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Systolic Heart Failure in a Patient With Hypertrophic Obstructive Cardiomyopathy: A Potentially Life-Threatening Complication

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Systolic Heart Failure in a Patient With Hypertrophic Obstructive Cardiomyopathy

A Potentially Life-Threatening Complication

Shannon Etheridge Whitten, MS, NP-C, ARPN, BC, CCRN

PRIME POINTS

- Hypertrophic obstructive cardiomyopathy is characterized by obstruction of the left ventricular outflow tract, ventricular hypertrophy, and volume overload.
- The disease is incurable; treatment is focused on improving patients' signs and symptoms and decreasing complications.
- Complications include heart failure, atrial and ventricular arrhythmias, and sudden cardiac death.
- Percutaneous transluminal septal myocardial ablation and surgical myectomy are good options for patients who do not respond to medications.

CASE STUDY

LB, a 61-year-old man, well known in our cardiology practice, came to the emergency department early one morning after experiencing 24 hours of profound weakness and increasing dyspnea out of proportion to his usual shortness of breath. He had a history of hypertrophic obstructive cardiomyopathy and paroxysmal atrial arrhythmias. He said he had no personal or family history of coronary artery disease (CAD), and a recent coronary angiogram showed no evidence of CAD. Coincidentally, he was scheduled to return later the same day for a septal ablation because his signs and symptoms were not improving with aggressive medical therapy that included the use of high-dose β -blockers and calcium channel blockers. An echocardiogram 1 week earlier had revealed an ejection fraction of 70%, an asymmetric thickening of the intracardiac septum, and marked systolic outflow obstruction with a gradient of almost 100 mm Hg on exertion. On arrival in the emergency department,

LB was diaphoretic with a systolic blood pressure of 60 mm Hg, a low-grade fever, and sinus tachycardia with a heart rate of 122/min. His dual-chamber, inhibited response, adaptive rate pacemaker was functioning appropriately. LB had not experienced any chest discomfort or had any indications of infection that

CE Continuing Education

This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Identify the pathophysiologic characteristics of hypertrophic obstructive cardiomyopathy (HOCM)
2. Describe common signs and symptoms of HOCM
3. Discuss the diagnostic and treatment modalities for HOCM

Discussion

Epidemiology

Hypertrophic obstructive cardiomyopathy (HOCM) is a genetically transmitted disease that affects approximately 0.2% to 4% of the US population.¹ It is the result of a mutation in multiple genes that encode

protein in the cardiac sarcomere, the primary contractile unit of the cardiac muscle.² HOCM is typically inherited in an autosomal manner, with variable penetrance and variable expressivity.³ Approximately half of all cases of HOCM have an autosomal dominant pattern; the rest are the

result of sporadic gene mutations.^{3,4} Recently, HOCM was linked to the cardiac myosin heavy-chain genes on chromosome 14 in some families, suggesting genetic heterogeneity.⁵ The age at which clinical manifestations become apparent varies. The existence of different disease genes or mutations in a gene may account for the differences in genotypic and phenotypic expressions.³

Morphological evidence of disease is visible on echocardiograms in approximately 25% of first-degree relatives of patients with HOCM.^{2,5} Genetic testing is still in its early

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could explain this precipitous exacerbation in his signs and symptoms.

Physical examination revealed diffuse crackles throughout all lung fields and profound dyspnea with an oxygen saturation of 87%. He had a harsh IV/VI systolic ejection murmur that intensified when he performed the Valsalva maneuver. An echocardiogram revealed an ejection fraction of 15% to 20% with anterior septal, apical, and lateral wall hypokinesis. LB was clearly in cardiogenic shock of unknown origin. The acute care nurse practitioner made arrangements for urgent transfer to the coronary care unit for stabilization because LB was decompensating quickly.

Laboratory results indicated a white blood cell count of 11 000/uL, a creatine kinase level of 2034 U/L (to convert to microkatal per liter, multiply by 0.0167), an MB fraction of 53 ng/mL, and a troponin level of 23.6 ng/mL. His renal function was stable, with a creatinine level of 1.3 mg/dL (to convert to micromoles per liter, multiply by 884). Blood, urine, and sputum cultures showed no growth of microorganisms, so the leukocytosis and fever were suspected to be a reactive process. Electrocardiography (ECG) revealed left ventricular hypertrophy, and 1-mm ST-segment depression was present in all the precordial leads. Comparison with an earlier ECG confirmed that the

ST-segment depression was a new finding. A pulmonary artery catheter was placed for continuous hemodynamic monitoring, and administration of dopamine was started for treatment of hypotension. Intravenous furosemide (Lasix) was started cautiously to treat the pulmonary edema.

