REVIEW ARTICLE

Cardiomyopathy: An Update and Anesthetic Considerations

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Abstract

It can affect young athletes. It may present with microscopic alteration of cardiomyocytes leading to cardiac failure. It has been found that early diagnosis may play a pivotal role in guiding treatment decisions, improving quality of life.

Keywords: Cardiomyopathy, Systolic blood pressure, The American Heart Association.

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Introduction

Hypertrophic cardiomyopathy (HCM) has become a common disease of the cardiovascular system. It can affect people of any age. It affects men and women equally. It is one of the common causes of sudden cardiac arrest in young people, it can affect young athletes. It may present with microscopic alteration of cardiomyocytes leading to cardiac failure.[1,2] The definition and classification of cardiomyopathy have been the subject of interest in recent years. The American Heart Association has classified cardiomyopathy as primary or secondary. Many authors say that this is not a perfect classification. They think primary should be classified as acquired, genetic or mixed[1] which is shown in Table 1. They believe that there is an overlap of these conditions creating confusion. Classification of cardiomyopathies is still evolving.

Cardiomyopathy has been classified as dilated, hypertrophic and restrictive. Over time each classification has become a recognized clinical condition. Arrhythmogenic cardiomyopathy has also been added in the classification, Cardiomyopathies are now defined as myocardial diseases associated with cardiac dysfunction. Most of all the cardiomyopathies maintain some relation to genetic transmission. Characteristics of all the cardiomyopathies are discussed in Table 1, described by Franz et al.[4] (Table 2).

The most common cardiac procedures for patients with dilated cardiomyopathy (DCM) are correction of atrioventricular valve problems, placement of an implantable cardioverter defibrillator for refractory ventricular arrhythmias and placement of left ventricular assist device or allograft transplantation, anesthetic goals include reduction of afterload, preload optimization and minimizing myocardial depression. Fentanyl produces excellent anesthesia and hemodynamics in patients with ejection fraction less than 30%. Ketamine is an excellent choice with a dose of around 0.5 mg/kg along with fentanyl in patients with severe myocardial depression secondary to cardiomyopathy. The use of propofol is still a concern because of myocardial depression. De Hert et al.[5] found that sevoflurane and desflurane were shown not to adversely affect the left ventricular performance in relation to stress in patients undergoing cardiac surgery. Echocardiography is useful in patients undergoing both cardiac and non-cardiac surgeries. In patients with DCM, signs and symptoms may not reflect the clinical situation. According to Stevenson et al.,[6] 80% of patients with PCWP of more than 25 mm of hg had no signs of pulmonary edema on auscultation. Hemodynamic instability can be managed with low-dose inotropes and vasodilators. Phosphodiesterase inhibitors along with beta agonists like milrinone and adrenaline provide transient hemodynamic stability. Afterload reduction improves ventricular performance under anesthesia along with control of valvular regurgitation and atrial volume. Levosimendan maintains stable hemodynamics augmenting myocardial performance without increasing oxygen demand. Hypokalemia, hypomagnesemia and excessive sympathetic stimulation should be avoided. Antiarrhythmic medications are hazardous in patients with poor ventricular function because of their negative inotropic effects. They are considered in DCM if symptomatic arrhythmias are present. Class I agents are not preferred as they are associated with increased mortality as suggested by Elliott P et al.[7] Amiodarone is the preferred agent because it is associated with lesser negative inotropic effects when compared to other drugs. In one of the recent larger multicenter trials conducted by Nul DR et al.[8] Amiodarone was associated with a significantly lower mortality rate. Recent evidence has indicated that previous cardiac arrest or sustained ventricular tachycardia do get benefitted from an implantable defibrillator. Patients who do not respond to pharmacological therapy required dual chamber pacing, cardiomyoplasty, left ventricular assist devices (LVAD), cardiac transplantation in recent years. Cardiac resynchronization therapy with dual ventricular pacing improves NYHA functional class and ejection fraction 6 months after implantation.[9]

The overall prevalence of HCM is around 0.2%. This is characterized by asymmetric left ventricular hypertrophy, manifesting as increased chamber stiffness, impaired and prolonged relaxation which is also associated with complex arrhythmias and sudden death.

