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# Hypertrophic Obstructive Cardiomyopathy and Septal Ablation

Susan Steinbis, RN, MSN, CCRN, ACNP-C

**J**.M. is a 75-year-old man with a 9-year history of light-headedness, shortness of breath during exertion, and chest tightness. During the past year, his symptoms have been increasing in frequency. At the time of evaluation, he was unable to walk any distance without shortness of breath and precordial tightness developing. When walking, he experienced less shortness of breath if allowed to lean on something such as a shopping cart. He also had orthopnea, which necessitated sleeping with blocks under the head of his bed. In the past, he had undergone percutaneous transluminal coronary angioplasty and had diagnoses of hyperlipidemia, anemia, and diastolic dysfunction. His medications included 81 mg aspirin daily, 50 mg metoprolol 3 times a day, 1000 mg niacin at bedtime, ascorbic acid, and vitamin E.

Physical examination revealed a grade 3/6 systolic ejection murmur consistent with mitral regurgitation, which had been noted on a previous echocardiogram. The murmur also increased in intensity with the Valsalva maneuver. An electrocardiogram (ECG) revealed left ventricular hypertrophy with strain, which is characterized by a tall R wave in  $V_5$  or  $V_6$  and a large S wave in  $V_1$ . Laboratory studies done at admission revealed thrombocytopenia (platelet count  $96 \times 10^9/L$ ) and anemia (hemoglobin 122 g/L and hematocrit 0.35). His other chemistry results were within normal limits. His cholesterol level was 5.35 mmol/L (207 mg/dL), his high-density lipoprotein level was 1.16 mmol/L (45 mg/dL), and his low-density lipoprotein level was 3.39 mmol/L (131 mg/dL). His last cardiac catheterization had

revealed an elevated left ventricular outflow tract gradient of 95 to 100 mm, and at that time hypertrophic obstructive cardiomyopathy (HOCM) was diagnosed.

### Pathophysiology

HOCM is a thickening of the ventricle in the heart that causes a decrease in blood flow. The hypertrophy that occurs often has obvious cause, such as aortic stenosis or systemic hypertension, and often involves the interventricular septum of the left ventricle.<sup>1,2</sup> The hypertrophy is typically out of proportion to the hemodynamic load, and this finding is the distinguishing feature of the disease.<sup>1</sup> The left ventricle has a stiffness that causes impaired ventricular filling, which in turn causes increased end-diastolic pressure in the left ventricle and eventually pulmonary congestion and dyspnea.<sup>2</sup> Because of left ventricular hypertrophy, the atria dilate and hypertrophy occurs. Patients with HOCM typically have mitral regurgitation because of the septal hypertrophy. The hypertrophy causes the papillary muscle to move out of alignment, thus causing mitral regurgitation. Dyspnea is the most common symptom in HOCM and is caused by the increased stiffness of the ventricle and the increased left ventricular diastolic pressure. Syncope can result from insufficient cardiac output during exertion.<sup>1,3,4</sup> In assessment of patients with dilated cardiomyopathy and HOCM, the main differences between the 2 conditions must be considered (see Table). With dilated cardiomy-

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Differences between hypertrophic obstructive cardiomyopathy and dilated cardiomyopathy

Abnormality	Timing of dysfunction	Left ventricular end-diastolic pressure	Left ventricular systolic function	Ejection fraction	Left ventricular mass
Hypertrophic obstructive cardiomyopathy	Diastolic	Elevated	Markedly increased	Normal or increased	Markedly increased
Dilated cardiomyopathy	Systolic	Normal	Decreased	Decreased	Increased

opathy, the left ventricular end-diastolic volume is increased and end-systolic volume is markedly increased. In HOCM, left ventricular end-diastolic volume is normal and left ventricular end-systolic volume is low.<sup>1</sup>

HOCM is genetic and is associated with high morbidity (1 in 500) and mortality (3% per year for adults and 6% per year for children).<sup>1,5</sup> Sudden cardiac death occurs in approximately 70% of patients who are less than 35 years old.<sup>3</sup> Genetic studies by Christine Seidman<sup>2</sup> indicated that mutations in several genes are responsible for the pathogenesis of HOCM and that HOCM is a disease of the sarcomere. Multiple genes are involved, including genes for cardiac  $\beta$ -myosin heavy chains,  $\alpha$ -tropomyosin, myosin-binding protein C, troponin I, cardiac troponin T, cardiac actin, titin, and the essential and regulatory myosin light chains.<sup>2,3</sup> The mutations are family specific, and most likely many more defects will be found.<sup>2</sup> The study of gene involvement in hypertrophic cardiomyopathy has progressed through the use of mice in which the mutation was induced. The mice have an increase in diastolic stiffness with delayed relaxation and an extended time to reach maximal filling. This finding suggests that dias-

tolic dysfunction is not a result of pathological remodeling, but a direct response to the sarcomere mutation.<sup>2</sup>