LB was taken to the cardiac catheterization laboratory and, as expected, did not have any evidence of coronary artery stenosis or thrombus. Because of the normal findings on the coronary angiogram, the cause of the cardiogenic shock was thought to be myocardial stunning. An intra-aortic balloon pump was successfully placed at a ratio of 1:2, providing further hemodynamic stabilization. The intravenous infusion of heparin started in the catheterization laboratory was continued, and LB returned to the coronary care unit. On his second day in the unit, atrial fibrillation with a rapid ventricular response developed. Treatment with β -blockers was resumed for rate control, and intravenous infusion of digoxin was started to provide positive inotropic stimulation for the diminished left ventricular function. LB was weaned off the balloon pump, mechanical ventilation, and dopamine, which were eventually discontinued without complications.

Two weeks later, a repeat echocardiogram revealed an ejection fraction of 50% with

stages; however, it can be used to detect asymptomatic family members with the same mutation. Patients with a known family history of HOCM should be considered for noninvasive screening, especially children and adolescents up to 18 years old, because HOCM is more progressive in children and young adults.^{2,3} An echocardiogram will usually suffice as an initial screening tool in this population.²

Pathophysiology

HOCM is the obstructive subvariant of hypertrophic cardiomyopathy.

It is characterized by left ventricular hypertrophy, a hyperdynamic left ventricle, systolic anterior motion of the mitral valve, and outflow obstruction in the absence of other identifiable diseases.^{6,7} Mutations in the myocardial sarcomere proteins result in muscle disarray and fibrosis, ultimately causing inappropriate left ventricular hypertrophy.^{1,3} The term HOCM is preferred to the older term idiopathic hypertrophic subaortic stenosis because hypertrophy can occur in any segment of the ventricle and interventricular septum, not just the subaortic septum.⁵

Hypertrophy of the subaortic septum alone or in combination with systolic anterior motion of the mitral valve produces obstruction of the left ventricular outflow tract (LVOT) that results in decreased cardiac output. Bulging of thickened septum into the outflow tract during systole causes anterior apposition of the mitral valve leaflet, further narrowing the outflow tract and creating an amplifying feedback loop whereby obstruction begets more obstruction^{1,8} (Figure 1). This obstruction increases left ventricular systolic pressure, decreases coronary perfusion pressure, and increases left ventricular end-diastolic pressure (LVEDP) in the absence of increased volume.¹⁰ Most patients with HOCM have a thick hypercontractile left ventricle and impaired diastolic relaxation. Ventricular hypertrophy is usually out of proportion to the hemodynamic load, meaning the LVEDP is elevated even with normal left ventricular volume.¹¹ The abnormally elevated LVEDP may lead to backward failure with pulmonary congestion, dyspnea, and even heart failure. In most cases, heart failure is due to diastolic dysfunction (Figure 2). Systolic function is usually preserved, even in advanced cases; importantly, the LVOT obstruction in HOCM is not fixed, and therefore a sudden variation in the amount of obstruction can lead to serious complications,¹⁰ such as the systolic failure in this case study. The obstruction varies considerably from day to day even in patients in stable condition and can change with the slightest alteration in physiological state, such as with exercise or illness.

LB had a significant outflow obstruction from asymmetric

midanterior apical hypokinesis that was clearly an improvement and an indication that the myocardium was stunned, not infarcted. Systolic anterior motion of the mitral valve was evident, and the peak systolic outflow gradient was 55 mm Hg at rest. LB was taken to the catheterization laboratory for reevaluation. His resting systolic gradient increased when he performed the Valsalva maneuver and peaked at 100 mm Hg with a dobutamine challenge. On the basis of these additional findings, a decision was made to proceed with percutaneous transluminal septal myocardial ablation (PTSMA). Per protocol, 98% ethanol (3 mL) was injected into a large first septal perforator artery with the catheter balloon inflated to prevent untoward effects. The septal ablation was successful; the left ventricular outflow gradient decreased to 40 mm Hg.

LB returned to the coronary care unit for observation overnight, which is standard. The same evening, a paroxysmal atrial flutter with a rapid ventricular response developed. With the addition of intravenous amiodarone, LB's cardiac rhythm converted to normal sinus rhythm. His pacemaker was reprogrammed to reduce the sensitivity of the rate response to allow for his own intrinsic beats. The rationale was that the atrial kick of late diastole produced by his intrinsic rhythm would allow more effective diastolic filling. At discharge to home, he was taking a long-acting β -blocker, amiodarone, and warfarin. Arrangements were made for follow-up with his primary cardiologist 1 week after discharge.

hypertrophy of the subaortic septum that was further compromised by systolic anterior motion of the mitral valve. In his case, the cardiogenic shock was attributed to myocardial stunning, the result of a critical change in the LVOT obstruction. Myocardial stunning is a phenomenon in which short recurrent, reversible episodes of ischemia result in prolonged depression of cardiac function that eventually recovers with time.⁴ The stunning is the result of oxidative stress, namely, the production of oxygen-derived free radicals during reperfusion of an ischemic myocardium.⁴ The exact incidence of myocardial stunning is unknown, but it is generally well tolerated in patients with normal structure and function of the left ventricle.^{4,12}

LB's manifestation was unique because his ejection fraction decreased precipitously from 70% to approximately 15% to 20%, not allowing time for compensation. Myocardial stunning in a patient whose cardiac output was already compromised by obstruction only complicated matters by further reducing cardiac output, leading to pulmonary congestion and cardiogenic shock.