Symptoms of HCM are nonspecific which includes chest pain, palpitation, dyspnea and syncope. ECG is abnormal in many of

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the patients which shows increased QRS voltage, ST-segment and T-Wave abnormalities and left ventricular hypertrophy with strain pattern. Echocardiography is used to evaluate HCM. TEE identifies the location of hypertrophy, the thickness of the ventricular septum, systolic anterior motion and LVOT obstruction. The only effective modality to prevent sudden death associated with HCM is the use of an implantable cardioverter defibrillator (Flowchart. 1).[10] Anesthetic management of patients with HCM includes prevention of any exacerbation of obstruction and the corresponding increase in the intraventricular gradient that will affect systolic blood pressure. Induction of anesthesia is hazardous because fasting in the preoperative period will reduce preload which provokes an increase in LVOT obstruction. In general beta blockers have been a main stay of treatment for years. Verapamil can be used if bets blockers are not tolerated. The combination of beta blockers and calcium channel blockers has not proven beneficial. Angina and dyspnea can be controlled with other beneficial effects including reduction of heart rate, reduced myocardial oxygen demand and longer diastolic filling time. In obstructive HCM Disopyramide is the most effective drug to reduce LVOT obstruction. Combination of beta blockers along with Disopyramide improves exercise tolerance[11] Vasoconstrictors are preferred to maintain SVR. Narcotics have been used successfully during induction of anesthesia. Use of propofol is not encouraged as it decreases systolic blood pressure.[12] For maintenance halothane is also advantageous. It is rarely used nowadays in comparison with Isoflurane. The potential for severe myocardial depression is less with newer volatile agents. is the preferred muscle relaxant as it maintains hemodynamic stability. Arrhythmias complicate in these patients during anesthesia. Complex ventricular arrhythmias are common. Implantable cardioverter defibrillators need to be temporarily suspended in patients before the use of cautery. Supraventricular tachycardia can also occur. Atrial fibrillation may rapidly progress to congestive heart failure. Prompt cardioversion is necessary.[13] Surgical correction of HCM is directed primarily to relieve symptoms of LVOT obstruction who do not respond to medications. A myotomy–myectomy through a transaortic approach is the primary method to relieve the obstruction. The reduction in left atrial size may account for a lower incidence of atrial fibrillation after myectomy. Complications of include complete heart block and septal perforation[14] Possible alternatives to surgery include dual chamber pacing and percutaneous alcohol septal ablation.[15] Septal ablation with alcohol is a non-surgical reduction based on interruption of blood supply. Alcohol is administered through an angioplasty balloon to the perforator branch of left anterior descending coronary artery. Alam M et al.[16] in their study showed effective gradient reduction and improvement in NYHA class compared with myectomy 12 months after ablation. But the toxic effect of alcohol on both coronary circulation and myocardium is still under evaluation.

Restrictive cardiomyopathy is defined by 1995 WHO guidelines as restrictive filling and reduced diastolic volume of either or both ventricles with normal or near normal systolic function and wall thickness. Systolic function is minimally affected. There are abnormalities of ventricular relaxation and compliance.

Table 1: Clinical, hemodynamic and morphological characteristics of cardiomyopathies

