Chapter 4

Tachycardia-Induced Cardiomyopathy

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Additional information is available at the end of the chapter

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Abstract

Virtually, any kind of tachycardia may lead to the development of tachycardia-induced cardiomyopathy. This term refers to left ventricular dysfunction and dilated cardiomyopathy pattern that occur as a consequence of persistent tachycardia. Impaired left ventricular function in the presence of tachycardia can be found accidentally, but it is often associated with progressive symptoms and signs of heart failure that force the individual to seek medical help. A hallmark of tachycardia-induced cardiomyopathy is the reversibility of both hemodynamic and structural changes after cessation of the index tachycardia. However, contractile dysfunction and structural changes may persist even weeks after the rhythm/rate correction. Therefore, tachycardia-induced cardiomyopathy should be considered as a probable reason of ventricular dysfunction and dilatation in any patient presenting with dilated cardiomyopathy pattern, despite that the initial rhythm is not pathological or the heart rate is well controlled. This review summarizes our current knowledge about this specific form of cardiomyopathy.

Keywords: tachycardia-induced cardiomyopathy, arrhythmia, tachycardia, heart failure, dilated cardiomyopathy

1. Introduction

Cardiomyopathies represent a heterogenous group of myocardial diseases in which the myocardium exhibits structural and/or functional dysfunction [1, 2]. Current definition of cardiomyopathies excludes structural myocardial processes and dysfunction secondary to specific cardiovascular disorders such as coronary artery disease, systemic arterial hyperten-

sion, congenital heart diseases or valvular diseases. Although tachycardia-induced cardiomyopathy may be seen as secondary due to well-defined causal cardiovascular factor, this disorder ranks among cardiomyopathies also in the current definition and classification of cardiomyopathies [1, 2].

Tachycardia-induced cardiomyopathy is a disease with attributes of dilated cardiomyopathy that develop as a consequence of persistent tachycardia. It is characterized by systolic ventricular dysfunction and dilatation with heart failure symptoms that occur as a result of long-term tachycardia of either supraventricular or ventricular origin. This disease should be also considered as a cause of ventricular dysfunction in the absence of tachycardia at the time of patient presentation since the tachyarrhythmia could disappear spontaneously before the initiation of patient examination, while the hemodynamic and especially structural changes of the heart induced by long-term tachycardia may persist even weeks after arrhythmia disappearance and/or rate control achievement.

The key feature of tachycardia-induced cardiomyopathy is the reversibility of both functional and structural changes as soon as the heart rate/rhythm is well controlled. In such case, improvement or even complete functional and structural normalization may be found. A correct diagnosis is thus often stated retrospectively based on the observation of ventricular systolic function improvement and regression of ventricular dilatation after appropriate rate/rhythm control.

2. Incidence, prevalence and prognosis

Although tachycardia-induced cardiomyopathy is considered to be a relatively rare instance, its precise incidence is not described well yet. In fact, this disease is very likely to be underdiagnosed in the clinical practice despite that the link between tachycardia and dilated cardiomyopathy pattern has been known for a long time [3-5]. One reason for its underestimation may be the uncertainty whether the detected tachycardia is the primary cause of the cardiomyopathy, or whether it is rather a consequence of cardiomyopathy of different origin.

Tachycardia-induced cardiomyopathy may develop at any age. It has been documented in fetuses with persistent supraventricular tachycardias [6], in children and adolescents [7] as well as in adults [8].

Virtually, any type of arrhythmia is capable of inducing ventricular dysfunction or cardiomyopathy; however, supraventricular arrhythmias are the most commonly reported causes of tachycardia-induced cardiomyopathy. It has been predominanly described in association with atrial fibrillation, but other supraventricular arrhythmias may lead to this pathology too. Tachycardia-induced cardiomyopathy may also develop as a result of persistent ventricular tachycardia, rapid atrial and/or ventricular pacing or as a consequence of some extracardiac diseases that are associated with persistent tachycardia (Table 1). Importantly, it may also occur in patients with "only" frequent ventricular premature beats [9].