Clinical

Manifestations

Most patients with HOCM are asymptomatic or only minimally symptomatic; the most common findings are dyspnea and chest pain, in that order.⁶ Shortness of breath usually correlates with the heart's inability to increase cardiac output, particularly upon exertion.¹³ Orthopnea and paroxysmal nocturnal dyspnea are less common and are thought to be due to pulmonary venous congestion.

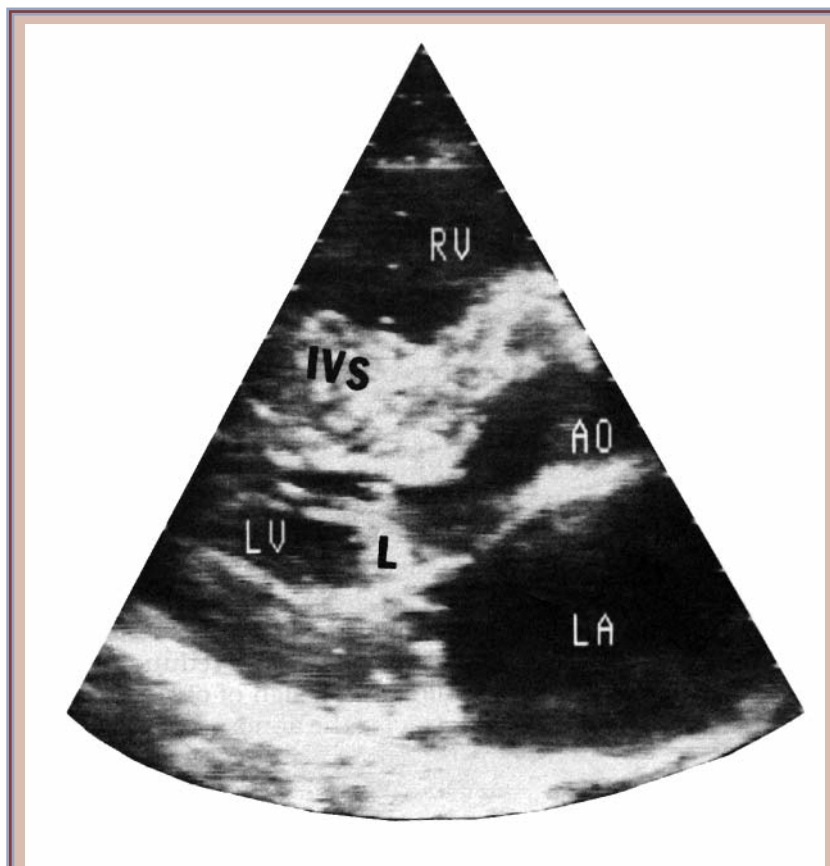


Figure 1 Classic features of hypertrophic obstructive cardiomyopathy. Long-axis view of obstruction of the left ventricular outflow tract by a thickened interventricular septum. Note the systolic anterior motion of the mitral valve obstructing the left ventricular outflow tract.

Abbreviations: AO, aorta; IVS, interventricular septum; L, anterior leaflet of the mitral valve; LA, left atrium; LV, left ventricle; RV, right ventricle.