	Hypertrophic cardiomyopathy	*Dilated cardiomyopathy*	*Arrhythmogenic right ventricular cardiomyopathy*	*Restrictive cardiomyopathy*
Clinical				
Heart failure	Occasional (LV)	Frequent (LV or BV)	Frequent (RV)	Frequent (BV)
Arrhythmias	Atrial and ventricular arrhythmias	Atrial and ventricular arrhythmias, conduction defects	Ventricular tachycardia (RV), conduction defects	Atrial fibrillation
Sudden death	0.7–11% per year	Frequent (ND)	Frequent (ND)	1–5% per year
Hemodynamic				
Systolic function	Hyperdynamic, outflow tract obstruction (occasionally)	Reduced	Normal-reduced	Near normal
Diastolic function	Reduced	Reduced	Reduced	Severely reduced
Morphologic				
Cavity size Ventricle	Reduced (LV)	Enlarged (LV or BV)	Enlarged (RV)	Normal or reduced (BV)
Atrium	Normal-enlarged (LA)	Enlarged (LA or BA)	Enlarged (RA)	Enlarged (BA)
Wall thickness	Enlarged, asymmetric (LV)	Normal-reduced (LV or BV)	Normal-reduced (RV)	Normal (BV)

LV, left ventricle; BV, both ventricles; RV, right ventricle; ND, not determined; BA, both atria; LA, left atrium; RA, right atrium.
From Franz WM, Müller OJ, Katus HA: Cardiomyopathies: From genetics to the prospect of treatment. Lancet 358:1628, 2001.

Table 2: Classification of primary cardiomyopathies[1]

Acquired
Myocarditis
Peripartum
Tachycardia induced
Takotsubo (stress induced)
Genetic
Arrhythmogenic right ventricular dysplasia
Hypertrophic
Ion channel disorders
Left ventricular compaction
Mitochondrial myopathies
Mixed
Dilated
Restrictive

Flowchart 1: Indication for implantable cardioverter defibrillator (ICD)[30]