Atrial fibrillation (the most frequently reported arrhythmia associated with tachycardia-induced cardiomyopathy development) is a frequent type of supraventricular tachycardia

Supraventricular arrhythmias	Atrial fibrillation [13, 73]
	Atrial flutter [14, 74]
	Focal atrial tachycardia [15, 16]
	AVNRT [17, 18]
	AVRT [19, 75]
	Permanent junctional reciprocating tachycardia (PJRT) [20]
Ventricular arrhythmias	Frequent ventricular premature beats (VPBs) [9, 76]
	Idiopathic right or left ventricular outflow tract tachycardia [7, 8]
	Bundle-branch reentry ventricular tachycardia [77]
Cardiac pacing	Atrial pacing at high rates [30]
	Ventricular pacing at high rates [32]
Extracardiac causes	Thyreotoxicosis [78]
	Glucagonoma [79]
Myocarditis [80]	

Table 1. Disorders associated with tachycardia-induced cardiomyopathy development (adapted from [81])

in patients with dilated cardiomyopathy and heart failure. It appears that there is a close relationship between atrial fibrillation and heart failure: heart failure progression supports electrical and structural remodeling of the heart, which finally leads to atrial fibrillation development. On the other hand, epidemiological studies have demonstrated that patients with atrial fibrillation are at higher risk of heart failure [10] and that abnormal left-ventricular systolic function is 2.5 times more likely in elderly patients (> 65 years) with atrial fibrillation than in those without this arrhythmia [11]. In addition, sinus rhythm restoration or adequate rate control of ongoing arrhythmia are associated with the improvement or even normalization of left-ventricular ejection fraction in some of these patients. These findings indicate that at least in some cases, left-ventricular dysfunction is primarily caused by rapid heart rate during atrial fibrillation rather than by preexisting dilated cardiomyopathy. Moreover, some studies indicate that approximately 25–50% of patients with atrial fibrillation, who simultaneously suffer from ventricular dysfunction, have some degree of tachycardia-induced cardiomyopathy [12, 13].

Another instance of tachyarrhythmia that used to be associated with a relatively higher prevalence of cardiomyopathy pattern is **atrial flutter**. Some observational studies indicated that left-ventricular systolic dysfunction is present in up to 25% of patients reported to atrial flutter ablation, and in more than half of these, it is possible to observe a significant improvement or even normalization of ejection fraction during the first twelve months after successful elimination of the tachycardia [14].

Prevalence of ventricular dysfunction is relatively high also in incessant **atrial tachycardia,** since it has been reported in approximately 10–19% of cases [15, 16]. It seems that children are more susceptible to tachycardia-induced cardiomyopathy than adults. Among adults, tachycardia-induced cardiomyopathy appears to be present more often in younger adult patients with persistent atrial tachycardia than in those of a higher age, although the lower

prevalence in the older population may be partially caused by difficulties to distinguish the effect of tachycardia alone from that of the underlying heart disease with regard to the systolic ventricular function. Interestingly, elimination of the arrhytmia leads to the restoration of left-ventricular function in up to 97% [16].

Supraventricular reentrant tachycardias (atrioventricular nodal reentry tachycardia (AVNRT) and atrioventricular reciprocating tachycardia (AVRT)) have usually only paroxysmal nature. If persistent, these may also induce cardiomyopathy and heart failure that are reversible after catheter ablation [17-20].

Ventricular tachycardias are usually associated with some forms of underlying structural heart diseases. Therefore, it is difficult to distinguish whether and to what extent is the observed systolic ventricular dysfunction caused by the primary disease, and how much has persistent tachycardia contributed to its severity. This may be determined more precisely in patients with idiopathic ventricular tachycardias, which (if persistent or repetitive enough) may lead to cardiomyopathy. Restoration of normal systolic performance of the left ventricle has been described in patients with successful elimination of both right-ventricular outflow tract tachycardia [8] and left-ventricular outflow tract tachycardia [7].

However, cardiomyopathy with or without symptomatic heart failure may be present also in patients with ”only” **frequent premature beats.** It has been described mainly in patients with frequent ventricular ectopy [9, 21-23], but high burden of atrial premature beats may also lead to reversible cardiomyopathy [24, 25]. A prospective multicenter study that included patients with reduced left-ventricular ejection fraction due to suspected ventricular premature beat-associated cardiomyopathy has found that both systolic function and neurohumoral response (levels of natriuretic peptide) improve as soon as the ectopy is successfully eliminated and that the extent of improvement is comparable between patients with and without known structural heart disease [26]. These authors suggested that the higher the burden of ventricular premature beats (VPB), the higher the probability of systolic function improvement after ectopy elimination, with 13% baseline VPB burden being 100% sensitive and 85% specific to predict left-ventricular ejection fraction increase by ≥5% after elimination of the ectopic focus. Although not confirmed in all studies [26], some researchers have also suggested that the QRS duration of VPB is an important factor in the development of cardiomyopathy, with wider QRS complexes more likely to lead to cardiomyopathy pattern with a lower total burden of PVBs [27, 28] and that epicardial origin of VPBs is also associated with delayed LV function recovery [29].

