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Research Article

PREVENTION OF DILATED CARDIOMYOPATHY DISEASE MEASURES TO TREAT IT

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Roxibjonov Adhamjon Raxmatjon O'g'li
Department Of Internal Medicine, Andijan State Medical Institute, Uzbekistan

ABSTRACT

Dilated cardiomyopathy is myocardial dysfunction causing heart failure in which ventricular dilation and systolic dysfunction predominate. Symptoms include dyspnea, fatigue, and peripheral edema. Diagnosis is clinical and by elevated natriuretic peptides, chest x-ray, echocardiography, and MRI. Treatment is directed at the cause. If heart failure is progressive and severe, cardiac resynchronization therapy, implantable cardioverter-defibrillator, repair of moderate to severe valvular regurgitation, left ventricular assist device, or heart transplantation may be needed.

KEYWORDS

Cardiomyopaty, heart, diagnosis, treatment.

INTRODUCTION

A cardiomyopathy is a primary disorder of the heart muscle. It is distinct from structural cardiac disorders such as coronary artery disease, valvular disorders, and congenital heart disorders. Cardiomyopathies are divided into 3 main types based on the pathologic features (see figure Forms of cardiomyopathy):

- Dilated
- Hypertrophic
- Restrictive

The term ischemic cardiomyopathy refers to the dilated, poorly contracting myocardium that can occur in patients with severe coronary artery disease (with or without areas of infarction). It is not classically considered to be in the above-listed categories

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because it does not describe a primary myocardial disorder.

Manifestations of cardiomyopathies are usually those of heart failure and vary depending on whether there is systolic dysfunction, diastolic dysfunction, or both. Some cardiomyopathies may also cause chest pain, syncope, arrhythmias, or sudden death.

Evaluation typically includes family history, blood tests, ECG, chest x-ray, echocardiography, and cardiac MRI. Some patients require endomyocardial biopsy. Other tests are done as needed to determine the cause. Treatment depends on the specific type and cause of cardiomyopathy

As a primary myocardial disorder, the myocardial dysfunction of dilated cardiomyopathy occurs in the absence of other disorders that can cause dilated myocardium, such as severe occlusive coronary artery disease or conditions that involve pressure or volume overload of the ventricle (eg, hypertension, valvular heart disease). In some patients, dilated cardiomyopathy is believed to start with acute myocarditis (probably viral in most cases), followed by a variable latent phase, a phase with diffuse necrosis of myocardial myocytes (due to an autoimmune reaction to virus-altered myocytes), and chronic fibrosis. Regardless of the cause, the myocardium dilates, thins, and hypertrophies in compensation (see figure Forms of cardiomyopathy), often leading to functional mitral regurgitation and/or tricuspid regurgitation and atrial dilation.

The disorder affects both ventricles in most patients, only the left ventricle (LV) in a few , and only the right ventricle (RV) rarely.

Mural thrombi may form due to stasis of blood once chamber dilation and dysfunction are significant. Cardiac tachyarrhythmias often complicate the acute myocarditis and late chronic dilated phases as may atrioventricular block. Atrial fibrillation commonly occurs as the left atrium dilates.

Dilated cardiomyopathy has many known and probably many unidentified causes (see table Causes of Dilated Cardiomyopathy). More than 20 viruses can cause dilated cardiomyopathy; in temperate zones, coxsackievirus B is most common. In Central and South America, Chagas disease due to Trypanosoma cruzi is the most common infectious cause.

Other causes include prolonged (chronic) tachycardia, HIV infection, toxoplasmosis, thyrotoxicosis, and beriberi. Many toxic substances, particularly alcohol, various organic solvents, iron or heavy metal ions, and certain chemotherapeutic drugs (eg, doxorubicin, trastuzumab), damage the heart. Frequent ventricular ectopy (> 10,000 ventricular premature beats/day) has been associated with left ventricular systolic dysfunction.

