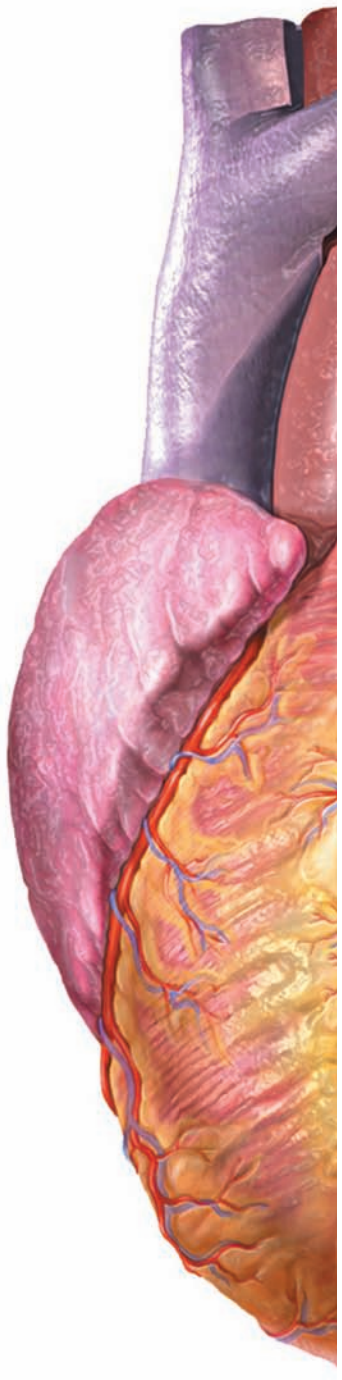


Abstract: Hypertrophic cardiomyopathy (HCM), a common congenital heart disease, is the leading cause of sudden cardiac death in adolescents, young adults, and athletes. Older adults with HCM are less likely to experience sudden cardiac death, but their quality of life can be impaired. This article discusses diagnostic criteria, treatments, and nursing interventions, including patient teaching, for adults with HCM.

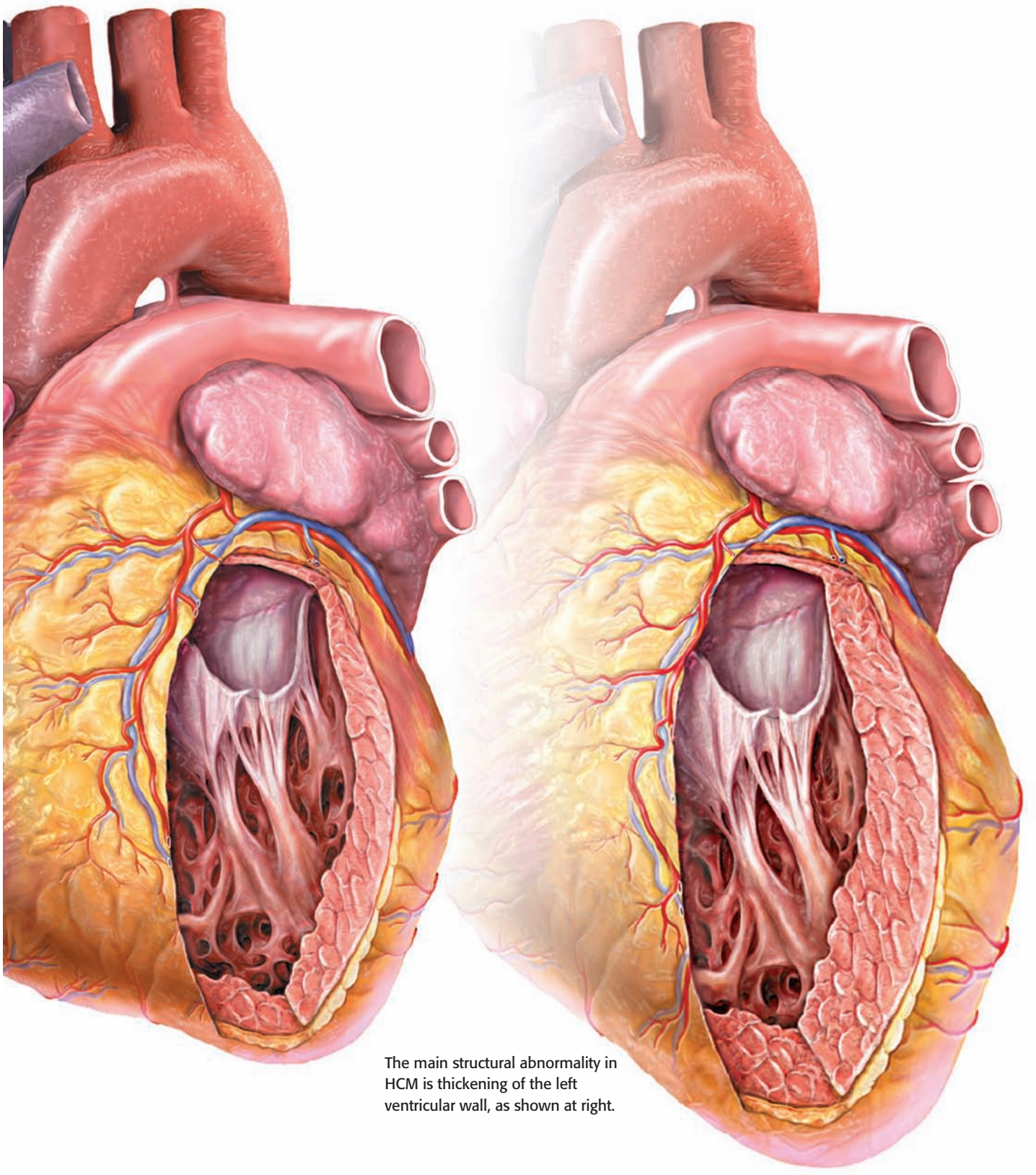
Keywords: alcohol septal ablation, HCM, HOCM, hypertrophic cardiomyopathy, hypertrophic obstructive cardiomyopathy, left ventricular outflow tract obstruction, LVOT obstruction, myocyte disarray, septal myectomy, sudden cardiac death