Prognosis of dilated cardiomyopathy may vary depending on the cause. Tachycardia-induced cardiomyopathy ranks among forms of dilated cardiomyopathy with (if treated appropriately) generally good prognoses. Although studies describing recovery of patients with tachycardia-induced cardiomyopathy after sinus rhythm restoration or rate control include only small samples of patients, it appears that these causal therapeutic options may result in an improvement of left-ventricular function, positive change in neurohumoral cascade (reduced levels of natriuretic peptides) [26] and that it is likely to be linked to a generally good prognosis of tachycardia-induced cardiomyopathy.

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3. Pathogenesis

Our current knowledge about the pathogenesis of tachycardia-induced cardiomyopathy is predominantly based on animal models that have been introduced to study heart failure [30-35]. In these experiments, heart failure is induced by rapid cardiac pacing of certain duration, at the atrial and/or ventricular level. Hemodynamic changes that result from such pacing strongly resemble findings in humans [31-35]. Similar abnormalities have been also identified in animals with "only" ventricular premature beats delivered artificially in bigeminal pattern, and these changes are reminiscent of those in humans as well [36]. Observations proving that all these alterations are fully or at least partially reversible with cessation of tachycardia also correspond with results found in humans [32, 37].

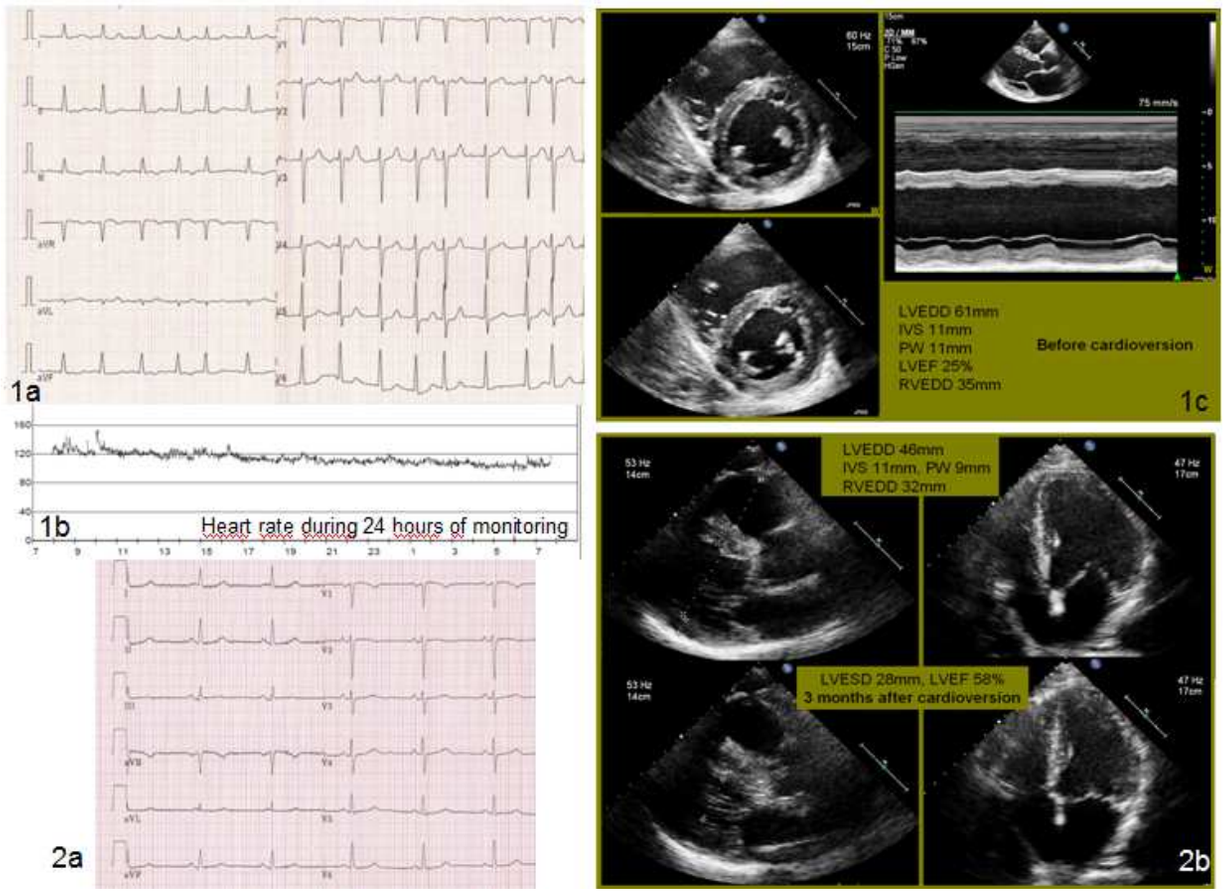

Figure 1. Tachycardia-induced cardiomyopathy in a 38-years old male with no previous history of any heart disease or arrhythmia. First episode of persistent atrial fibrillation with rapid heart rate (1a, 1b) in his life caused progressive deterioration of ventricular systolic function leading to clinical manifestation of heart failure during 4 weeks. 1c shows significantly reduced ejection fraction of the left ventricle with its incipient enlargement during the tachycardia. Both heart failure symptoms and left-ventricular systolic function rapidly improved during the following 3 weeks after sinus rhythm restoration (2a). Size and systolic function of the left ventricle then almost normalized in the horizon of 3 months after the tachycardia termination (2b).

3.1. Hemodynamic changes

Rapid atrial or ventricular pacing in experimental models leads to biventricular systolic and diastolic dysfunctions that occur relatively early after the onset of tachycardia [38, 39]. Typical findings include reduced systolic function of both ventricles reaching up to 55%, decline in cardiac output, elevation of ventricular filling pressures, systemic vascular resistance as well as ventricular systolic wall stress [33-35, 40, 41]. Systolic ventricular dysfunction is caused by the loss of contractility. In addition, contractile reserve in response to volume, inotropic agents or post-extrasystolic potentiation is affected [42, 43] and myocardial relaxation is impaired [44]. As a consequence of altered hemodynamic situation, mitral regurgitation may develop in longer-term perspective that leads to volume overload of the failing heart [45].