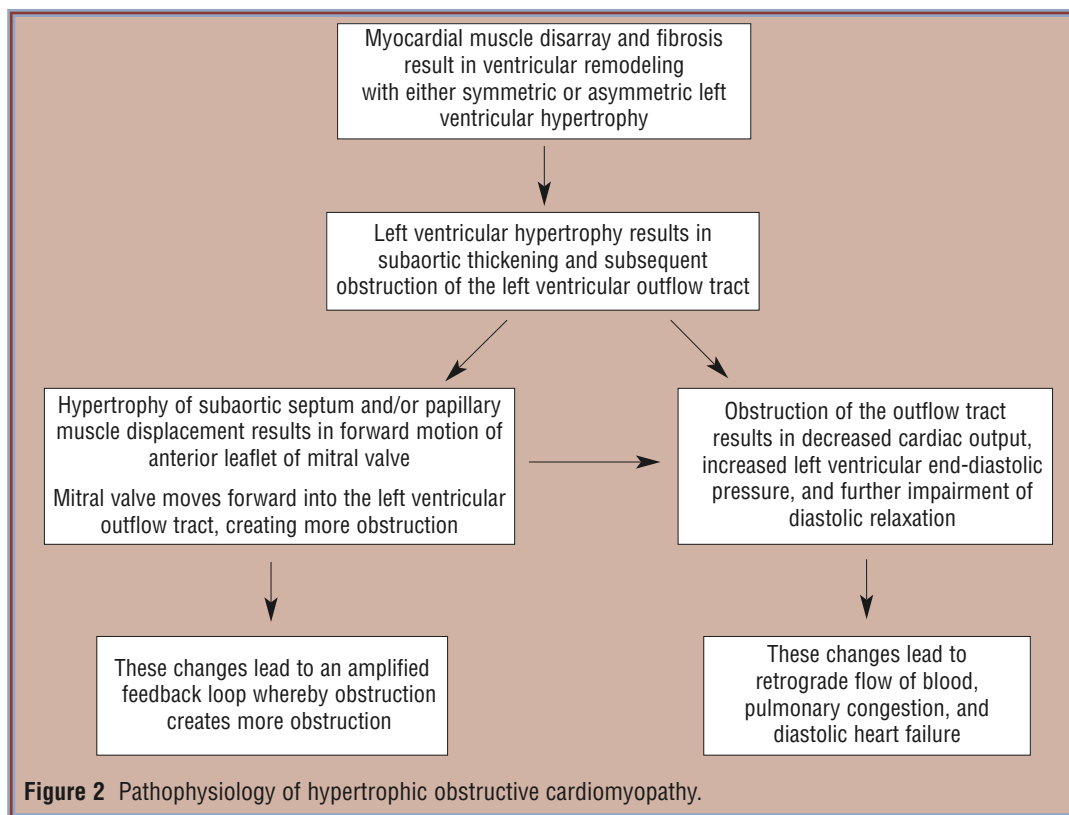
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The cause of chest pain is less clear but it may stem from decreased perfusion in the microcoronary circulation.¹⁴ This perfusion defect is thought to be the result of phasic compression of intramural vessels and dynamic coronary bridges by the hypercontractile left ventricle.⁸ Myocardial ischemia has been linked to the increase in oxygen demand associated with small-vessel coronary artery disease. Chest pain typically occurs in response to activity or strenuous exercise but may occur at rest.⁸ The pain varies in quality from pressure to crushing pain and typi-

cally improves with rest or cessation of the offending activity.

Syncope and presyncope are common and may occur as the result of both atrial and ventricular arrhythmias, heart block, decreased cerebral perfusion, or even abnormal reflex vasodilatation during or after physical activity.^{8,10} Congestive heart failure is usually the result of increasing LVOT obstruction and impaired diastolic relaxation that lead to increased filling pressures, which in turn cause pulmonary and systemic congestion.

Palpitations are sometimes just the patient's sensation of strong



A bifid apex and a bifid carotid pulse may also be detected on physical examination. A bifid apex is a powerful systolic impulse or thrust created by the increased muscle mass of the hypertrophied left ventricle and may be detected on palpation.^{5,10} A bifid pulse may be evident particularly in the carotid arteries.

contractions; however, atrial or ventricular arrhythmias are more often the real culprit.³ Arrhythmias are thought to be the product of ventricular remodeling, decreased cardiac output, microcoronary ischemia, and hypotension.¹⁰ Arrhythmias generally are not well tolerated because they further contribute to reductions in cardiac output. The most common arrhythmias seen in HOCM are atrial fibrillation, atrial flutter, supraventricular tachycardia, ventricular tachycardia, and heart block of various degrees. Sudden cardiac death from ventricular arrhythmias is not uncommon and, in fact, is the leading cause of sudden cardiac death in patients age 35 years or younger.^{3,10}

Findings on Physical Examination

Physical examination of a patient with HOCM should be focused on

arterial pulsations and auscultation.³ The most common finding is a systolic ejection murmur that varies in intensity and is best heard at the sternal border and/or apex.⁵ As a rule of thumb, anything that increases venous return decreases the murmur's intensity, and anything that decreases venous return increases the murmur's intensity. The murmur usually increases with standing, with exercise, or when the Valsalva maneuver is performed, and it decreases with squatting, gripping the hand, or use of vasopressors. The holosystolic murmur of mitral regurgitation is best heard at the apex and axilla, and it is often heard in patients with significant LVOT obstruction.^{2,10} Other common findings include the S₄ gallop of ventricular noncompliance and paradoxical splitting of the second heart sound from delayed closure of the aortic valve (Table 1).

