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Management of tachycardia

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Abstract

Tachycardia, conventionally, but arbitrarily, defined as an atrial and/or ventricular rate of >100 beats per minute, is encountered commonly and can be physiological or pathological in origin. Various adverse consequences from tachycardia have been recognized, and an important one is the association between persistent tachycardia and cardiomyopathy. In this article, we provide an up-to-date review on the etiology of tachycardia, management strategies, and the prognosis of patients presenting with tachycardia and cardiomyopathy.

Introduction

Heart rate, perhaps the most commonly measured vital sign in clinical practice, is a key determinant of myocardial metabolism and cardiac output [1]. Tachycardia, conventionally defined as an atrial and/or ventricular rate of >100 beats per minute (bpm) has an arbitrary and debated definition [2,3]. Nevertheless, tachycardia can be of importance, since it can cause myocardial ischemia, hypotension, low cardiac output, peripheral hypoperfusion, severe symptoms (chest pain, weakness, syncope, lightheadedness), cardiomyopathy, cardiac arrest and death.

Tachycardias can be broadly classified as: sinus tachycardia (appropriate physiologically and inappropriate); postural orthostatic tachycardia syndrome (POTS); supraventricular tachycardia (atrial tachycardia, AV nodal reentrant tachycardia and AV reentrant tachycardia); atrial flutter with rapid ventricular response; atrial fibrillation with rapid ventricular response; junctional tachycardia; or ventricular tachycardia.

When is tachycardia a problem?

Sinus tachycardia (heart rate >100 bpm) is the form encountered most commonly in clinical practice. The vast majority of sinus tachycardia is physiological and associated with catecholaminergic triggers (e.g. emotions, physical activity, and other stresses). The evaluation and management of persistent sinus tachycardia involves careful assessment of whether tachycardia is an appropriate response or not, the discussion of which is beyond the scope of this manuscript.

A small percentage of patients can have persistent sinus tachycardia without any underlying illness or structural heart disease, and are classified as having inappropriate sinus tachycardia [4,5]. Inappropriate sinus tachycardia is under-recognized, can be associated with debilitating symptoms and poses significant management challenges. Postural orthostatic tachycardia syndrome (POTS), a neurally-mediated disorder (defined as orthostatic tachycardia of >30 beats from baseline or a heart rate >120 bpm with no significant blood pressure changes), can be associated with significant symptoms. Supraventricular tachycardia (i.e. tachycardia requiring tissue above the His bundle to perpetuate) can be associated with severe symptoms, but is rarely life-threatening. Atrial fibrillation and atrial flutter may be associated with rapid ventricular rates. Ventricular tachyarrhythmias can be idiopathic (in the setting of a structurally normal heart) to life-threatening (in the setting of structural heart disease including cardiomyopathy). Ventricular tachycardia can be monomorphic or polymorphic, sustained or non-sustained.

Tachycardia-mediated cardiomyopathy

Persistent tachycardia of any form can cause tachycardia-mediated cardiomyopathy (TMC), can precipitate heart failure and can result in death [6]. If TMC is the direct consequence of tachycardia, it is referred to as tachycardia-induced cardiomyopathy or "pure" TMC [7]. Tachycardia can also worsen pre-existing cardiomyopathy ("impure" TMC). TMC is partially or completely reversible, when measured by heart failure symptoms and left ventricular ejection fraction, once the culprit tachycardia is treated adequately.

Tachycardias causing cardiomyopathy

Any persistent tachycardia (Table 1) can result in TMC. Atrial fibrillation with persistent rapid ventricular rates is the most common cause [6,8]. Sinus tachycardia and POTS are usually not associated with TMC for unclear reasons. Thyrotoxicosis resulting in persistent sinus tachycardia or atrial fibrillation and consequent high output heart failure does not usually cause TMC [9].

Management of a patient with tachycardia and cardiomyopathy

The primary management strategy in TMC is focused on aggressive attempts to control tachycardia with the aim of improving heart failure symptoms and reversing left ventricular dysfunction [6]. Depending on the clinical condition of the patient and type of tachycardia, rate control and/or rhythm control strategies are usually employed. Underlying disease conditions, if present, should be optimized as much as possible and as soon as possible.

If successful rate control or tachycardia elimination can reverse heart failure symptoms and cardiomyopathy, TMC is confirmed [8]. In patients with TMC, standard heart failure therapy (beta-blockers, angiotensin-converting enzyme inhibitors, and spironolactone) can attenuate neurohumoral response and affect favorable remodeling [6].