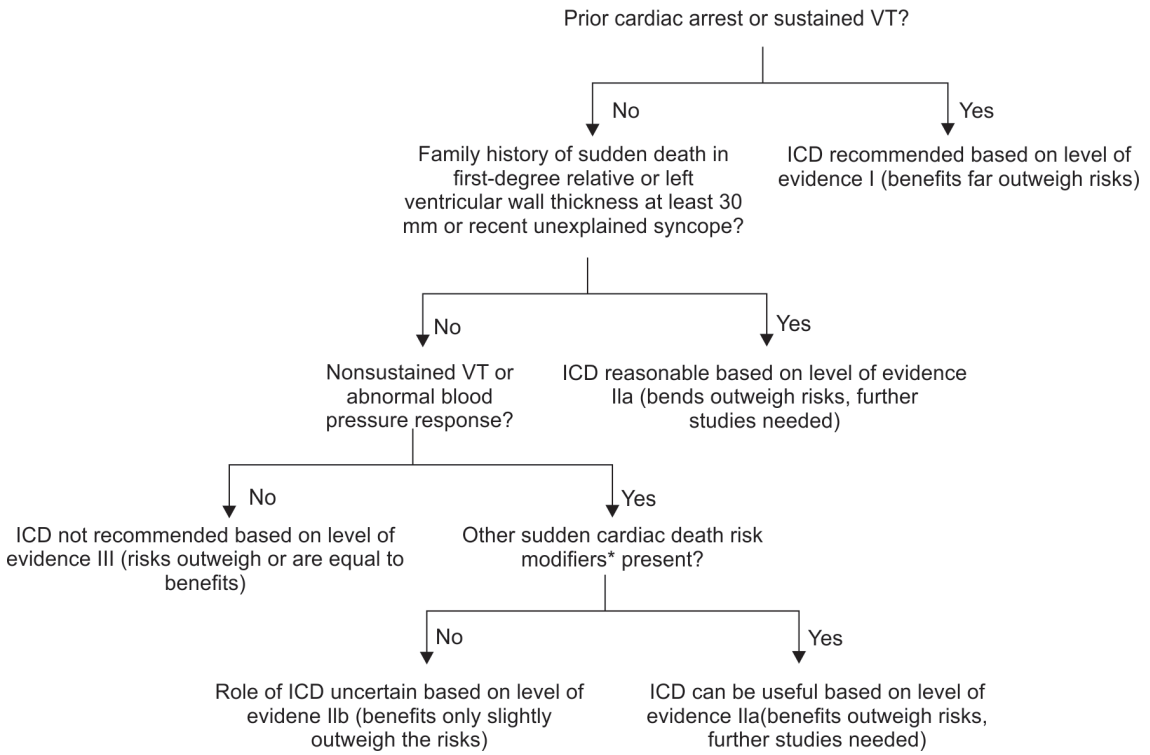
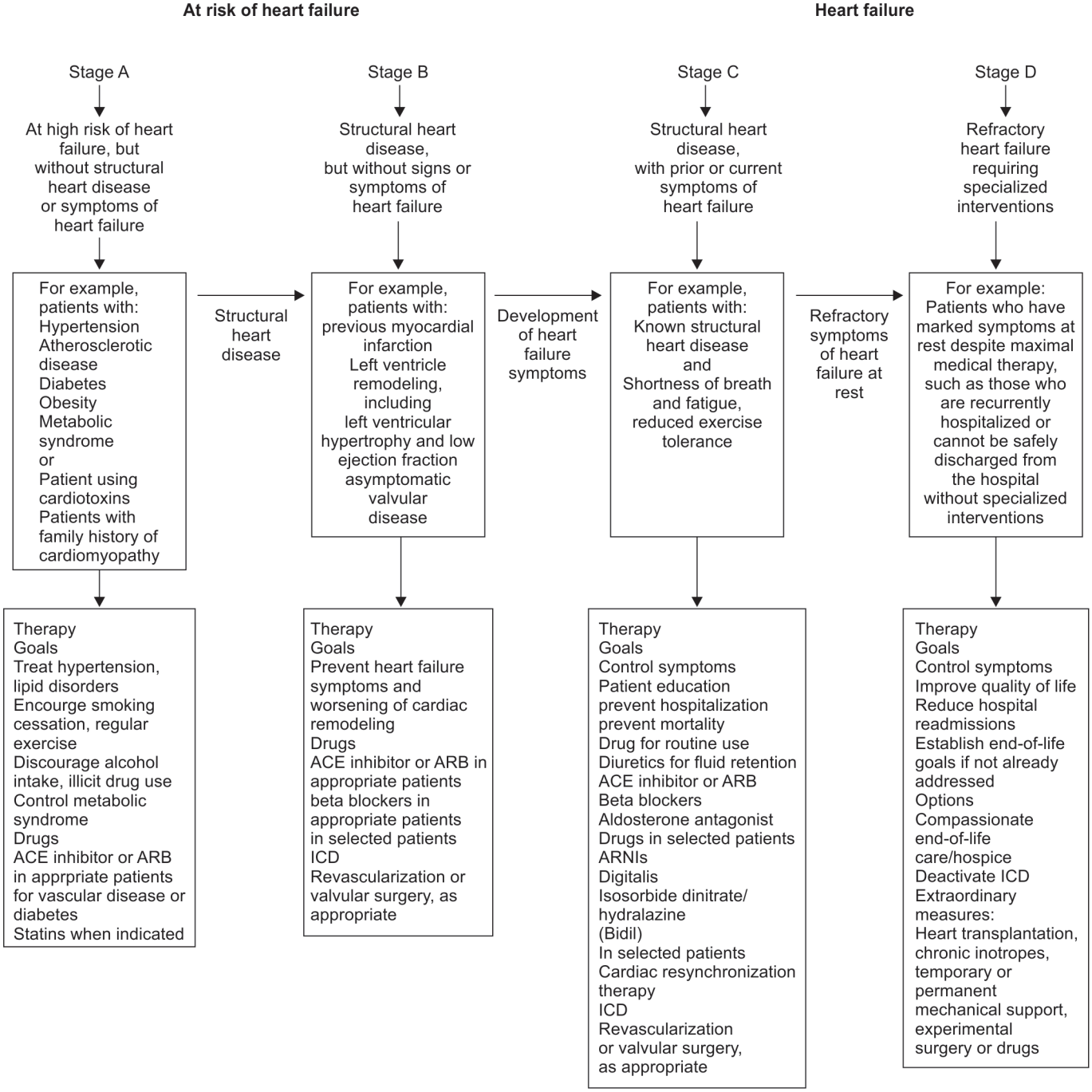