The cause is the rapid increase in pressure through the outflow tract in early systole, followed by a decrease during midsystole as the gradient increases, and finally followed by another increase in pressure during late systole.¹⁰ Common hemodynamic findings in a patient with HOCM include decreased cardiac output and increased LVEDP. Pulmonary artery pressures may be elevated in those patients with diastolic and systolic heart failure.

Diagnosis

Echocardiography is the test of choice for the initial diagnosis.¹⁵ Most patients with HOCM have echocardiographic evidence of septal hypertrophy, and this test has a sensitivity of 90% for detection of HOCM.³ Table 2 gives additional diagnostic echocardiographic findings.^{5,10} A chest radiograph may reveal

an enlarged cardiac silhouette. ECG changes are seen in up to 95% of patients with HOCM.³ The classic ECG findings include typical left ventricular hypertrophy with or without strain in leads V₂ through V₆, marked left-axis deviation; deep, narrow q waves in the leftward-oriented leads (aVL and V₆); and left atrial enlargement as indicated by terminal P-wave negativity in lead V₁.¹⁷

Cardiac catheterization is another useful test but is usually reserved for patients who are not responding to medical therapy. Catheterization can be used to assess the degree of outflow obstruction in patients with refractory disease.² It is also useful to determine coronary anatomy before intervention. Electrophysiology studies are rarely used in diagnosis but can be useful to determine which patients are at high risk for life-threatening ventricular arrhythmias.³ Finally, exercise testing is not routinely used in patients with HOCM because of the increased risk for syncope, hypotension, and arrhythmias.⁵

Treatment Strategies

The treatment goal for patients with HOCM is to improve signs and symptoms by decreasing heart rate, decreasing outflow obstruction, decreasing oxygen demand, improving left ventricular and septal relaxation, improving filling parameters, and preventing major complications.¹⁰ β -Blockers, nondihydropyridine calcium channel blockers such as verapamil, disopyramide (Norpace), and amiodarone are the preferred medications to treat this disease.¹ β -Blockade is useful in preventing an exercise-related increase in pressure gradient but is less help-

ful in reducing high resting gradients.¹⁰ Furthermore, β -blockade can markedly decrease outflow obstruction by promoting septal relaxation. Disopyramide, on the other hand, is used to reduce resting gradients and is best used in combination with a β -blocker.^{10,18} Amiodarone was selected for LB in an attempt to

Table 1 Clinical manifestations and findings on physical examination of patients with hypertrophic obstructive cardiomyopathy

Feature	Cause
Syncope	Inadequate cardiac output and/or cardiac arrhythmias
Dyspnea	Left ventricular diastolic dysfunction and impaired ventricular filling cause an elevation in left atrial and pulmonary venous pressures, resulting in pulmonary congestion
Chest pain	Microcoronary artery perfusion defects resulting in ischemia
Palpitations	Ventricular remodeling, decreased cardiac output, microcoronary ischemia, and hypotension
Systolic ejection murmur	Turbulent blood flow through the obstructed left ventricular outflow tract
Holosystolic murmur	Mitral valve regurgitation
S ₄ gallop	A thickened noncompliant left ventricle
Paradoxical splitting S ₂	Splitting of the second heart sound during expiration rather than during inspiration (physiological splitting), caused by delayed closure in the aortic valve resulting in the aortic valve closing after the pulmonic valve
Bifid carotid pulse	Brisk increase in carotid pressure during early systole, followed by a sharp decline in pressure during midsystole, followed once again by a sharp increase in carotid pressure during late systole
Prominent a wave on jugular venous pulse	Diminished right ventricular compliance due to severe septal hypertrophy

Table 2 Echocardiographic findings in patients with hypertrophic obstructive cardiomyopathy

Two-dimensional echocardiography
Massive LV hypertrophy
Asymmetric LV wall thickness
Asymmetric or symmetric thickening of the interventricular septum
Normal cavity size
Dilated left atrium
Hyperdynamic LV function as evidenced by an EF% of 70
Systolic anterior motion of the mitral valve leaflet
Thickened elongated anterior leaflet
Endocardial thickening of LVOT
Hypodynamic basal septum
Doppler echocardiography
Mitral valve regurgitation
Mitral inflow: diastolic dysfunction pattern with impaired relaxation
Pulsed-wave Doppler
High velocities in the LVOT
Color-flow Doppler
High LV outflow velocities
Continuous wave Doppler
Dagger-shaped velocity waveform in LVOT

Abbreviations: EF%, ejection fraction; LV, left ventricle; LVOT, left ventricular outflow tract.
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convert his rhythm to sinus rhythm and to decrease the risk for sudden cardiac death from arrhythmias.