Table 1. Arrhythmias responsible for tachycardia-mediated cardiomyopathy

Supraventricular
Atrial fibrillation
Atrial flutter
Atrial tachycardia
AV nodal reentrant tachycardia
AV reentrant tachycardia
Permanent junctional reciprocating tachycardia (PJRT)
Ventricular
Idiopathic ventricular tachycardia
Fascicular tachycardia (left septal ventricular tachycardia)
Ectopy
Frequent premature ventricular contractions
Frequent premature atrial contractions
Pacing
High-rate atrial pacing
Persistent rapid ventricular pacing

Rate control is commonly employed to manage atrial fibrillation causing TMC. Beta-blockers, calcium-channel antagonists and/or digoxin are commonly utilized for rate control. The optimal rate control strategy in TMC is yet to be identified, although a combination of drugs is often needed for adequate rate control; a beta-blocker combined with digoxin may have superior benefits [10,11].

The requirements for adequate rate control in TMC remain uncertain. In permanent atrial fibrillation, lenient rate control (resting ventricular rate <110 bpm) has been shown to have similar short-term outcomes to a strict rate control approach (resting heart rate <80 bpm and exercise heart rate <110 bpm). Whether a lenient approach to rate control applies to patients at risk of developing TMC is unlikely but requires further evaluation [12]. In patients with atrial fibrillation-mediated TMC, who are difficult to rate control and in whom a rhythm control strategy is not feasible or desired, AV node ablation with pacemaker implantation provides an effective means of rate control [13]. Recent data, however, arguably point to a cardiac resynchronization therapy (CRT) pacing approach [14].

Although rate control is important in atrial fibrillation-mediated TMC, rhythm control may be important as well, as irregularity of the rhythm may also result in development of cardiomyopathy and heart failure [15]. Further 'rate control' in atrial fibrillation may be a misnomer, as the rate is never exactly as it would be in sinus rhythm. Rhythm control strategies involve chronic or intermittent use of antiarrhythmic drugs, pharmacological or electrical cardioversion, and catheter ablation. A hemodynamically unstable patient with atrial fibrillation and rapid ventricular rates often needs emergent cardioversion. Elective external cardioversion can be performed safely in an outpatient setting with minimal complications [16]. Intravenous ibutilide, a class III antiarrhythmic drug, can be effective for pharmacological cardioversion. The dose is 1 mg over 10 minutes, but carries a risk of QT prolongation and polymorphic ventricular tachycardia, and therefore requires several hours of telemetry monitoring following the dose [17]. Vernakalant, an atrium-specific K+ channel blocker, has been shown to be effective in restoring sinus rhythm, may not affect QT prolongation as much, and can be a safer option [18]. However, vernakalant is not approved for use in the US.

It is critical in TMC patients to maintain sinus rhythm in the long-term. Antiarrhythmic drugs and catheter ablation are commonly employed strategies. Amiodarone, which

blocks multiple cardiac ion channels, is most commonly used for this purpose and is superior to sotalol, another class III antiarrhythmic drug [19,20]. However, long-term use of amiodarone has the potential for extracardiac toxicity (hypo- and hyperthyroidism, abnormal liver enzymes, neuropathy, dermatitis, optic neuritis, and interstitial lung disease). Close follow-up of thyroid and liver function every 6 months should be performed [20].

An underutilized, but highly effective, drug is dofetilide, a pure class III antiarrhythmic drug with no beta-blocking properties (unlike sotalol) [21]. No head-to-head comparison has been undertaken with amiodarone but, when used properly and with careful monitoring, this drug can be quite effective in maintaining sinus rhythm without substantial risk for QT prolongation and torsade de pointes. Dofetilide requires initiation in the hospital with strict electrocardiographic supervision. Dosing depends on the initial QTc, renal function and concomitant QT-prolonging drugs [21].

Catheter ablation has emerged as a promising therapy to maintain sinus rhythm in patients with both paroxysmal and persistent atrial fibrillation. In patients with atrial fibrillation and heart failure, catheter ablation of atrial fibrillation can improve quality of life and left ventricular function [22]. In a systematic review of the efficacy of catheter ablation in atrial fibrillation patients with concomitant left ventricular dysfunction, sinus rhythm was maintained in 57% of patients following a single procedure. The success rate increased to 82% with more than one procedure and/or use of antiarrhythmic drug therapy. The mean increase in left ventricular ejection fraction was 13.3%, suggesting effectiveness of catheter ablation in this TMC population [23].