TM, 54, WAS ADMITTED to the ED after a syncopal episode that resulted in a head injury. Based on an ECG and echocardiogram findings, she was diagnosed with hypertrophic cardiomyopathy (HCM). After her head injury was determined to be non-life-threatening, the treating physician discharged her with instructions to follow up with a cardiologist and consider having family members tested for this genetic disorder.

HCM IS A COMMON congenital heart condition. Worldwide, without discrimination for race, gender, or ethnicity, HCM has a prevalence of approximately 1:200 to 300 in the general population. Based on current data, the Hypertrophic Cardiomyopathy Association estimates that HCM affects 1 million or more people in the US and 36 million or more worldwide.[1]

HCM is differentiated from other types of cardiomyopathy by the absence of any known clinical cause. In some instances, its presence is unknown until the occurrence of sudden cardiac death (SCD).[2,3] HCM is the leading cause of SCD in adolescents, young adults, and athletes.[4]

Although older adults with HCM are less likely to experience SCD, their quality of life can be impaired. Diagnosis followed by appropriate treatment can greatly improve their quality of life. This article discusses diagnostic criteria, treatments, and nursing interventions, including patient teaching, for adults with HCM.



The main structural abnormality in HCM is thickening of the left ventricular wall, as shown at right.

Genetic mutations

HCM can be traced to one of several sarcomere genetic mutations that affect the heart's structure and function. Because it is an autosomal dominant disorder, just one parent needs to have the defective gene to pass it on to offspring.[5-7] But because genotype-positive family members may be phenotype-negative (meaning the gene is present with no disease expression), HCM may not be recognized until, as in TM's case, some health event or series of events leads to its discovery.[7,8] (See *Genotype or phenotype?*)

Although the gene for HCM is present at birth, the disorder is rarely diagnosed until patients are in their teens or early 20s. HCM is unlikely to cause signs and symptoms in younger children, so it is not often diagnosed in childhood. But for reasons not yet fully understood, HCM is being increasingly diagnosed in later adulthood, even in patients over age 60.[1,9-12]