3.2. Structural changes

Persistent tachycardia, either induced artificially by chronic cardiac pacing or resulting from persistent tachyarrhythmia, finally leads to the dilatation of all cardiac chambers. Left ventricle increases both its end-systolic and end-diastolic volume (with end-systolic volume being affected more [33, 34]) and it further changes its geometry to a spherical shape [46]. This dilatation is usually associated with wall thinning or preservation of wall thickness without either increased heart weight or hypertrophy [45, 46].

On microscopical level, remodeling of both cardiomyocytes and extracellular matrix may be detected: disruptions of sarcolemma-basement membrane interface, myofibrillar misalignment, cellular elongation and myocyte loss (up to 39% of the total myocyte mass) have been found [40, 47, 48]. Architecture of the extracellular matrix is usually remarkably modified as well and this also contributes to myocyte misalignment and adversely affects force coupling and transmission.

In addition to functional and structural changes, electrophysiologic remodeling has been documented. Abnormal functioning of calcium channels, stretch-sensitive channel dysfunction and other abnormalities often affect and prolong repolarization that can finally result in ventricular tachycardias manifestation [49].

3.3. Neurohumoral changes

Similar to other forms of heart failure, marked neurohumoral activation occurs and leads to elevated plasma levels of natriuretic peptides, epinephrine, norepinephrine, aldosterone and renin activity [39]. Downregulation of beta-1 receptor density with the resulting decrease in beta-adrenergic responsiveness have been found as well [50, 51].

All the above-mentioned hemodynamic, structural and neurohumoral changes have been demonstrated also in humans, which supports the conclusion that pacing-induced model of heart failure could be a useful tool to study pathogenesis of tachycardia-induced cardiomyopathy.

3.4. Pathophysiology of tachycardia-induced cardiomyopathy

The development of tachycardia-induced myocardial dysfunction appears to be a result of multiple factors. Various alternations of neurohumoral and cellular activation have been identified; however, it is still uncertain whether they represent the causal factor of the functional and subsequent structural changes in all cases, or whether they are rather a consequence of tachycardia. Although the precise mechanism behind contractile dysfunction with the resulting structural changes is not fully understood yet, the research has focused mainly on three potential factors: *1) depletion of high-energy stores in the myocardium with impaired energy utilization, 2) myocardial ischemia and 3) abnormality in calcium handling*.

On subcellular level, persistent tachycardia leads to energy-stores depletion, which is associated with the reduction of adenosin-triphosphate (ATP), phosphocreatinine and creatinine levels in the myocardium. In addition, a reduced activity of the Na/K-ATPase pump has been described [52, 53a, 53b]. It is very likely due to enhanced activity of Krebs cycle oxidative enzymes and mitochondrial injury [40, 41].