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Sudden emotional stress and other hyperadrenergic states can trigger acute dilated cardiomyopathy that is typically reversible (as is that caused by prolonged tachycardia). An example is acute apical ballooning cardiomyopathy (also called takotsubo cardiomyopathy, stress cardiomyopathy, or broken heart syndrome). In this disorder, usually the apex and occasionally other segments of the left ventricle are affected, causing regional wall dysfunction and sometimes focal dilation (ballooning).

Genetic factors play a role in 20 to 35% of cases; > 60 genes and loci have been implicated.

Onset of dilated cardiomyopathy is usually gradual except in acute myocarditis, acute apical ballooning cardiomyopathy, and tachyarrhythmia-induced cardiomyopathy. About 25% of all patients with dilated cardiomyopathy have atypical chest pain. Other symptoms depend on which ventricle is affected.

Left ventricular dysfunction causes exertional dyspnea and fatigue due to elevated left ventricular diastolic pressure and low cardiac output.

Right ventricular failure causes peripheral edema and neck vein distention. Infrequently the right ventricle is predominantly affected in younger patients, and atrial arrhythmias and sudden death due to malignant ventricular tachyarrhythmias are typical.

- Chest x-ray
- ECG
- Echocardiography
- Cardiac MRI
- Endomyocardial biopsy (select cases)
- Testing for cause as indicated

Diagnosis of dilated cardiomyopathy is by history, physical examination, and exclusion of other common causes of ventricular failure (eg, systemic hypertension, primary valvular disorders, myocardial infarction—see table Diagnosis and Treatment of Cardiomyopathies). Particularly in cases of dilated cardiomyopathy without a clear cause, a careful family history should be taken to identify family members with possible early-onset heart disease, heart failure, or sudden death. In many centers, first-degree family members are screened for cardiac dysfunction (such as with echocardiography). Because other common causes of ventricular failure must be excluded, chest x-ray, ECG, echocardiography, and cardiac MRI are required. Endomyocardial biopsy is done in selected cases.

Serum cardiac markers are measured if acute symptoms or chest pain is present. Although typically indicative of coronary ischemia, troponin elevation often occurs in heart failure, especially if renal function

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is decreased. Serum natriuretic peptide levels are typically elevated when heart failure is present.

Specific causes suspected clinically are diagnosed (see elsewhere in THE MANUAL). If no specific cause is clinically apparent, serum ferritin and iron-binding capacity and thyroid-stimulating hormone levels are measured.

Serologic tests for Toxoplasma, T. cruzi, coxsackievirus, HIV, and echovirus may be done in appropriate cases.

Chest x-ray shows cardiomegaly, usually of all chambers. Pleural effusion, particularly on the right, often accompanies increased pulmonary venous pressure and interstitial edema.

The ECG may show sinus tachycardia and nonspecific ST-segment depression with low voltage or inverted T waves. Sometimes pathologic Q waves are present in the precordial leads, simulating previous myocardial infarction. Left bundle branch block and atrial fibrillation are common.

Echocardiography shows dilated, hypokinetic cardiac chambers and rules out primary valvular disorders. Segmental wall motion abnormalities can also occur in dilated cardiomyopathy because the process may be patchy. Echocardiography may also show a mural thrombus.

Cardiac MRI is increasingly done and is useful in providing detailed imaging of myocardial structure and function. MRI with gadolinium contrast may show abnormal myocardial tissue texture or scarring pattern (ie, late gadolinium enhancement, or LGE). The pattern of LGE can be diagnostic in active myocarditis, sarcoidosis, muscular dystrophy, or Chagas disease).

Positron-emission tomography (PET) has been shown to be sensitive for diagnosis of cardiac sarcoidosis.

Coronary angiography may be required to exclude coronary artery disease as the cause of LV dysfunction when the diagnosis is in doubt after noninvasive tests. Patients with chest pain or several cardiovascular risk factors and older patients are more likely to have coronary artery disease. Either ventricle can be biopsied during catheterization in select cases where the results will change management.

Endomyocardial biopsy is indicated if giant cell myocarditis, eosinophilic myocarditis, or sarcoidosis is suspected, as the results will affect management.

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