Typically, however, the first clinical manifestations of HCM appear during the rapid growth and development of the heart during adolescence. Unfortunately, because HCM is difficult to detect with standard health screenings, SCD may be the first (and only) sign.[7,13]

Pathophysiology

The main structural abnormality in HCM is thickening of the left ventricular (LV) wall. Expression of the disease results in other heart abnormalities as well, including thickening of the left region of the ventricular septum, mitral valve abnormalities, myocyte disarray (clusters of ventricular myocytes that are disorganized instead of neatly aligned), narrowed intramural coronary arteries, and clusters of interstitial fibrosis throughout the ventricular myocardium.[2,9,10,14]

Key clinical characteristics of HCM include:

- *hypertrophy.* A thickened LV wall occurs in HCM without any dilation or enlargement of the inside (chamber) of the ventricle. In fact, the LV chamber may be smaller than normal due to thickening of the wall. The ventricular septum is often the region with the most significant thickening.[7,10,11] Systolic function in patients with HCM is typically normal or even hyperdynamic. Diastolic dysfunction with decreased LV compliance is common regardless of the presence or absence of outflow tract obstruction.[15]
- *mitral valve abnormalities*. Abnormalities of the structure and motion of the mitral valve are common features of HCM.[2,7] In many patients with HCM, the mitral valve moves forward during systole, in some cases actually touching the septum. Known as systolic anterior motion (SAM), this movement blocks the outflow of blood from the left ventricle.[2,7,16] Combine this with an increase in heart rate or force of contraction, as occur in vigorous bursts of exercise, and cardiac output could reach dangerously low levels, resulting in syncope or near-syncopal episodes. In addition, the increased myocardial oxygen demand may not be met, resulting in ischemia and chest pain.
- *left ventricular outflow tract (LVOT) obstruction.* Most research points to SAM of the mitral valve leaflet as the direct cause of obstruction rather than ventricular septal hypertrophy.[7,17] HCM with LVOT obstruction is sometimes termed hypertrophic obstructive cardiomyopathy, or HOCM.
- *myocyte disarray*. In the healthy myocardium, myocytes are neatly aligned, allowing for optimal electrical conduction and contraction. In HCM, the myocytes are disorganized in some areas. This disarray not only affects contraction but can also contribute to conduction defects.[1,9,10]
- *dysrhythmias.* Various factors can contribute to ventricular dysrhythmias in HCM, such as myocyte disarray, myocardial ischemia, interstitial fibrosis, and other structural changes. Dysrhythmias include nonsustained and sustained ventricular tachycardia, which can deteriorate to ventricular fibrillation and SCD.[7,10]
- *interstitial fibrosis.* In some cases of HCM, scarring and fibrosis cause a stiffening of the left ventricle.[9,10] Inadequate relaxation of the heart during diastole impairs blood flow from the atrium to the ventricle. Diastolic dysfunction may persist for years before systolic dysfunction occurs. In addition, research suggests that the increased load on the atrium causes enlargement and other changes that can increase the risk of atrial dysrhythmias.[4,10,18]
- *atrial fibrillation*. Between 18% and 28% of HCM patients experience atrial fibrillation (AF).[4] In some cases, the AF will convert spontaneously to normal sinus rhythm; in others, cardioversion is necessary. Synchronized electrical cardioversion is the treatment of choice in symptomatic patients. If pharmacologic cardioversion

Genotype or phenotype?[39,40]

All people have a large amount of genes in common, but many individual variations exist. ***Genotype*** refers to a person's individual sequence of genes. The genotype is expressed when the genetic information is used to make protein and RNA molecules.

