Tachycardia-induced Cardiomyopathy (Tachycardiomyopathy)

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Abstract: The term tachycardia-induced cardio-myopathy or tachycardiomyopathy refers to impairment in left ventricular function secondary to chronic tachycardia, which is partially or completely reversible once the tachyarrhythmia is controlled. Tachycardia-induced cardiomyopathy has been shown to occur both in experimental models and in patients with incessant tachyarrhythmia. Data from several studies and from case reports have shown that rate control by means of cardioversion, negative chronotropic agents, and surgical or catheter-based atrio-ventricular node ablation, resulted in significant improvement of systolic function. The diagnosis of tachycardia-induced cardiomyopathy is usually made following observation of marked improvement in systolic function after normalization of heart rate. Clinicians should be aware that patients with unexplained systolic dysfunction may have tachycardia-induced cardiomyo-pathy, and that controlling the arrhythmia may result in improvement of systolic function.

Introduction

Tachycardia-induced cardiomyopathy is caused by persistent suprav-entricular or ventricular tachy-arrhythmia. It is characterized by ventricular systolic dysfunction and dilatation and clinical manifestation of heart failure that are reversible with normalization of heart rate. The concept that chronic tachyarrhythmia may lead to reversible ventricular dysfunction date back to the early 1900s [1,2]. Phillips and Levine were first to describe the relationship between tachyarrhythmia and reversible heart failure in 1949 [3]. Tachycardia-induced cardiomyopathy has been shown to occur both in experimental models and in patients with tachyarrhythmia. Tachycardia-induced cardiomyopathy may follow any type of chronic or frequently recurring paroxysmal tachyarrhythm-ias. This review describes the pathophysiology and clinical manifestations and management of tachycardia-induced cardiomyopathy.

Pathophysiology

Whipple et al. first described experim-ental tachycardia-induced cardiomyo-pathy in 1962 [4]. They provided the first experimental model for the condition; demonstrating that rapid and protracted atrial pacing led to low output heart failure. Sustained rapid atrial or ventricular pacing in experimental models produces a markedly dilated cardiomyopathy involving all cardiac chambers and severe biventricular systolic and diastolic dysfunction [5]. This profound cardiac dilatation is typically accompanied by right and left ventricular wall thinning, markedly elevated ventricular filling pressures and decreased contractile state with severe impairment of systolic function. [5-8]. Cardiac output is severely reduced, and systemic vascular resistance is typically elevated. Moderate mitral valve regurgitation may develop late in the evolution of heart failure. Mitral valve regurgitation

is caused by dilatation of the left ventricle and stretching of the mitral valve annulus.

Neurohormonal activation due to low cardiac output, results in marked elevations of plasma catecholamines, atrial natriuretic peptide, rennin and aldosterone levels; further worsening the left ventricular function. The precise mechanisms responsible for the contractile dysfunction and structural changes of pacing-induced cardiomyopathy are not fully understood.

Myocardial energy depletion, impaired energy utilization and myocardial ischemia have been proposed as possible mechanisms for myocardial dysfunction [9,10].

At the cellular level, the myocyte is the elemental component of the heart and is responsible for force generation. Translation of this force into mechanical pump performance is dependent on the relationship between the myocyte and extracellular matrix. The extracellular matrix ensures proper myocyte alignment during diastole, coordinates myocyte contraction during systole, and maintains capillary patency throughout the cardiac cycle. It has been found that chronic supraventricular tachycardia caused significant increase in myocyte length, and significant disruption of the sarcolemmal-basement membrane interface [7]. This could reduce mechanical pump performance and impair ventricular function.

Abnormal calcium handling may also be responsible for tachycardia-induced cardiomyopathy. Extensive abnorm-alities in calcium channel activity and sarcoplasmic reticulum calcium transport may appear as early as 24 hours after the initiation of rapid atrial pacing, and may persist for up to 4 weeks after discontinuation of pacing [11]. The severity of calcium cycling abnormalities correlates with the degree of ventricular dysfunction. In this manner, calcium availability to myocytes may be

decreased, with subsequent reduction in contractility.

Clinical features and diagnostic considerations

Tachycardia-induced cardiomyopathy may follow any type of chronic or frequently recurring paroxysmal tachyarrhythmias. Atrial fibrillation, atrial flutter, ectopic atrial tachycardia, atrioventricular tachycardia, and ventricular tachycardia have all been reported to cause tachycardiomyo-pathy. Tachycardia-induced cardio-myopathy can occur at any age. It has been reported in infants, children [12], adolescents [13], and adults [14,15].