### Diagnosis

Two-dimensional echocardiography usually reveals a left ventricular wall that is more than 13 mm thick.<sup>2,4</sup> Diastolic function is typically abnormal, with normal systolic function. Signs and symptoms are related to the diastolic dysfunction and include angina, dyspnea on exertion, and palpitations. As these patients become older, their signs and symptoms usually worsen. The age of patients at onset of hypertrophy varies, but most persons manifest signs by their 30s. Patients with HOCM are definitely at risk for sudden death, and those with a family history of sudden death and/or hypertrophy greater than 35 mm are at an increased risk.<sup>2</sup>

Early evidence obtained from the study previously noted<sup>2</sup> indicates that men have a younger age of onset with a quicker progression of histopathological changes. Vigorous exercise causes detrimental effects such as ventricular tachycardia, ventricular fibrillation, ischemia, and even sudden death.<sup>2,4</sup> These findings may indicate a link between the 2 noted observations: hypertrophic

cardiomyopathy is the most common cause of death in athletes, and most sudden deaths on the sports field occur in men.<sup>2</sup>

### Treatment Medications

Medical therapy is the initial approach used in treating HOCM. The goal is to reduce the effects of the hypercontractile heart. Medications such as  $\beta$ -blockers and calcium channel blockers are used to control signs and symptoms.<sup>6</sup>  $\beta$ -Blockers are used to prevent any increases in the outflow obstruction when patients exercise. These medications are thought to decrease myocardial oxygen consumption and slow heart rate to improve ventricular filling. If  $\beta$ -blockers are discontinued for any reason, patients must be weaned off the medications slowly.<sup>1,3</sup> Non-dihydropyridine calcium channel blockers, such as verapamil and diltiazem, may also be used to treat HOCM. Verapamil improves diastolic filling by improving relaxation of the heart muscle and also increases exercise tolerance in many patients.<sup>1,3</sup> If a patient does not respond to medical therapy, then treatment is aimed at reducing the amount of hypertrophied muscle along the outflow tract.<sup>7</sup>

## Surgery

The standard treatment for HOCM until recently was ventricular septal myectomy. This complex procedure involves open-chest surgery and may have a mortality risk of 1% to 17%, with complications such as complete heart block, severe aortic insufficiency, ventricular septal defect, septal perforation, thromboembolism, and severe mitral regurgitation.<sup>1,8</sup> With this procedure, part of the septum that is hypertrophied is removed, hopefully relieving the obstruction and decreasing the outflow gradient.<sup>1</sup> Beneficial hemodynamic results have occurred that are associated with a decrease in signs and symptoms, but patients continue to have persistent functional limitations. Because of the complications and newer treatments available, ventricular septal myectomy is becoming less popular.<sup>6,7</sup>

## Pacing

Another option for the treatment of HOCM is dual-chamber pacing. The atrioventricular conduction interval is shortened with dual pacing, and this shortening causes an alteration in contraction. This alteration, in turn, minimizes the septal contraction, which is responsible for the narrowing of the left ventricular outflow tract, and the left ventricular outflow tract gradient decreases as a result.<sup>6,8</sup> Dual chamber pacing is referred to as DDD pacing. The first letter shows the chamber in which stimulation occurs (A = atrium; V = ventricle; D = atrium plus ventricle, or dual chamber). The second letter refers to the chamber in which sensing occurs (A = atrium; V = ventricle; D = atrium plus ventricle, or dual chamber). The third letter refers to the mode of sensing or how

the pacemaker responds to a sensed event. In this instance, the third letter indicates dual action. A sensed event can inhibit the output pulse and cause the pacer to recycle, or the output pulse may be triggered in response to a sensed event.<sup>1</sup> The only concern with DDD pacing is that its effects may not be long-term. Drug therapy is often used in combination with DDD pacing to relieve signs and symptoms that occur when patients are at rest. This type of pacing is not effective in patients with atrial arrhythmias.<sup>6,7</sup>

## Percutaneous Transluminal Septal Myocardial Ablation

A nonsurgical therapy called percutaneous transluminal septal myocardial ablation (PT SMA) has been used since 1994 for HOCM in some patients. This procedure reduces the left ventricular outflow tract gradient and thus causes a reduction in signs and symptoms.<sup>3,8-10</sup> PT SMA causes a therapeutic infarction within the septal myocardium in order to reduce the hypertrophy of the septum. The area involved becomes thin and contractile dysfunction develops, reducing the left ventricular outflow tract gradient by expanding the left ventricular outflow tract.<sup>11</sup>

A temporary pacemaker is placed at the start of the procedure. The patient then has cardiac catheterization to visualize and mark the targeted septal perforator(s). An angioplasty guide catheter is inserted into the septal perforator, and the vessel is occluded. Care is taken not to obstruct the left anterior descending branch of the coronary artery. Contrast material is then injected in an attempt to localize the area in the septal perforator and to verify

that no contrast material is leaking into the left anterior descending branch. Transthoracic echocardiography is performed to assess for a reduction in the left ventricular outflow tract gradient while the balloon is inflated.