The expression of the genotype contributes to the individual's observable traits, called the ***phenotype***. These traits include height, eye color, and blood type. Some individual traits are largely determined by the genotype, others by environmental factors.

is indicated, amiodarone is the drug of choice for patients with HCM.[19]

In patients with persistent AF, the risk of embolic stroke is high. These patients are routinely placed on anticoagulation unless contraindicated.[4,18]

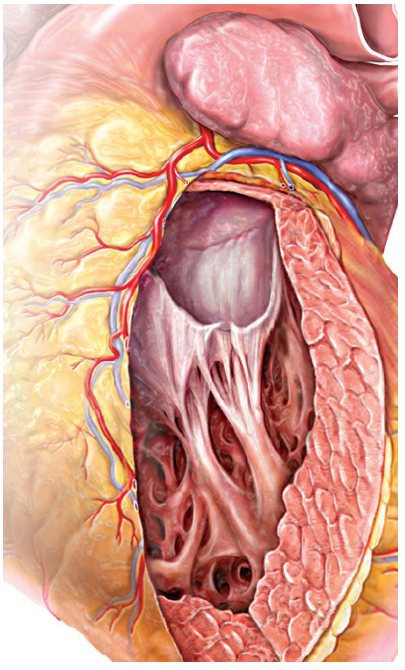
• *heart failure*. Most patients with HCM maintain an adequate ejection fraction (EF) of 50% to 70% for years. But in a small percentage of patients, roughly 5% to 7%, the end-stage or "burn-out" phase of HCM results in systolic heart failure with a thinning and dilation of the left ventricle and decreased EF (below 50%). At this point, patients need more aggressive treatment, as discussed below, or may be considered for heart transplantation.[13,14]

Recognizing signs and symptoms

Because she remained asymptomatic after discharge, TM did not follow up with a cardiologist. Over the next year, she experienced severe dyspnea on exertion (DOE), exercise intolerance, and several additional near-syncopal and syncopal episodes. Each syncopal episode was preceded by extreme fatigue and what she described as a "pounding in her chest." Frightened after one such episode, she called 911 and was taken to the ED. Based on history, physical assessment findings, and prior diagnosis, the ED physician arranged a consult with a cardiologist specializing in HCM.

As critical members of the healthcare team, nurses and paramedics must know how to recognize and report possible signs and symptoms of HCM, which include:

• syncope or near-syncopal episodes, which may be due to inadequate diastolic filling, reduced stroke volume, or dysrhythmias[20]
• fatigue
• DOE
• exercise intolerance, usually due to fatigue, DOE, dizziness, and/or tachycardia
• palpitations, often perceived as "pounding" of the heart



Diastolic dysfunction with decreased LV compliance is common regardless of the presence or absence of outflow tract obstruction.

• angina. Although the patient's epicardial (major) coronary arteries may be healthy, intramural coronary arteries may be narrowed, resulting in ischemia and chest pain. The angina of HCM may be atypical and fluctuating, occurring sometimes at rest.[2,4,10,14]
• postprandial shortness of breath due to reduced myocardial blood flow[21]
• fluid retention (more likely at a more advanced stage of disease). Sudden or unexplained weight gain is an indicator of fluid retention.

Additional physical assessment findings associated with HCM include an exaggerated apical impulse that will also likely be displaced laterally due to the hypertrophy. Some patients have a systolic murmur, believed to be caused by turbulent blood flow around the thickened septum.[22] It is best heard between the apex and the left sternal border. Some patients also have an S4.[9,10,14]

Diagnosis

Although genetic testing can confirm the presence of one of the several known genes for HCM, in many cases the gene is never expressed and hypertrophy does not develop. This can change over time, however, and family members of patients with HCM are advised to have routine cardiac evaluations throughout life.[14]

Most patients with suspected heart disease, including HCM, will first have an ECG. Although the ECG will, in many instances, show changes consistent with HCM, this should simply raise suspicion and not be the sole basis for diagnosis.[23]

The most common ECG abnormalities are voltage changes such as tall R waves and deep S waves. Abnormally deep Q waves may also be present. In some patients, the ECG shows what looks like a left bundle branch block.[10,22]

All patients being evaluated for HCM should undergo comprehensive transthoracic echocardiography with two-dimensional, color Doppler, spectral Doppler, and tissue Doppler imaging. An unexplained increased LV wall thickness of 15 mm or more anywhere in the LV wall is considered diagnostic of HCM. A wall thickness of 13 mm or more may also be considered diagnostic in a patient with a family history of HCM.[22]

Although many patients with HCM are diagnosed with echocardiography alone, this study can also yield results that are within normal limits.[2,14] All test results must be evaluated in the context of a thorough history and physical assessment to determine clinical manifestations of HCM.