Surgical myectomy and PTSMA are invasive procedures usually reserved for patients in whom medical management is unsuccessful.¹⁷ Surgical myectomy involves excision of a rectangular part of the thickened subaortic septum. For patients with minimal septal hypertrophy (15-18 mm), a mitral valve replacement is preferred over myectomy because of the risk of septal perforation.¹ Surgical myectomy ameliorates signs and symptoms in about 70% of patients and is the preferred procedure for patients who have concomitant cardiac disease that requires surgery.¹¹

PTSMA is a nonsurgical alternative for patients in whom traditional medical therapy has not been helpful or who are deemed too high risk for surgery. PTSMA is indicated for symptomatic patients with New York Heart Association class III heart failure; however, patients with less severe signs and symptoms are considered if they have high outflow tract gradients or documented risk factors for sudden cardiac death. An outflow tract obstruction is considered significant if the gradient is greater than 50 mm Hg at rest or is 30 mm Hg at rest and increases to 80 mm Hg under stress.^{1,13} PTSMA involves injection of ethanol into 1 or more septal perforator arteries, producing a controlled infarction of the myocardial septum. A successful PTSMA results in septal thinning with reduction in the LVOT obstruction. Abolishing the gradient during the procedure is not necessary because remodeling after ablation shrinks the subaortic septum further.¹⁹ Although the long-term safety

Table 3 Disease management

Problem	Management
Outflow tract obstruction and impaired ventricular relaxation	β -Adrenergic blockade, calcium channel blockers, and disopyramide (Norpace); surgical myectomy, percutaneous transluminal septal myocardial ablation, and cardiac pacemaker implantation are options for patients who do not respond to medical therapy
Ventricular arrhythmias	Amiodarone with or without an implantable cardiac defibrillator can reduce sudden cardiac deaths in high-risk patients
Atrial arrhythmias	β -Blockers and amiodarone are used
Diastolic heart failure	β -Blockers, calcium channel blockers, and judicious use of diuretics; occasionally α -agonists are used for patients with severe pulmonary congestion; α -agonists increase the size of the outflow tract and decrease the outflow tract gradient; however, they must be used at lower doses to prevent inotropic effects
Outflow tract obstruction due to systolic anterior motion of the mitral valve	Mitral valve replacement may be indicated in patients with congestive heart failure due to severe mitral regurgitation or in patients with pulmonary hypertension due to mitral regurgitation

of PTSMA has been established in clinical trials, the risk for mortality is 1.1% to 4%, and the procedure is slightly less effective than myectomy for long-term control of signs and symptoms.¹³ Furthermore, up to 10% of patients require a permanent pacemaker/defibrillator after PTSMA because of arrhythmias or heart block.²⁰

Finally, dual-chamber, inhibited response, adaptive rate pacemakers may be used to decrease septal contractility, but at best provide only modest improvements in LVOT obstruction.² Even so, pacemakers may have another useful role in HOCM. A recent study²¹ suggests left ventricular endocardial temporary pacing may be useful in predicting patients' response to PTSMA. Implantable cardiac defibrillators with or without the use of amiodarone are another useful treatment and do prevent cardiac death in high-risk populations.^{3,10} Adolescents, children, and patients more than 65 years old are considered high risk.³

Contrary to the standard medical management of HOCM, digoxin, vasopressors, and a balloon pump were required in LB's case to maintain adequate filling pressures and forward flow. Vasodilators, diuretics, positive inotropic medications, and balloon pumps normally would increase the LVOT obstruction in a patient with HOCM and therefore should be avoided; however, they were necessary to treat the cardiogenic shock in LB.

Nursing Considerations

Critical care nurses must understand the pathophysiology, management (Table 3), and dynamic features of HOCM to individualize a plan of care for patients and to prevent potential life-threatening complications. Nursing care after myectomy is similar to postoperative care of any patient undergoing invasive heart surgery.

Patients are admitted to the intensive care unit after PTSMA for no less than 24 hours (Table 4).

Table 4 Nursing protocols after percutaneous transluminal septal myocardial ablation

Obtain a baseline 12-lead electrocardiogram, complete blood cell count, basic metabolic panel, and full set of vital signs before the procedure
Assess vital signs every 30 to 60 minutes after the procedure, then every 2 hours
Obtain an electrocardiogram every hour after the procedure for 4 hours unless otherwise specified by unit protocol
Assess cardiac enzyme levels immediately after procedure then every 8 hours for 3 sets unless otherwise specified by the cardiologist
Perform hemodynamic monitoring if indicated for patients in overt heart failure or cardiogenic shock
Monitor intravenous catheters and insertion sites per unit protocol
Check catheterization site for bleeding, hematoma, infection, and pseudoaneurysm
Assess patients for pain because they are experiencing a myocardial infarction; remember the analgesics of choice are morphine sulfate and nonsteroidal anti-inflammatory drugs (nitroglycerin must be used cautiously in patients with high residual gradients due to decreased cardiac output)
Monitor for arrhythmias
Inspect transvenous pacemaker for appropriate function and response of patient
Initiate discharge planning on admission to intensive care unit; education should include discharge medications, management of complications, activity, and follow-up; patients taking anticoagulants should receive education and be given instructions on monitoring prothrombin time and international normalized ratio; patients should follow up with their cardiologist within 2 to 4 weeks of discharge