The incidence of tachycardia-induced cardiomyopathy is unknown; most reports have been small retrospective series or case reports involving mostly patients with atrial fibrillation. In selected studies of patients with atrial fibrillation, approximately 25% to 75% of those with left ventricular dysfunction had some degree of tachycardia-induced cardiomyopathy [16-20].

It is unclear why some patients with chronic tachyarrhythmia develop ventricular dysfunction whereas others tolerate high rates and maintain normal systolic function. Presumed risk factors include the type, rate and duration of tachyarrhythmia, patient's age, underlying heart disease, drugs, and coexisting medical conditions [17]. The diagnosis of tachycardiomyopathy can be difficult, and is frequently made in retrospect. The diagnosis of tachycardia-related cardiomyopathy is made when left ventricular systolic function improves to normal or near-normal level after rate control in patients with tachyarrhythmia.

There are no specific tests or markers available to diagnose tachycardia-induced cardiomyopathy. A high index of suspicion derived from history and clinical features remains the only available tool to diagnose this entity. Therefore, the diagnosis of tachycardiomyopathy should be considered in any patient with left ventricular systolic dysfunction and chronic or frequently recurring cardiac arrhythmia. Evidence of previously normal systolic function, is particularly suggestive of this disorder. The ventricular rate that causes tachycardia-induced cardiomyopathy has not been determined, although any prolonged heart rate greater than 100 beats per minute may be important. It is important to recognize that resting heart rates are poor indicators of overall heart rats in patients with atrial fibrillation, because the heart rate response to exercise may vary. Patients with well-controlled resting heart rates may have a rapid ventricular response with minimal activity and develop tachycardia-induced cardiomyopathy [18,19].

Assessment of exercise heart rates and 24-hour Holter monitoring may be useful in diagnosing tachycardiomyopathy in patients with atrial fibrillation and ventricular systolic dysfunction.

Tachycardia-induced cardiomyopathy should be suspected in patients with structural heart disease and heart failure who suffer from chronic or frequently recurring tachyarrhythmias. A tachycardiomyopathy should be always considered in patients with Idiopathic" dilated cardiomyopathy. Grogan et al. [20] reported 10 patients with atrial fibrillation and severe LV dysfunction, initially believed to have idiopathic dilated cardiomyopathy with secondary atrial fibrillation. After a mean follow-up of 30 months, improvement occurred in all patients after control of heart rate with antiarrhythmic drug therapy and/or DC cardioversion. Noninvasive imaging techniques, such as echocardiography or radionuclide ventriculography, usually show left and right ventricular dilatation and systolic dysfunction. Right ventricular biopsy studies revealed nonspecific findings of varying degrees of cellular hypertrophy and interstitial fibrosis consistent with a nonspecific cardiomyopathy [17,21].

Treatment

Heart rate normalization, either by rate or rhythm control, is the cornerstone of therapy. This usually results in increase in ejection fraction, reduction in end-systolic and end-diastolic volumes and improvement of both symptoms and exercise tolerance [16,21-25].

Regardless of the therapeutic approach used, control of the heart rate in patients with chronic supraventricular or ventricular tachycardia has often resulted in a significant improvement of the ventricular function [20,23,24,26,27]. The best means to achieve heart rate control vary depending on the type of arrhythmia. These may include antiarrhythmic drug therapy, external DC cardioversion, radiofrequency catheter ablation, pacemaker therapy or insertion of an implantable cardioverter defibrillator.

Prognosis

Reduction of ventricular rate, either by restoration of sinus rhythm or by slowing the AV conduction, is followed by a slow resolution of the cardiomyopathy [24].

The recovery of ventricular function after termination of or control of the tachyarrhythmia is extremely variable. Recovery may be complete, partial, or totally absent [17,21,24]. The greatest improvement in left ventricular function generally occurs after 1 month of termination or control of

tachyarrhythmia. This is followed by a slower improvement that reaches its maximum after 6-8months [20].

The gradual time course of recovery of left ventricular function after conversion to sinus rhythm or heart rate control, resembles the time course of recovery of left ventricular dysfunction in hibernating myocardium after revascularization and may take up to 1 year [17,24,28]. The recovery of left ventricular function is substantially greater in patients with more profoundly depressed left ventricular function at initial evaluation [26].

Conclusion

Tachycardiomyopathy is a rare but potentially curable form of dilated cardiomyopathy. It should be considered in all patients whose systolic dysfunction is diagnosed subsequent to or concomitant with atrial fibrillation or chronic tachyarrhythmia.

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