Whether or not rhythm control is superior to rate control in TMC patients with atrial fibrillation is not fully established. Large randomized studies of atrial fibrillation patients (AF-CHF, AFFIRM) have shown that rate control was equivalent to rhythm control for hard clinical endpoints for the patients who were selected to be enrolled [24,25]. However, it is likely that the pathophysiology of atrial fibrillation-mediated TMC varies in the patient population studied in these trials, and these results may not apply to TMC patients. Moreover, these studies compared rate control to antiarrhythmic drugs, mostly amiodarone, and the toxicity of antiarrhythmic drugs may offset benefits gained from maintaining sinus rhythm [26]. In the real world, achieving and monitoring rate control is a challenge in the cardiomyopathy patient. In patients with drug-refractory atrial fibrillation and heart failure, pulmonary vein isolation has been shown to have better outcomes than AV node ablation and CRT pacing, suggesting the superiority of rhythm control [27]. A recent randomized trial (AATAC-AF) compared catheter ablation with amiodarone for rhythm control of persistent atrial fibrillation in patients with heart failure and cardiomyopathy. A total of 203 patients were followed for 24 months and catheter ablation was shown to be far superior to amiodarone in freedom from atrial fibrillation (70% in the ablation arm *vs.* 34% in the amiodarone arm), quality of life, hospitalization rate, and mortality. Left ventricular ejection fraction improved 9.6±7.4% in the ablation arm *vs.* 4.2±6.2% in the amiodarone arm ($P<0.01$) (NCT00729911).

For atrial flutter and supraventricular tachycardias (atrial tachycardia, AV nodal reentry tachycardia, AV reentrant tachycardias, permanent junctional reciprocating tachycardia), a curative strategy by catheter ablation should be pursued whenever possible with a goal of complete elimination of the tachycardia. Success rates are high. Similarly, early use of catheter ablation should be employed for TMC due to idiopathic ventricular tachycardias and/or frequent ventricular premature beats, as it can achieve a complete cure [28].

In summary, our management approach for patients with suspected TMC is to pursue an aggressive rhythm control strategy whenever possible, with the goal of restoring and maintaining sinus rhythm. Aggressive rate control should be pursued in situations where rhythm control is not feasible or desired. Concomitant heart failure therapy with angiotensin-converting enzyme inhibitors and beta-blockers adds value.

Recovery and prognosis of TMC

Once pathologic tachycardia is controlled or eliminated, gradual recovery in left ventricular function and heart failure symptoms is the rule in a patient with TMC. Most "pure" TMC patients are expected to recover within 3-6 months after tachycardia suppression. However, only limited long-term data are available for these patients. A major factor affecting prognosis is tachycardia recurrence. In a study of 24 patients with TMC and heart failure, 5 patients had recurrent tachycardia after recovery in left ventricular function and all had rapid decline in ventricular function within 6 months of recurrence[29], suggesting that structural abnormalities at the ultrastructural level persist despite tachycardia resolution [30]. Thus, careful follow-up and monitoring for arrhythmia recurrence is necessary for these patients. Sudden death has been reported in TMC patients even after recovery in ventricular function [29], highlighting the fact that tachycardia should be controlled before cardiomyopathy ensues.

Conclusions

Tachycardia, a common problem in clinical practice, can be secondary to physiological and/or pathological causes. One major adverse consequence of pathological tachycardia is development of cardiomyopathy with subsequent heart failure. Early recognition is important, and an aggressive approach towards rate and rhythm control of the culprit tachycardia can result in resolution of symptoms and partial or complete recovery of left ventricular function. Concomitant heart failure therapy to aid favorable remodeling is recommended. Close surveillance for arrhythmia recurrence during follow-up is warranted to avoid further decline in ventricular function.

Abbreviations

bpm, beats per minute; CRT, cardiac resynchronization therapy; POTS, postural orthostatic tachycardia syndrome; TMC, tachycardia-mediated cardiomyopathy.

Disclosures

Dr. Rakesh Gopinathannair is a consultant for St. Jude Medical and Abiomed, and a member of the Speaker's Bureau at Pfizer/BMS. Dr. Brian Olshansky is a consultant at Boston Scientific, Medtronic, Daichi Sankyo, Biotronik, Cardionomics, BioControl, Amarin, Boehringer Ingelheim, and On-X.

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