Elderly patients and patients with class IV heart failure have a higher risk of complications than others do after the intervention and typically require additional time in the unit.¹⁹ Vital signs are monitored every 30 to 60 minutes for 4 hours, and then monitoring is tapered to every 2 hours unless otherwise specified. Patients with overt heart failure may require monitoring of cardiac output, systemic vascular resistance, and pulmonary artery wedge pressures to guide medical therapy. Assessment of catheter insertion sites for bleeding, hematomas, and signs of infection is important. Access sites are managed the same as any site after any catheterization procedure.

ECG should be performed before ablation, when the patient arrives in the unit, and hourly for 4 hours. An ECG should be ordered if the

patient has indications of extension of the myocardial infarction because this change is a potentially life-threatening complication of PTSMA. Serial measurements of cardiac enzyme levels should be done per preference of the cardiologist or per unit protocol. Patients after ablation are experiencing an infarct and require intensive monitoring. They should be assessed for chest pain and dyspnea. Chest pain is common, and management usually involves the use of opiate analgesics such as morphine sulfate, nonsteroidal anti-inflammatory drugs, or both. Patients should be monitored for arrhythmias; any patient who has undergone PTSMA is at increased risk for atrial and ventricular arrhythmias.

To prevent deleterious complications, critical care nurses must be clear on medications that are indi-

cated and contraindicated for patients with HOCM. The use of inotropes, angiotensin-converting enzyme inhibitors, and vasodilators such as nitroglycerin should be avoided and questioned if they are ordered for a patient with HOCM. Nitrates may be ordered for chest pain after PTSMA, but their use should be clarified because nitrates are contraindicated in any patient with a significant gradient. If patients are given antiplatelet or anticoagulant therapy, they must be monitored closely for bleeding complications, drug reactions, and medication interactions.

Patients are typically transferred to a telemetry step-down unit after the initial 24-hour observation period once the transvenous pacemaker has been discontinued, but patients are still at risk for complications and should be monitored closely until discharge. Discharge planning should start when the patient arrives in the unit. Patients must understand which medications they are to continue and which ones they should discontinue, because their disease process may have changed dramatically after successful intervention. Treatment with β -blockers is usually continued after discharge. Patients must be instructed to follow up with their cardiologist or nurse practitioner within 2 to 4 weeks of discharge unless otherwise instructed. Patients with any marked residual obstruction must be counseled to avoid strenuous activity and contact sports because of the risk of sudden cardiac death. Finally, patients should be instructed to seek additional medical attention if they experience any indications of infection, pain, or alteration in temperature/sensation in the catheterized extremity.

Summary

HOCM is a genetic disease characterized by LVOT obstruction, ventricular hypertrophy, and volume overload. Its defining characteristics are a hyperdynamic left ventricle with or without septal hypertrophy and dynamic obstruction of the outflow tract. The most common findings include dyspnea, chest pain, and syncope, and these are usually the result of the heart's inability to increase cardiac output with activity.¹⁰ Because HOCM is not curable, the goals of treatment are focused on improving patients' signs and symptoms and decreasing complications.¹

Patients with HOCM require lifelong medical therapy; therefore, medical and interventional therapies are considered complementary, not competitive.¹⁹ PTSMAs and surgical myectomy are good options for patients with medication-refractory disease, but because of the potential complications, these interventions must be used only in the appropriate patients. Complications of HOCM include, but are not limited to, heart failure, atrial and ventricular arrhythmias, and sudden cardiac death. Although HOCM is a disease typified by diastolic dysfunction, complications may occur with progressive or uncompensated disease, resulting in systolic heart failure as in LB's case. Critical care nurses' over-

all knowledge of this disease and their ability to detect problems early can make every bit of difference for the patient, as it did in this case. LB has continued to do well without any further complications thanks to the diligence of his health care team. **CCN**

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Financial Disclosures

None reported.

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To learn more about hypertrophic obstructive cardiomyopathy, read "Cardiogenic Shock in a Patient With Hypertrophic Obstructive Cardiomyopathy After Insertion of a Pacemaker" by Anna Barkman and Judy McCay in the *American Journal of Critical Care*, 2002;11:537-542. Available at www.ajconline.org.