Similar findings have been described in the case of myocardial ischemia. In the ischemic model, rapid depletion of energy-stores and left-ventricular dysfunction occur shortly after vessel occlusion [54]. However, their return to normal values lasts mostly about days long after the ischemic attack, which also corresponds with findings in tachycardia-induced cardiomyopathy where altered hemodynamic and structural changes resolve in prolonged time interval. Proceeding from these facts, myocardial ischemia is considered to be a potential factor that contributes to the tachycardia-induced cardiomyopathy development. In fact, abnormal coronary flow and changed ratios of subendocardial and subepicardial flow have been observed in tachycardia-induced cardiomyopathy [55, 56].

The hypothesis that abnormal handling of calcium plays a role in the genesis of tachycardia-induced cardiomyopathy has received substantial support, because the severity of calcium cycling abnormalities has been shown to correlate with the extent of ventricular dysfunction [41]. Abnormal calcium handling occurs already in the first 24 hours of rapid cardiac pacing and it may persist for more than 4 weeks after tachycardia termination [41]. Altered functions of calcium channels and transport system of the sarcoplasmatic reticulum have been identified [41, 57] and they may thus contribute to myocardial dysfunction observed in tachycardia-induced cardiomyopathy due to lower calcium availability to myocytes with the resulting contractility reduction. Some other studies suggest altered calcium-sensitivity and excitation-contraction coupling [57, 58].

An observation that tachycardia-induced cardiomyopathy does not evolve in every patient with the same type, duration and rate of a tachycardia implies *possible genetic predisposition* of some patients to develop a dilated cardiomyopathy pattern during tachycardia. In fact, one study [59] suggests that polymorphism in angiotensin-converting enzyme (ACE) gene may be involved, since one type of such polymorphism (which is associated with higher serum levels of ACE) is more frequently linked to idiopathic and ischemic cardiomyopathy manifestation. Looking at the prevalence of this polymorphism among 20 patients with tachycardia-induced cardiomyopathy as compared to another sample of 20 patients without this pattern, the authors reported a higher detection of this ACE polymorphism in the cardiomyopathic group [59].

3.5. Course of the changes over time and their reversibility

Experimental studies demonstrate that changes of hemodynamics with the reduction of cardiac output or altered systemic arterial pressure occur already in the first 24 hours of rapid pacing [32]. When continued, fast cardiac pacing then induces an elevation of ventricular filling pressures, pulmonary artery pressure and a decrease of systemic arterial pressure that reach certain plateau after one week, while cardiac output, volumes and ejection fraction deteriorate continually for 3–5 weeks with the final end-stage heart failure development [35, 45].

Cessation of tachycardia results in a resolution of these changes: in the first 48 hours after termination of cardiac pacing, a significant improvement of cardiac output, systemic vascular resistance, mean arterial pressure and filling pressures are present [39]. Left-ventricular ejection fraction also improves dramatically and normalizes within 1–2 weeks [39]. All hemodynamic variables normalize within the horizon of four weeks after tachycardia interruption, but diastolic dysfunction remains detectable even after the first month period. Importantly, elevated end-diastolic and end-systolic volumes are still present after twelve weeks of pacing discontinuation, which is consistent with substantial ventricular remodeling [33, 46], that requires longer time for its resolution.

Although the hallmark of tachycardia-induced cardiomyopathy is an improvement or even normalization of cardiac function and size with the resulting disappearance of heart failure symptoms after cessation of tachycardia or rate control achievement, there is growing evidence that the ultrastructural abnormalities of the myocardium and residual contractility dysfunction may persist. This has already been suggested in the experimental studies [53b], but similar conclusions have also been made in clinical observational reports [60, 61]. In one of these studies, ventricular function has been assessed using specle tracking and contrast-enhanced MRI with ventricular T1 mapping used as an index of diffuse fibrosis. Although the ejection fraction normalized three months after a successful ablation of initial tachycardia already, it has been possible to detect a somewhat greater indexed end-diastolic and end-systolic volume of the left ventricle in patients with tachycardia-induced cardiomyopathy as compared to healthy controls. Moreover, patients with previous tachycardia-induced cardiomyopathy have demonstrated reduced global left-ventricular corrected T1 time that implies a diffuse fibrosis [61]. In addition, another study [60] suggests that tachycardia tends to recur in some patients initially diagnosed with tachycardia-induced cardiomyopathy pattern and this recurrence of arrhythmia leads to a new decline of systolic ventricular function. Moreover, sudden death may occur in some of these patients despite normal or almost normal systolic function during the last evaluation [60].

4. Clinical manifestation and diagnosis

It is important to consider tachycardia-induced cardiomyopathy in all cases as a possible cause of systolic dysfunction and manifest heart failure, especially in patients with a new or worsened ventricular systolic dysfunction or in cases with their uncertain duration in which persistent tachycardia is found simultaneously. Since contractile dysfunction and structural

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changes may persist even weeks after the rhythm/rate correction, tachycardia-induced cardiomyopathy should be considered as a probable reason of ventricular dysfunction and dilatation in any patient presenting with dilated cardiomyopathy pattern, despite that the initial rhythm is not pathological or the heart rate is well controlled.

