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CASE STUDY

Tachycardia-Induced Heart Failure

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Abstract

Heart failure associated with tachyarrhythmias can very often be reversed by dealing with the underlying tachyarrhythmia. Typically characterized by left ventricular dilation and subsequent systolic dysfunction, this disorder can be caused by both atrial and ventricular arrhythmias, most commonly chronic atrial fibrillation. Whereas for most cardiomyopathies there is little that can be done to reverse the progression of the disease, in tachycardia-induced heart failure the patient's often debilitating symptoms can be ameliorated. This is particularly important in the primary care setting because tachyarrhythmias, particularly atrial fibrillation, are commonly encountered. The alert physician will be able to diagnose and treat tachyarrhythmias, which can result in improvement of systolic function within weeks and often normalization within several months.

Introduction

Congestive heart failure (HF) affects close to five million patients in the United States, with coronary artery disease being the leading etiology, accounting for 70% of HF cases.[1,2] This often debilitating disease is typically progressive unless a reversible etiology can be identified. Among the reversible causes, tachycardia-induced heart failure (TIHF) provides a unique opportunity in the primary care setting for intervention to halt and possibly reverse HF in affected patients. The incidence of TIHF is largely unknown; however, studies involving patients with atrial fibrillation (AF) show that up to 50% of patients with AF and left ventricular dysfunction have some degree of TIHF.[3] This disorder can occur at any age, from the fetal period to old age.[4] From innovative techniques such as intrauterine cardioversion to simply controlling a patient's heart rate with traditional medications, reversal of this form of HF can provide a gratifying experience for both the patient and practitioner in an otherwise progressive condition. The following case report illustrates the importance of recognizing TIHF and reviews the pathophysiologic, diagnostic, and treatment considerations of this disorder.

Case Example

A man, age 53 years, with a medical history of morbid obesity presented to the Emergency Department complaining of shortness of breath. This symptom was new and had started approximately one week before presentation. The shortness of breath was worsened by walking and decreased with rest. He noted that he had had two-pillow orthopnea for the past week.

On examination, the patient had an irregular pulse of 120 beats per minute with no murmur. Lung examination findings were normal, and there was no peripheral edema. Troponin I levels were elevated but indeterminate, both initially and when serially repeated. An electrocardiogram was obtained and revealed AF with a ventricular rate of 150 beats per minute. The patient's chest radiograph showed slight cardiomegaly. His heart rate was controlled with carvedilol (Coreg).

An echocardiogram showed four-chamber enlargement, global hypokinesis, and an estimated ejection fraction of 15%. Cardiac catheterization showed a 90% stenosis in the first obtuse marginal coronary artery, which was dilated with stent deployment. Severe hypokinesis was again observed during catheterization, with an ejection fraction of 20%. A cardiology consultant suggested that the patient's cardiomyopathy was out of proportion to his degree of coronary artery disease and that his tachyarrhythmia was a likely major contributor.

After six months of controlled heart rate anticoagulation, an echocardiogram showed an improvement in ejection fraction from 20% to 60%. The patient subsequently had successful radiofrequency ablation treatment for his AF and his improvement continued until this report.

Discussion

Pathophysiology

Several tachyarrhythmias have been associated with the devel-


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opment of HF, including AF, atrial flutter, automatic atrial tachycardia, atrioventricular nodal reentry tachycardia, automatic atrioventricular junctional tachycardia, and ventricular tachycardias.[3,5] Although ventricular tachyarrhythmias cause a more severe depression in left ventricular function, more common are the supraventricular variety, specifically AF. The hemodynamic changes that occur include ventricular systolic dysfunction, decreased cardiac output, increased ventricular filling pressures, and increased vascular resistance. Perhaps the most convincing evidence relating tachyarrhythmias to HF comes from animal studies in which HF was induced secondary to rapid pacing.[6] After one day, a decrease in cardiac output was observed and the decrease continued to decline for up to five weeks. Prompt cessation of pacing then resulted in improvement of left ventricular function as soon as 24 hours after termination and a subsequent return to normal within weeks.