Once this procedure is done, 3 to 5 mL of 98% ethanol is injected into the septal perforator. The balloon remains inflated throughout this period, and the alcohol remains in place for 10 minutes. At the end of 10 minutes, the catheter is flushed with saline and the balloon is deflated and removed. The left ventricular outflow tract gradient is measured and contrast material is again injected into the left anterior descending branch to confirm 100% stenosis of the septal branch. Transthoracic echocardiography is repeated to reassess the left ventricular outflow tract gradient. The patient then proceeds to the coronary care unit, where he or she is monitored for 24 to 48 hours. If the cardiac rhythm remains stable after 24 to 48 hours, the temporary pacemaker is removed and the patient can be discharged home.

Levels of creatine kinase-MB and troponin I are measured and an ECG is obtained after the procedure. The level of cardiac isoenzymes should increase because of the therapeutic infarct. The ECG should show changes consistent with a septal infarct, including Q waves in V<sub>1</sub> and V<sub>2</sub> that are at least 25% of the amplitude of the following R wave.<sup>1</sup> At the time of discharge, the patient is told to take a  $\beta$ -blocker and aspirin.<sup>6,12</sup> Follow-up studies<sup>8,11</sup> have indicated an improvement in left ventricular end-diastolic pressure and a decrease in pulmonary artery pressures.

Complications of PTSMA include arrhythmias, complete heart block, cerebrovascular accident, hypotension, and cardiac tamponade. Contraindications to septal ablation include inadequate septal thickness (<18 mm), right bundle branch block, and intrinsic mitral valve diseases.<sup>3,7</sup> Mortality rates associated with PTSMA have ranged between 0% and 4%.<sup>4</sup>

### Nursing Interventions

Nursing care for patients who have PTSMA revolves around monitoring of enzyme levels, ECG findings, and vital signs. Regular monitoring of the catheter site and distal pulses after cardiac catheterization is done per each facility's routine orders as well. After the procedure, patients are monitored in an intensive care unit for 24 hours, and then they are transferred to a telemetry unit. Levels of cardiac enzymes and ECG findings are assessed every 4 hours for 24 hours. Vital signs are monitored every hour for 4 hours and then every 2 to 4 hours, depending on the physician's preference.  $\beta$ -Blockers and aspirin are continued at the time of discharge from the hospital. Follow-up consists of an initial visit at 6 weeks and every 3 months for a year after that and then yearly visits. Again, the physician's preference dictates the frequency of visits.<sup>6,11</sup>

### Patient's Course

J.M. had been treated with metoprolol, and increases in metoprolol had not been tolerated. Because of the increasing signs and symptoms, he was evaluated for PTSMA. He agreed to have the procedure and underwent ablation of his first septal perforator. No heart block was noted during or after the procedure.

Echocardiography revealed a decrease in left ventricular outflow tract gradient from 95 mm Hg to 25 mm Hg after the procedure. Imaging after ablation revealed 100% stenosis of the septal artery. After the procedure, he had an increase in the level of creatine kinase-MB to 173 U/L, and his course after the procedure was uneventful. The peak level of creatine kinase after the procedure can be as high as 200 U/L, with creatine kinase-MB up to 280 U/L.<sup>7,11</sup> J.M.'s temporary pacemaker was removed after 48 hours. His signs and symptoms improved markedly, as evidenced by his increase in ambulation without chest pain or shortness of breath. He continued to take metoprolol and aspirin and subsequently was discharged home in stable condition.

### Future Studies

The long-term complications of PTSMA are unknown. Patients with HOCM continue to remain prone to arrhythmias, such as atrial fibrillation, nonsustained ventricular tachycardia, ventricular fibrillation, and sudden death despite interventions.<sup>4,7</sup> PTSMA may increase the risk of arrhythmias because of the induced myocardial infarction and the electromechanical remodeling that occurs. The electromechanical remodeling raises some concern for the long-term risk associated with the procedure, especially in younger patients.<sup>8</sup> Signs and symptoms of HOCM may also be intensified by mitral regurgitation.<sup>1,7</sup> This possibility should be kept in mind and evaluated in patients who continue to have signs and symptoms after intervention. New studies will be done as potential treatment options

for HOCM emerge. Genetic analyses and genotyping will also lead the field to interventions that may modify the disease-producing mutations. Currently, good results are being obtained with PTSMA, and the number of procedures done will continue to increase as more clinicians become skilled in septal ablation.

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