Generally, persistent tachycardia (i.e. tachycardia lasting usually weeks or months) predisposes an individual to ventricular dysfunction and dilatation development, regardless of the rhythm disturbance characteristics. However, the resulting degree of systolic dysfunction and heart dilatation, the rate of their progression and the reversibility of hemodynamic and structural abnormalities after rhythm/rate correction are partially dependent on the heart rate, type and duration of the tachycardia and also on the concomitant presence of other heart diseases. In addition, there is growing evidence that irregularity of the rhythm alone may contribute to these changes manifestation and that it may also affect the rate and the extent of their resolution after rhythm correction.

Heart rate is apparently one of the most important factors. Tachycardia-induced cardiomyopathy is rate dependent: tachycardias with higher rates manifest themselves usually earlier than those with slower rates [31, 62]. In an experimental setting, pacing at slower rate or for a shorter time usually yields a lesser degree of left-ventricular systolic dysfunction [39, 63]. However, some observational studies imply that tachycardia-induced cardiomyopathy may be found more often in patients with a slower heart rate than in those with a higher rate. This was reported in retrospective observational studies that included patients referred to catheterization ablation of focal atrial tachycardia [15, 16]. Tachycardia-induced cardiomyopathy was present in 9–19% of these cases, more frequently in patients with heart rate of less than 120 bpm. Possible explanation for this finding may include the fact that faster tachycardia is associated with early symptom manifestation in form of palpitation, whereas tachycardias with lower heart rate are better tolerated by the patient, so the remodeling and signs of heart failure have enough time to develop before the patient visits a doctor.

How fast does the tachycardia have to be to induce tachycardia-induced cardiomyopathy is still not clear. Basically, any rhythm with the rate exceeding 100 bpm for a longer period of time may lead to this pathology. In this context, it is important to note that heart rate (especially in atrial fibrillation) may vary significantly as a result of physical or mental activity, i.e. patients may show well-controlled heart rate at rest that increases abnormally during minimal exercise. Therefore, Holter ECG monitoring may be useful to identify such behavior and to raise suspicion of tachycardic origin of an observed ventricular dysfunction. Cut-offs for adequate rate control have been derived from atrial fibrillation patients. They generally vary with age, but heart rate ranging between 60 and 80 bpm at rest and 90–115 bpm during a moderate exercise are usually considered as adequate (so-called strict rate control) [64]. These target rates are sometimes difficult to achieve, however. A lenient rate control strategy [65] aiming at resting heart rate <110 bpm seems to have similar long-term results as the strict rate control of atrial fibrillation and is thus preferred nowadays.

In the experimental model, persistent **ventricular tachyarrhythmias induce** generally **more significant ventricular dysfunction** than supraventricular tachycardias [38].

Tachycardia-induced cardiomyopathy may develop in a variable time-horizon since the tachycardia onset (even after many months). Together with heart rate and type of the arrhythmia, the **tachycardia duration** is responsible for the severity and reversibility of ventricular systolic dysfunction [31] as it has been discussed earlier.

Tachycardia-induced cardiomyopathy can be present as either sole pathology or it **may accompany another heart disease**. It means that the presence of other structural heart disease does not exclude concurrent presence of tachycardia-induced cardiomyopathy. In these cases, tachycardia may worsen the pre-existing ventricular dysfunction and dilatation [12]. The degree of a ventricular dysfunction is then usually inadequate to the severity of the underlying heart disease (i.e. atherosclerotic changes of the coronary arteries). Tachycardia-induced cardiomyopathy is highly probable especially in patients with recently normal systolic function of the ventricles and in those with an improvement of dysfunction after the achievement of an adequate rate/rhythm control.

Another factor which appears to contribute to tachycardia-induced cardiomyopathy is the **irregularity** of a rhythm. It is based not only on the observations that frequent ectopic supraventricular or ventricular beats are able to induce the pattern of reversible dilated cardiomyopathy [9, 22, 24], but also on studies on rate control in atrial fibrillation [66], which demonstrate that the irregularity of a fast rhythm may worsen cardiac function whereas it does not cause any significant hemodynamic worsening if the heart rate of the irregular rhythm falls generally within a normal range.

Assessment of a patient with suspected tachycardia-induced cardiomyopathy includes a detailed history and physical examination with the subsequent laboratory and imaging tests to state the diagnosis and severity of the disease.

