Richard, 52, presents to the ED complaining of exertional palpitations and difficulty breathing. Richard’s examination reveals:

- A long history of atrial fibrillation which was treated with warfarin
- Hypertension
- An initial echocardiogram showing normal left ventricular (LV) size and function, with no valvular abnormalities
- Failed rhythm control with sotalol and propafenone, yet Richard was unwilling to take amiodarone
- His tolerance to various atrioventricular (AV) node blocking agents (such as, metoprolol, digoxin and subsequently diltiazem), was poor due to perceived side-effects and persistent symptoms with inadequate heart rate control
- A 24-hour Holter monitor reveals persistent atrial fibrillation and a rapid heart rate (averaging 130 beats per minute [bpm])

Richard’s ECG is shown in Figure 1.

For more on Richard, see page 20.

Figure 1. The ECG shows atrial fibrillation with a ventricular rate of 160 bpm. There are non-specific T wave changes.

About the authors...

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CardioCase presentation

Richard's palpitations and dyspnea continued...
As an alternative management to the failed drug therapy, AV node ablation and the implantation of VVIR ventricular pacemaker was planned. Richard was admitted to the hospital for the implantation of the pacemaker.

In the immediate post-operative period, he became unwell and developed acute dyspnea, hypoxia and hypotension. There was no evidence of pneumothorax, pericardial effusion, or acute ECG changes. An echocardiography study showed a dilated LV with a markedly reduced ejection fraction (EF) at 10% to 15%. Richard was admitted to the coronary care unit for resuscitation, inotropic support and he required mechanical ventilation. Cardiac catheterization revealed normal coronary arteries.

CardioCase diagnosis
Various causes of dilated cardiomyopathy should be considered. Tachycardia-induced cardiomyopathy was suspected since his systolic cardiac function was normal in a recent evaluation. Furthermore, there was an absence of myocardial ischemia. He also had a poorly controlled heart rate.

Richard's follow-up
Richard required mechanical ventilation and circulatory support in the form of an intra-aortic balloon pump. An emergency AV node ablation was performed. Within two days, he was extubated and was taken off inotropes. Within one week, he was well enough to be discharged. Six weeks post discharge, his EF had improved to over 50%.

Figure 2 shows Richard’s ECG post AV node ablation.

CardioCase discussion

What is tachycardia-induced cardiomyopathy?
Tachycardia-induced cardiomyopathy is a form of dilated cardiomyopathy and heart failure, which is caused by supraventricular and ventricular tachyarrhythmias. The clinical manifestations of heart failure are associated with ventricular systolic dysfunction and dilatation associated with persistent tachyarrhythmias. The condition is generally considered reversible with the normalization of the heart rate.

Persistent tachycardia (i.e., lasting weeks to months), regardless of etiology, predisposes a person to ventricular dilatation and left ventricular (LV) dysfunction. This disorder has been associated with several arrhythmias including:
• atrial fibrillation,
• atrial flutter,
• incessant (persistent) supraventricular tachycardia and
• ventricular tachycardia.

The exact mechanism of tachycardia-induced cardiomyopathy is unknown, though several theories exist. Some of the theories include:
• abnormal calcium handling,
• downregulation of myocardial beta-1 receptors,
• depletion of myocardial energy stores and
• chronic ischemia similar to the stunning phenomenon.

How common is it?

Tachycardia-induced cardiomyopathy can occur at any age. The incidence of tachycardia-induced cardiomyopathy is unknown. In selected studies of patients with atrial fibrillation, approximately 25% to 50% of those with LV dysfunction had some degree of tachycardia-induced cardiomyopathy.

Diagnosis

The diagnosis of tachycardia-induced cardiomyopathy requires a high index of suspicion. The clinician should consider the diagnosis in patients with unexplained systolic dysfunction and any form of tachyarrhythmia. Non-invasive evaluation of cardiac function with echocardiography usually shows LV and right ventricular dilation along with systolic dysfunction. Cardiac catheterization and coronary angiography are usually required for patient evaluation.

Tachycardia-induced cardiomyopathy may occur in association with other forms of heart disease and persistent tachycardia may aggravate already reduced systolic function.

Management

Heart rate normalization, either by rate or rhythm control, is the cornerstone of therapy. This may result in the improvement, or the normalization of systolic function. Treating the underlying cause of the tachycardia, either with ablation or medications, results in significant improvement in cardiac function.

Adequate heart rate control can prevent development of tachycardia-induced cardiomyopathy. During atrial fibrillation, the heart rate is considered optimally controlled when the ventricular response is:
• between 60 beats per minute (bpm) and 80 bpm at rest and
• between 90 bpm to 115 bpm during moderate exercise (according to the American Heart Association criteria).

Concluding thoughts

Tachycardia-induced cardiomyopathy is a well recognized cause of LV systolic dysfunction and has been associated with a variety of tachyarrhythmias. It is extremely important to recognize this condition, as it is a cause of heart failure that is potentially reversible.

Selected Readings: