

ARRHYTHMIA-INDUCED CARDIOMYOPATHY

Definition

The term mainly denotes arrhythmia-induced cardiomyopathy (AiC) that can lead to heart failure and death.¹⁻⁴ Frequent VPCs and mechanical dyssynchrony are also reversible causes of impaired left ventricular function and cardiomyopathy.

Epidemiology

AiC has been reported at any age group, from the fetus to the elderly. AiCM is present in most patients with arrhythmias and LV systolic dysfunction. Approximately 8% of patients with an arrhythmia develop AiCM.⁵

Aetiology

The syndrome has been initially described with the so-called persistent reciprocating junctional tachycardia, a term that denoted, mainly, atrioventricular reentry due to a septal decremental pathway, but also atypical (fast-slow) atrioventricular nodal reentry. We know now that any chronic cardiac arrhythmia may cause tachycardia-induced cardiomyopathy: incessant atrioventricular reentrant tachycardia due to septal accessory pathways, rapid atrial fibrillation, idiopathic ventricular tachycardia, inappropriate sinus nodal tachycardia, atrial flutter, and persistent ectopic beats are the most described causes.¹⁻⁴ In young patients <18 years of age, ectopic atrial tachycardia is the commonest cause.⁶ **Abnormal conduction-induced cardiomyopathy** refers to LV systolic dysfunction caused by high-burden, apical right ventricular pacing, LBBB, or pre-excitation.⁷⁻¹⁰

Pathophysiology

Rapid pacing in animal models have documented cytoskeletal changes and remodeling of the extracellular matrix attributed to abnormal calcium cycling, increased catecholamines and decreased beta 1 adrenergic receptor density, oxidative stress, depletion of myocardial energy stores, and myocardial ischemia due to increased heart rate.^{11, 12} Endomyocardial biopsy specimens from patients with tachycardiomyopathy exhibit features distinct from those of other types of cardiomyopathy, including abnormal cardiomyocyte and mitochondrial morphology and macrophage-dominated cardiac inflammation.¹³ However, it is not known why the majority of patients with frequent PVCs have a benign course, whereas up to 30% of them may develop cardiomyopathy.¹⁴ Cardiomyopathy induced by PVCs is mechanistically different to a tachycardia-mediated cardiomyopathy produced by regular

ventricular tachy-pacing, with more severe LV systolic dysfunction associated with higher levels of LV dyssynchrony, wider QRS complexes, lower peak positive ectopic dP/dt values, and lower stroke volumes.¹⁵

Presentation

Patients present with possibly unexplained left ventricular dilatation and systolic dysfunction and a history of paroxysmal tachycardias or permanent AF. Heart failure develops slowly but sudden death is possible.¹⁶

Diagnosis

No strict criteria exist. Diagnosis is established by excluding other causes of cardiomyopathy and demonstrating recovery of LV function after eradication of the arrhythmia or control of the ventricular rate. The ventricular rate that causes cardiomyopathy is not known, although rates >100 bpm for prolonged periods are considered to be responsible.¹² The range of PVCs to be accounted for the cardiomyopathy ranges from 13,000 to 20,000 beats/day (or alternatively a 24-hour burden of >24% on Holter),^{3, 17, 18} although ectopy-induced cardiomyopathy has been reported in a patient with only 5502 beats/day (6%).¹⁹ It is now accepted that the diagnosis should be considered in patients with a mean heart rate of >100 beats/min, atrial fibrillation, or a PVC burden of >10%.²⁰ Worsening of the LV function has been reported with >1000 PVCs/day,²¹ but the clear-cut point that marks the critical frequency for cardiomyopathy is not known. It seems, however, that most cases of PVC-induced cardiomyopathy occur at burdens >10%.²² PVC QRS duration (≥ 140 -150 ms) is also predictive of impaired LV function.¹ Usually, LVEDD is <65 and LVESD < 50 mm in patients with LVEF <30% due to TIC.²³ Larger values suggest dilated cardiomyopathy although overlapping exists.

Therapy

In 59% of patients presenting with both arrhythmia and LV systolic dysfunction, the LVEF will improve after antiarrhythmic treatment.⁵ Usually, LV function improves approximately 3 months following restoration of normal heart rate. In inappropriate sinus nodal tachycardia beta blockers are indicated. Sinus node modification with catheter ablation carries a 10% risk of sinus nodal damage and need of a PPM. Catheter ablation is indicated when tachycardiomyopathy is due to a clinical arrhythmia, such as AF, PJRT, idiopathic VT, and monomorphic VPBs.²² In AF both pulmonary vein isolation,²⁴ and AV nodal modification²⁵ improve LV function, but PV isolation has been found superior to AV nodal ablation and biventricular pacing in this respect.²⁶ When the tachycardia itself cannot be ablated, AV

nodal modification with biventricular pacing is appropriate. Specific His bundle pacing may offer comparable,²⁷ or even better results with CRT pacing.²⁸ Long-term medical therapy with beta blockers and ACE inhibitors or ARBs is indicated before and after successful ablation attempts for LV remodeling purposes. However, withdrawal of heart failure therapy following AF rhythm control and recovered LVEF may be possible.²⁹ Treatment of pacing induced cardiomyopathy consists of upgrade to cardiac resynchronization therapy or conduction system pacing.⁹ Recommendations for therapy are provided in Tables 1 and 2, and Figure 1.

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