4.1. Symptoms and signs

The manifestation of tachycardia-induced cardiomyopathy is various. The most common symptoms include palpitations and signs of heart failure. Palpitations resulting from either high rate or irregularity of the arrhythmia are often the dominant complaint of a patient with fast tachycardia. These patients thus often consult a doctor early in search of intervention and cardiomyopathy pattern then does not have enough time to develop. On the other hand, symptoms and signs of a heart failure may predominate in those patients who do not feel palpitation and are not aware of a rhythm disturbance. In such patients, decreased exercise capacity, fatigue or congestion may be the main complaint.

Due to the fact that acuteness of the symptoms forces a patient with rapid heart rate to seek medical help shortly after the arrhythmia onset, some scholars hypothesize that tachycardia-induced cardiomyopathy is present rather in patients with slower arrhythmias. Some clinical observations support such conclusions [15, 16]: The first retrospective observational study [16] included a sample of 331 patients without structural heart diseases who underwent ablation of atrial tachycardia. Tachycardia-induced cardiomyopathy was present in 9% of them and affected rather younger patients (mean age 39 vs. 51 years), more often males (60% versus 38%) with incessant or very frequent paroxysmal tachycardia (100% vs. 20%) with a slower heart

rate (120 bpm vs. 149 bpm), than arrhythmias that have not been associated with ventricular dysfunction. Similar findings have been reported also in children with persistent atrial tachycardias [15]: atrial tachycardia originating within the atrial appendage was more often associated with slower heart rate (<120 bpm) at examination, asymptomatic course (75% versus 25%) and higher prevalence of tachycardia-induced cardiomyopathy as compared to atrial tachycardia of other origins.

In all other aspects, tachycardia-induced cardiomyopathy resembles other forms of dilated cardiomyopathy: symptoms and signs of heart failure, their severity as well as neurohumoral activation are principally similar.

4.2. Diagnosis

As soon as tachycardia-induced cardiomyopathy is suspected, tests that aim to confirm the diagnosis take place.

All patients should have **12-lead ECG** to document basic heart rhythm and its rate at patient presentation. Especially in cases with persistent atrial tachycardia, it is helpful to compare recent ECG tracing with an older one (if available) to distinguish whether the current P wave morphology corresponds with the documented sinus rhythm morphology or if it is rather suggestive for atrial focus. Since the heart rate may change over time, due to mental or physical activity, patients with suspected tachycardia-induced cardiomyopathy should undergo **continuous ECG monitoring** for at least 24 hours (ambulatory Holter ECG monitoring or in-patient telemetry). If uncertainty regarding the underlying rhythm persists, electrophysiologic testing should be considered.

Besides arrhythmia detection, the presence of left-ventricular dysfunction with/without ventricular dilatation should be documented. With this regard, **transthoracic echocardiography** represents a gold standard. Morphological findings are principally similar as in the dilated cardiomyopathy of other origins and differentiation between tachycardia-induced cardiomyopathy and other forms of dilated cardiomyopathy is generally not possible based on the echocardiographic pattern only, although left-ventricular end-diastolic diameter tends to be usually smaller in cases of tachycardia-induced cardiomyopathy [67].

Despite that the patient presents with tachyarrhythmia, other reasons of dilated cardiomyopathy pattern should be therefore considered as soon as the ventricular dysfunction and dilatation is documented at any imaging modality. Adult patients are thus often indicated to coronary angiography to exclude the most common substrate of ventricular dysfunction, i.e. a significant underlying coronary artery disease. History of alcohol intake, drug abuse, cancer and its treatment, thyreopathy or other metabolic or congenital disease should be searched for further.

A single-center experience suggests that also **serial NT-pro BNP** measurement may be useful to distinguish between tachycardia-induced cardiomyopathy and cardiomyopathies due to structural heart disease [68]. This study included patients who presented with supraventricular tachycardia and reduced left-ventricular ejection fraction <40% and underwent cardioversion. The NT-pro BNP level has initially been elevated in all patients. After a successful

cardioversion, NT-pro BNP has decreased in virtually all patients, but the decrease has been quicker in tachycardia-induced cardiopmyopathy patients. Therefore, the ratio between the baseline NT-pro BNP and NT-pro BNP after one week following cardioversion ≥ 2.3 has had 90% sensitivity, 95% specificity and 90% accuracy to predict tachycardia-induced origin of ventricular dysfunction based on these authors.

5. Therapy

Therapeutic approach to the tachycardia-induced cardiomyopathy includes two steps: 1. tachyarrhythmia correction as it represents causal therapeutic intervention and 2. heart failure treatment.