A stress echocardiogram can reveal changes that are not apparent while the patient is at rest. Approximately one-third of patients with HCM have evidence of LVOT obstruction while at rest. Another one-third will demonstrate LVOT obstruction during such provocations as exercise stress echocardiography or Valsalva maneuver.[9,10]

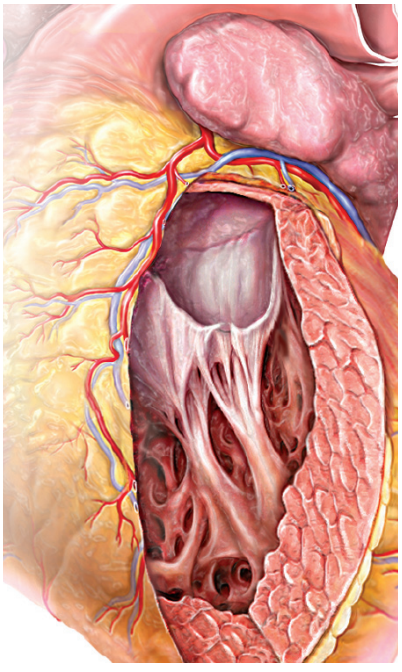
If echocardiography findings do not definitively diagnose HCM, cardiovascular magnetic resonance (CMR) may be performed. Most experts agree that contrast-enhanced CMR should be routinely performed in determining HCM severity. A contrast-enhanced CMR can also help the provider determine the presence of scarring and interstitial fibrosis in the left ventricle.[4,7,10,24] A significant level of fibrosis is considered a major risk factor for SCD.[10,24]

To determine the presence of dysrhythmias, most patients will also have at least 24 hours of continuous ambulatory ECG monitoring. For some patients, the length of cardiac monitoring may be extended for days or even weeks.[9,14,23,25]

Weighing a treatment approach

Most patients diagnosed with HCM can manage signs and symptoms without surgery.[2] In determining the most appropriate therapy, several factors must be considered, including diagnostic study results and severity of signs and symptoms. The provider will weigh the following considerations:

- the risk for SCD. This is determined using risk stratification that includes presence (or absence) of certain factors including history of sudden cardiac arrest, a first-degree relative who died from SCD, unexplained syncope, hypertrophy of 30 mm or more, history of sustained or nonsustained ventricular tachycardia or ventricular fibrillation, and abnormal BP response to exercise.[12,14,23,26]
- diastolic dysfunction (diastolic heart failure), which may be present at the time of diagnosis of HCM due to significant myocyte disarray and interstitial fibrosis that impairs ventricular diastole. This "stiffening" of the left ventricle, while not usually directly impairing ejection fraction, can lead to eventual systolic dysfunction.
- systolic dysfunction. This must be evaluated by EF, which, as explained above, may initially be above 70% because of LV thickening and subsequent increased LV contractility. Expressed as a percentage, EF indicates the amount of blood the left ventricle ejects with each systole. For example, an EF of 60% means that 60% of blood in the left ventricle is ejected with each contraction.[27] However, keep in mind that in HCM, the thickened ventricular muscle may cause a smaller ventricular chamber holding a smaller volume of blood. So even if EF is normal, the amount of blood ejected may be insufficient. Each patient's signs and symptoms must be evaluated individually.[4,14,28]
- baseline EF to assess the progression of HCM and determine the patient's response to medications and other treatment
- presence and degree of LVOT obstruction
- mitral valve abnormalities to evaluate the need to repair or replace the mitral valve
- thickness of the ventricular septum
- thickness of the left ventricle
- presence of interstitial fibrosis
- dysrhythmias, which can usually be well managed with a beta-blocker such as atenolol or, if that is not effective, a calcium channel blocker such as verapamil.



"Stiffening" of the left ventricle may not directly impair ejection fraction but can lead to systolic dysfunction.

Treatment options

Based on patient history, physical assessment findings, and diagnostic study results, the cardiologist considers the following treatment options.

- An implantable cardioverter defibrillator (ICD) is indicated for patients at highest risk for SCD.[2,29]
- Beta-blockers such as atenolol can help minimize dysrhythmias as well as reduce heart rate, thereby increasing diastolic time to allow for better filling of the ventricles.[2,14] A slower heart rate will also decrease myocardial oxygen demand, which helps alleviate chest pain caused by poor coronary perfusion. Controlling heart rate is important because increased heart rate can worsen LVOT obstruction and decrease cardiac output.[14,30]
- Nondihydropyridine calcium channel blockers such as verapamil and diltiazem can help improve ventricular filling and may help reduce angina, DOE, and dysrhythmias.[7,14,31]

• Disopyramide. This Class Ia agent depresses phase 0 (sodium-dependent) depolarization, slowing conduction and decreasing ventricular dysrhythmias.[2,7,14]

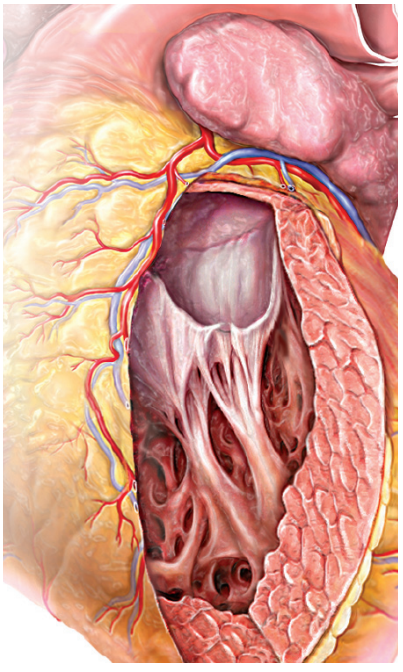
• Surgical septal myectomy to widen the LV outflow tract area. If drug therapy does not significantly improve a patient's signs and symptoms, a surgical septal myectomy is performed to remove a portion of the thickened ventricular septum.[32-34]

• Mitral valve intervention. If the mitral valve leaflet is interfering with the outflow of blood to the aorta, a procedure to correct this is often performed at the same time as the septal myectomy.[14,35] Both procedures usually significantly and permanently relieve much of the obstruction, and the patient experiences dramatic relief of signs and symptoms. Maintenance medications such as beta-blockers are usually prescribed, as are regular follow-up visits.

• Alcohol (ethanol) septal ablation (ASA). This procedure is an alternative for patients who could benefit from a reduction of the thickness of the ventricular septum to relieve obstruction, but who, for many reasons, cannot undergo the surgery needed.[32] By creating a localized myocardial infarction, ASA remodels the area and widens the LVOT, relieving the obstruction. However, unlike surgical septal myectomy, ASA does not address mitral valve abnormalities.

• Patients with end-stage heart failure may be considered for a heart transplant.[36] Some are fortunate enough to gain a new level of health with a successful transplant, but the tradeoff is a lifetime of immunosuppressive medications that place them at higher risk for other illnesses. Fortunately, only a very small percentage (2% to 3%) of patients with HCM progress to the burn-out stage of the disease.[14]

• Lifestyle modifications. As prescribed by the patient's cardiologist, these may include fluid intake management to avoid dehydration, weight control, and exercise (limited to non-burst, noncompetitive sports).[9,10,12,19]



Vasodilators are generally contraindicated in HCM because they may cause hypotension and worsen heart failure symptoms.

Certain medications should be avoided in patients with HCM, especially those with LVOT obstruction. For example, vasodilators are generally contraindicated because they may cause hypotension and worsen heart failure symptoms.[37] These drugs include nitroglycerin, angiotensin-converting enzyme inhibitors, dihydropyridine calcium channel blockers such as amlodipine and nifedipine, and angiotensin II receptor blockers. Similarly, diuretics should be avoided because they reduce preload, resulting in less LV filling and greater LVOT obstruction. Patients with HCM tend to be very preload-dependent, and vasodilatory or diuretic medications may worsen their condition.

Nursing considerations

Patient education is essential to help patients manage HCM and maintain their quality of life. The nurse can provide the following guidelines and information.