Table 3: Secondary causes of cardiomyopathy[2,3]

Infiltrative disorders	
Polyarteritis nodosa	*Infiltrative disorders*
Rheumatoid arthritis	Amyloidosis
Sarcoidosis	Gaucher disease
Scleroderma	Hunter syndrome
Systemic lupus erythematosus	Hurler syndrome
Endocrine	*Neuromuscular and storage disorders*
Acromegaly	Glycogen storage disorders
Diabetes mellitus	Muscular dystrophy (Becker, Duchenne, Emery-Dreifuss, myotonic)
Hyperparathyroidism	Neurofibromatosis
Hyperthyroidism	*Nutritional deficiencies*
Hypothyroidism	Kwashiorkor L-carnitine, niacin, selenium, thiamine, vitamin C deficiencies
Obesity	*Toxic*
Infectious	Alcohol
Chagas disease	Anabolic steroids
Hepatitis C	Chemotherapeutic agents (anthracyclines, cyclophosphamide, doxorubicin [Adriamycin])
Human immunodeficiency virus	Chloroquine (Aralen)
Mycobacteria	Heavy metals (arsenic, cobalt, lead, mercury)
Rickettsia	Iron excess (hemochromatosis)
Viral (adenovirus, coxsackie, Epstein-Barr, parvovirus)	Radiation
	Stimulants (cocaine, methylphenidate

2D echocardiography with doppler indicates the severity of RCM. Diastolic dysfunction results in poor cardiac output and systemic perfusion.[17] Preoperative diuretic therapy causes hypotension. During induction of anesthesia, drugs reduce venous return and heart rate should be avoided. Fentanyl provides stable hemodynamic along with etomidate. Sevoflurane and desflurane are excellent choices for maintenance. Invasive hemodynamic monitoring is important. Diuretics and vasodilators are deleterious despite the presence of CHF as greater filling pressures are needed to maintain cardiac output. Careful titration of antiarrhythmics are required

Flowchart. 2: American College of Cardiology American Heart Association heart failure guidelines. (ACE, angiotensin- converting enzyme; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; ICD, implantable cardioverter-defibrillator)[30]



to reduce myocardial contractility and conduction.[21] Cardiac transplantation is not always an option in RCM as the processes responsible for RCM will also affect the newly transplanted heart.[18]

Arrhythmogenic right ventricular cardiomyopathy: When considering anesthetic implications for arrhythmogenic right ventricular cardiomyopathy arrhythmias of both supraventricular and ventricular may occur at any time. Hypovolemia, hypercarbia, acidosis and a lighter plane of anesthesia should be minimized. Anesthesia has been conducted successfully with propofol, midazolam and fentanyl.[19] Currently there are no guidelines for arrhythmia prophylaxis. Amiodarone is the first line of medication used to treat rhythm disturbances. Placement of implantable cardioverter defibrillator has shown beneficial effects with an excellent prognosis in patients with ARVC as identified by.[20,21]

Acquired Causes

Peripartum cardiomyopathy: Peripartum cardiomyopathy is defined as left ventricular systolic dysfunction at the end of pregnancy or in the months following delivery[22] It occurs most commonly in the first month after delivery. It may also develop in the second trimester and four months after delivery.[23] The

estimated incidence is 1:1,000 to 4,000 live births.[22] The factors associated with this disease are, increasing age, black race, preeclampsia, hypertension, cardiomyopathy in a prior pregnancy, and multiple gestations.[22] It may present as heart failure and can be confused with complications such as preeclampsia, and leading to delayed diagnosis of cardiomyopathy.[23,24] More commonly sinus tachycardia is seen on Electrocardiography findings and are nonspecific. Common echocardiography findings include left ventricular dilation, left ventricular systolic dysfunction, and pulmonary hypertension.[23] These patients are Treated with standard heart failure therapy, keeping in mind that patients are still pregnant. Hence, it is better to avoid ACE inhibitors and ARBs. in pregnant patients. Due considerations to be given while using diuretic therapy.[23] so as to avoid hypotension and reduced uterine perfusion. Most women with peripartum cardiomyopathy recover left ventricular function. Long-term mortality rates are not certain but range from 11 to 16% in the literature.[23]