Due to the potential reversibility of hemodynamic and structural changes in tachycardia-induced cardiomyopathy, all efforts should be made to achieve **heart rate correction or appropriate rate control.** Rhythm or rate control may be achieved using both pharmacological and non-pharmacological tools. Depending on the type of arrhythmia and presence/absence of concomitant structural heart disease, various antiarrhytmic drugs may be used to terminate the arrhythmia or to achieve adequate rate control. Especially, betablockers and class III antiarrhythmics play an irreplaceable role regarding this treatment. It is very important to avoid drugs with higher pro-arrhythmic effect (e.g. flecainide) in the presence of systolic dysfunction or drugs that may contribute to further progression of systolic dysfunction (e.g. disopyramide). Most arrhythmias that lead to tachycardia-induced cardiomyopathy are currently treatable using catheterization ablation, success rate of which reaches 60–90% depending on the type of arrhythmia. This therapeutic approach should be therefore considered as a first-line treatment in the absence of contraindication.

In atrial fibrillation, rate and rhythm control strategy have been shown to be comparable with respect to quality of life, mortality or stroke rate [64, 69]. The decision to favor rhythm control over rate control should thus be made on an individual basis, and discussed with the patient [70]. In case rate control strategy is chosen, repeated long-term ECG monitoring is instrumental to decide whether the selected treatment is appropriate and ensures acceptable rate control (strict rate control requires 60–80 bpm at rest and 90–115 bpm at moderate exercise; lenient rate control requests resting rate < 110 bpm). Atrial arrhythmias are often refractory to antiarrhythmic drugs and an acceptable rate control may be then achieved only with higher doses of AV nodal blocking agent. In such cases, catheter ablation is an option. By other supraventricular tachyarrhythmias, which lead to tachycardia-induced cardiomyopathy, restoration of sinus rhythm is usually preferred. Rhythm correction may be achieved through either pharmacological or electrical cardioversion or (preferably) via catheter ablation of the arrhythmia in these patients.

In rare cases, failing to restore sinus rhythm (even using catheter ablation) and to achieve adequate ventricular control, an ablation of AV node with insertion of a permanent pacemaker may be considered. Because of the present ventricular dysfunction prior to pacemaker insertion, biventricular systems are usually favored [71].

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Treatment of heart failure symptoms due to tachycardia-induced cardiomyopathy includes standard regimen and drug spectrum as in heart failure of other origin, i.e. ACE inhibitors, beta-blockers, angiotensin-receptor blockers, diuretics and digoxin.

6. Risk of tachycardia-induced cardiomyopathy recurrence

Similarly as in the experimental models, improvement of ventricular systolic function may be often found in one week horizon and full recovery (including chamber size reduction), usually over a time period of 4–6 weeks after rhythm/rate correction. In some patients, size of the left ventricle may remain slightly enlarged [72].

Although there are no recommendations regarding follow-up of the patients who once experienced tachycardia-induced cardiomyopathy, it is advisable to observe such patients closely for at least one or two years after the initial manifestation. The reason for it is certain, although not specifically determinable risk of arrhythmia recurrence that may induce new and rapid decrease of left-ventricular ejection fraction despite cardiomyopathy could develop within months during the initial episode. Patients experiencing the recurrence of tachycardia-induced cardiomyopathy are at higher risk of sudden death [72] and implantation of an ICD may be thus considered in these cases.

7. Conclusion

The difficulties to differentiate reliably between tachycardia-induced cardiomyopathy and other forms of dilated cardiomyopathy, and the fact that the correct diagnosis is often established only retrospectively based on the improved systolic function after heart rate correction are the two most important reasons why the real prevalence of tachycardia-induced cardiomyopathy may be much higher than it is currently reported. Since it is a potentially reversible cause of heart failure, it is very important to always consider this option in the concomitant presence of dilated cardiomyopathy pattern and persistent tachycardia. An early heart rate intervention may then have substantial clinical impact.

Although experimental studies helped us to understand the basic pathophysiologic process behind the tachycardia-induced cardiomyopathy development, there is still a number of questions that need to be answered. For example, it is not clear why the tachycardia-induced cardiomyopathy does not occur in all patients with persistent tachycardia of the same type and heart rate. Furthermore, it is not clear whether or not are the patients who once developed tachycardia-induced cardiomyopathy in their life more susceptible to heart failure of another origin in the long-term perspective, similarly as are women with gestational diabetes more prone to develop the second type of diabetes in their future life. In addition, the exact pathophysiology behind tachycardia-induced cardiomyopathy is still not well understood. All these and other questions should therefore become the subjects of future research.

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