• Help the patient understand why certain medications are prescribed and how they will help maintain cardiac function. Be sure the patient understands dosage instructions, potential drug interactions, and adverse reactions. For example, some beta-blockers may cause erectile dysfunction. A patient who experiences this adverse reaction should report it to his provider and discuss what alternative medications are available.

Also explain why patients with HCM should not take nitroglycerin or any other medication that will reduce preload.[9,14] Patients need to understand this in case they experience angina and a well-meaning friend offers some of his or her own nitroglycerin.

• Explain the importance of maintaining hydration, especially in patients with LVOT obstruction. Cardiac output in HCM is particularly dependent upon preload. Dehydration and anything else that reduces preload, such as diuretics, can reduce stroke volume, raising the risk of inadequate cardiac output or other complications.

• Encourage patients to keep their weight at or close to normal in order to minimize myocardial oxygen demand. Inform the patient that any sudden weight gain may indicate fluid retention and should be reported to the provider immediately.

• Because of the risk of SCD, most patients with HCM are advised to avoid competitive sports or any activity that requires bursts of effort or long, sustained high-intensity action.[7,13,26]

• Tell patients that smoking, alcohol, and drug use may worsen signs and symptoms.

• Advise patients to talk with their provider about whether they need to take antibiotics for infective endocarditis prophylaxis before dental procedures. Although current American Heart Association guidelines do not recommend routine prophylaxis for patients with HCM, some practitioners believe that taking this precaution is appropriate, particularly in patients with LVOT obstruction.[37]
• If an ICD has been implanted, be ready to educate the patient and family about its functions and answer their questions.[38] If it is newly implanted, teach them to assess the site for signs and symptoms of infection.
• All patients should have clear instructions on the importance of regular checkups as directed by their cardiologist. Be sure patients understand the importance of immediately reporting any changes in their signs and symptoms, such as irregular heartbeat, syncope, chest pain or anginal equivalent, or peripheral edema.
• Encourage the patient to have all close (first-degree) family members tested for HCM (see *After diagnosis, consider the family*).

Be prepared to refer the patient for postdiagnosis/postsurgical psychological support. HCM can mean significant lifestyle changes that can be difficult to adjust to. Consider referring your patient to outside resources including the Hypertrophic Cardiomyopathy Association website (www.4hcm.org) for support and ongoing research and resources.

Encourage your patient to purchase one of the many medical-alert jewelry services. Wearing medical alert jewelry will let emergency healthcare providers know about the patient's specific health disorders. In addition, some of the service providers, such as the MedicAlert Foundation, have 24/7 access for healthcare providers to get detailed health information on the patient. The MedicAlert Foundation website is www.medicalert.org.

TM's success story

After confirming the HCM diagnosis, TM's cardiologist placed her on trials of medications, including verapamil and atenolol, with no significant relief of signs or symptoms. This qualified her for a surgical correction of her HCM, and she underwent a septal myectomy that successfully reduced her DOE and other signs and symptoms. Today, she has, in her own words, excellent exercise capacity and feels great. She continues to take daily beta-blockers to control dysrhythmias. Regular well-visits over the past 3 years have been positive, and she has not had any additional syncopal episodes. With some healthy lifestyle changes, medication, and regular visits to her cardiologist, TM follows an exercise program and has a healthy, active professional life.

For many patients like TM, HCM no longer carries the poor prognosis that it once did. You can help your patients make the most of the expert care and long-term positive outlook of this once-dreaded disease. ■

After diagnosis, consider the family

Because HCM is an autosomal dominant disorder, all first-degree relatives of the patient should be evaluated for HCM using basic assessment tools: history, physical examination, electrocardiography, and echocardiography.[7,11] If family members prefer, commercial genetic testing is also available to provide information about whether they are genotype-positive or -negative. Teaching points include the following:

- Even in the absence of disease, genotype-positive family members should be advised to have cardiac examinations regularly to detect any changes.[14]
- Family members can have the gene but never develop signs or symptoms.[2,7]
- The results of genetic testing can be inconclusive because many patients do not have an identifiable mutation.[5]

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