CE Test Test ID C0852: Systolic Heart Failure in a Patient With Hypertrophic Obstructive Cardiomyopathy

Learning objectives: 1. Identify the pathophysiologic characteristics of hypertrophic obstructive cardiomyopathy (HOCM) 2. Describe common signs and symptoms of HOCM 3. Discuss the diagnostic and treatment modalities for HOCM

1. Which of the following correctly describes hypertrophic obstructive cardiomyopathy (HOCM)?
 - a. HOCM is a sex-linked recessive genetic trait.
 - b. HOCM is always the result of the same genetic mutation.
 - c. HOCM is a genetically transmitted disease that affects approximately 0.2% to 4% of the US population.
 - d. HOCM is the result of a mutation in a single gene that encodes protein in the cardiac sarcomere.
2. Which of the following describe the characteristics of HOCM?
 - a. Right ventricular (RV) hypertrophy, hyperdynamic right ventricle, systolic anterior motion of the mitral valve, and outflow obstruction
 - b. Left ventricular (LV) hypertrophy, hyperdynamic left ventricle, systolic anterior motion of the mitral valve, and outflow obstruction
 - c. LV hypertrophy, hyperdynamic left ventricle, systolic anterior motion of the tricuspid valve, and outflow obstruction
 - d. RV hypertrophy, hyperdynamic left ventricle, systolic anterior motion of the tricuspid valve, and outflow obstruction
3. What causes decreased cardiac output in patients with HOCM?
 - a. Obstruction of the LV outflow tract produced by a hypertrophic subaortic septum alone or in combination with systolic anterior motion of the mitral valve
 - b. Obstruction of the RV outflow tract produced by a hypertrophic subaortic septum alone or in combination with systolic anterior motion of the tricuspid valve
 - c. Increased LV systolic pressure, decreased coronary perfusion pressure, and increased LV end-diastolic pressure in the absence of increased volume
 - d. The thick hypercontractile left ventricle and impaired diastolic relaxation
4. Which of the following statements is accurate about LV outflow tract obstruction in patients with HOCM?
 - a. The obstruction is fixed and does not vary with exercise or illness.
 - b. The obstruction is not fixed and can change with alterations in physiologic state.
 - c. The obstruction is not fixed but is not impacted by physiologic state.
 - d. The obstruction is fixed but can change with alterations in physiologic state.
5. What is the most common reason for heart failure in HOCM?
 - a. Systolic dysfunction
 - b. Combined systolic and diastolic dysfunction
 - c. Inadequate coronary perfusion
 - d. Diastolic dysfunction
6. What are the symptoms most commonly found in patients with HOCM?
 - a. Dyspnea and chest pain
 - b. Orthopnea and chest pain
 - c. Paroxysmal nocturnal dyspnea and chest pain
 - d. Dyspnea and cough
7. Which of the following are causes of syncope and presyncope in the patient with HOCM?
 - a. Heart failure
 - b. Myocardial ischemia
 - c. Atrial and ventricular arrhythmias
 - d. Increased cerebral perfusion
8. What is the leading cause of sudden cardiac death in patients age 35 years or younger?
 - a. Ventricular arrhythmias associated with HOCM
 - b. Heart block associated with HOCM
 - c. Supraventricular tachycardia associated with HOCM
 - d. Atrial fibrillation associated with HOCM
9. What is the most common finding on physical examination in a patient with HOCM?
 - a. Diastolic murmur
 - b. Systolic ejection murmur
 - c. S3 and S4
 - d. Pansystolic murmur
10. What are the most common hemodynamic findings in a patient with HOCM?
 - a. Increased cardiac output and increased left ventricular end-diastolic pressure (LVEDP)
 - b. Decreased cardiac output and decreased LVEDP
 - c. Increased cardiac output and decreased LVEDP
 - d. Decreased cardiac output and increased LVEDP
11. What diagnostic test is the test of choice for the initial diagnosis of HOCM?
 - a. Echocardiogram
 - b. Electrocardiogram
 - c. Tilt table test
 - d. Cardiac catheterization
12. Which of the following is NOT a treatment goal for patients with HOCM?
 - a. Decrease heart rate
 - b. Decrease outflow obstruction
 - c. Increase oxygen demand
 - d. Improve LV and septal relaxation
13. Which of the following is not a treatment modality for patients with HOCM?
 - a. Dual-chamber inhibited response, adaptive rate pacemaker
 - b. Surgical myectomy or percutaneous transluminal septal myocardial ablation (PTSMa)
 - c. Medical therapy with beta blockade, nonhydrophiridine calcium channel blockers, and amiodarone
 - d. Medical therapy with angiotensin-converting enzyme inhibitors and nitroglycerin
14. Which of the following is a potentially life-threatening complication of PTSMa?
 - a. Extension of the septal infarction
 - b. Atrial arrhythmias
 - c. Heart failure
 - d. Access site hematoma

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