Takotsubo cardiomyopathy: It is also known as stress-induced cardiomyopathy or broken-heart syndrome. Now it is defined as a sudden onset of left ventricular dysfunction in response to severe emotional or physiologic stress.[1] Woman after menopausal are most commonly affected. The prevalence has been estimated at 0.02% of all hospitalized patients. It is roughly estimated to be 1–2% of admissions for the acute coronary syndrome.[25,26] This syndrome often presents with angina, pectoris and we may see the features of ischemia in electrocardiography. On echocardiography, we may see apical ballooning of the left ventricle. Elevated cardiac enzymes may be seen as Laboratory findings.[27] It may present as an acute coronary syndrome. Hence Takotsubo cardiomyopathy should be treated in the same way as we treat any ischemic event. Shock or heart failure are often seen as severe complications and should be managed appropriately. Others may be treated with diuretics, ACE inhibitors or ARBs, and beta blockers. Patients with loss of wall motion in the left ventricular apex should be prescribed anticoagulants.[27] It is also known that symptoms and signs reverse within one month, and treatments should be stopped.[27]

Secondary cardiomyopathies: Disease of cardiac muscle resulting from an extracardiovascular cause is known as secondary cardiomyopathy.[2,3] Some causes of secondary cardiomyopathies are associated with specific disease patterns for, e.g., abuse of alcohol leading to dilated cardiomyopathy and amyloidosis may lead to restrictive cardiomyopathy. Pathophysiology caused by systemic diseases are variable. Secondary causes are mentioned in Table 3. Systematic evaluation to know the cause and the management are aimed at removing causative factors, and symptomatic treatment of heart failure (Flowchart 2).

As we are aware that genetics has a definite influence on cardiomyopathy, it is better that patients with any cardiomyopathy be referred for genetic counseling, if the treating doctor is not familiar with current treatment guidelines.[28] It has been found that early diagnosis may play a pivotal role in guiding treatment decisions, improving quality of life, or may prolong life expectancy.[28-30] It is recommended that relatives of patients with cardiomyopathy should be considered for clinical and genetic screening.[28]

Recommendations for clinical practice guidelines: It is felt that heart failure with reduced ejection practice and HCM should be managed as per the recent AHA (American Heart Association) guidelines as per reference 30. Other guidelines are shown in Table 4.

Latest update: Diseases with known cardiovascular etiology have been excluded from CARDIOMYOPATHY.[31] Ex. Hypertensive heart disease, congenital heart disease, valvular heart disease and coronary heart disease. Recently Left ventricular non-compaction cardiomyopathy has been added to the list of congenital causes. In this condition, trabeculation of the myocardium is seen and they, in turn, develop into intertrabecular recesses in the left ventricle.

Table 4: Recommendations for clinical practice guidelines

Clinical recommendation	*Evidence rating*	
Heartfailure withreduced ejection fraction should be managed according to the most recent American College of Cardiology/American Heart Association guidelines	C	
Hypertrophic cardiomyopathy should be managed according to the most recent American College of Cardiology Foundation/American Heart Association guidelines	C	
An implanted cardioverter-defibrillator should be placed in patients who are at risk of sudden cardiac death	C	
Heart transplantation should be considered if cardiomyopathy is refractory to medical therapy	C	
Patients with cardiomyopathy should be referred for genetic counseling	C	4

A, consistent, good-quality patient-oriented evidence; B, inconsistent or limited-quality patient-oriented evidence; C, consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to http://www.aafp.org/afpsort.

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