The pathophysiologic changes that lead to the development of HF in patients who have a tachyarrhythmia are not clearly understood. Furthermore, it is not clear whether the structural cardiac changes observed in cardiomyopathy are the result of the tachyarrhythmia or if the tachycardia is due to the changes seen in cardiomyopathy. Nonetheless, a number of mechanisms have been proposed that seek to explain why tachycardia leads to left ventricular dysfunction: myocardial energy depletion, abnormal calcium handling, myocardial ischemia, and extracellular matrix remodeling.[3,6–9]

The means by which myocardial energy depletion may play a role in TIHF was demonstrated in pacing studies with animals. In these animals that developed tachycardia-induced HF, it was demonstrated that myocardial energy stores were exhausted, leading to decreased levels of high-energy phosphates, including adenosine triphosphate and creatine. Mitochondrial structural and functional defects were also observed.[6,7,9]

Additionally, it has been postulated that abnormal calcium handling may be an underlying mechanism by which tachycardia induces cardiomyopathy.[6] Calcium channel activity as well as calcium transport in the sarcoplasmic reticulum have been found to be considerably abnormal in myocytes after pacing. These changes occur soon after tachycardia is induced and may remain for up to four weeks after cessation of pacing. Exactly how calcium abnormalities lead to left ventricular dysfunction, however, is not clear.

It has also been proposed that chronic rapid heart rates may result in ischemia, which can lead to some form of reversible ventricular dysfunction. This claim is supported by observations that there are abnormal subendocardial and subepicardial blood flow ratios in addition to impaired coronary flow reserve. This damage likely leads not to cell death but rather to myocardial shock or stunning, which is reversible.[6,7]

The final mechanism often related to the development of TIHF is that of extracellular matrix remodeling.[6,7] In paced animals, cellular changes are noted, including loss of myocytes, contractile dysfunction, myofibril misalignment, and abnormalities in the attachment of myocytes to the basement membrane. The changes can affect overall contractility, leading to the observed cardiomyopathy.

Diagnosis

Perhaps the most important factor in regard to the diagnosis of TIHF is a high index of suspicion. Because evidence of gradually worsening ventricular function in the setting of chronic tachycardia is not often apparent clinically, awareness of this reversible condition is vital. Evaluation may be done noninvasively using imaging modalities such as echocardiography or multiple gated acquisition scan, which demonstrate systolic dysfunction and left and right ventricular dilation.

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Prognosis

Although actual recovery of ventricular function can vary, it is clear that control of tachycardia can produce some, if not complete, reversal of cardiomyopathy in most patients.[10] Reports on the recovery time of ventricular function differ, but the most improvement can be seen within the first several weeks, with continued slow improvement for up to six months.[7,11] This will vary depending on the duration of the tachycardia and if other forms of heart disease are present.

Management

It is clear that controlling tachyarrhythmia can reverse TIHF, at least partially. However, case management will vary according to the particular tachyarrhythmia and so is outside the scope of this case report. Of interest, however, is AF as the underlying cause of TIHF, because it both has been well studied and is the most common cause. It has been shown that patients with HF whose AF is controlled experience an overall lower mortality, but whether this is because of rate control or rhythm control is not as clear. Several studies attempting to discern which is superior, rate control or rhythm control, have shown that either one is acceptable.[3,8,12,13]

If initial attempts at drug therapy fail, atrioventricular node ablation with pacemaker implantation is an effective alternative.[14]

Summary

Perhaps the most relevant aspect of TIHF to primary care practice is that control of tachycardia can improve or completely resolve a patient's cardiomyopathy. For a disease in which most only have hopes of delaying progression and limiting symptoms, the idea that a cure, or even a modest improvement in quality of life is available, makes early detection a critical task for primary care providers because they are in a setting where early detection is more likely. With a high index of suspicion, diagnosis followed by heart rate control can provide marked improvement in systolic function in as little as one month. ❖

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An Occasional Heart Attack

As for me, except for an occasional heart attack,
I feel as young as I ever did.

— Robert Benchley, 1889-1945, American humorist and actor