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Tachycardiomyopathy

Tachykardiomiopatie

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S u m m a r y

Tachycardiomyopathy (TCM) or tachycardia-induced cardiomyopathy, caused by improper tachycardia in the course of supraventricular or ventricular arrhythmias is a form of secondary dilated cardiomyopathy.

This disease may occur at any age and leads to both dysfunction and changes in heart muscle structure.

Tachycardiomyopathy is characterized by a significant reduced left ventricular function. This dysfunction is partially or completely reversible with normalization of basic sinus rhythms and heart rate.

The concept of tachycardiomyopathy does not include disorders resulting from hypertensive disease, heart valve damage and coronary heart disease.

Tachycardiomyopathy is characterized by a wide range of symptoms, most commonly corresponding to heart failure in course of cardiomyopathy. If treated casually, tachycardiomyopathy is fully reversible. In the extreme, not treated cases, this condition leads to severe heart failure and death. The diagnosis of tachycardiomyopathy is not easy because dilation of the ventricles may cause or be a result of tachyarrhythmias. Moreover, there are no specific markers available to precise diagnose and clinical features remain the most useful tool in everyday practice.

S t r e s z c z e n i e

Tachykardiomiopatię (TCM) określaną również jako kardiomiopatią indukowaną tachykardią można zdefiniować jako wtórną formę kardiomiopatii rozstrzeniowej indukowaną nadmierną tachykardią towarzyszącą różnym postaciom tachyarytmii nadkomorowych i komorowych.

Pojawia się w każdym wieku i prowadzić może do dysfunkcji czynnościowej jak również zmian morfologicznych mięśnia sercowego.

Tachykardiomiopatia cechuje się istotnym upośledzeniem czynności lewej komory, częściowo lub nawet całkowicie odwracalnym po przywróceniu podstawowego rytmu zatokowego lub uzyskaniu kontrolowanej względnie prawidłowej częstości akcji serca.

Pojęcie tachykardiomiopatii nie obejmuje swoim zakresem stanów wynikających z choroby nadciśnieniowej serca, uszkodzenia zastawek serca i choroby wieńcowej.

Chorzy z tachykardiomiopatią prezentują szeroki zakres różnorodnych objawów, najczęściej jednak objawy te wynikają z postępującej wraz z czasem trwania tachyarytmii niewydolności serca.

Leczona, tachykardiomiopatia jest w większości przypadków w pełni odwracalna. Nieleczona prowadzić może do ciężkiej niewydolności serca i zgonu chorego. Rozpoznanie tachykardiomiopatii jest utrudnione z uwagi na fakt, że poszerzenie jam serca może być zarówno przyczyną tachyarytmii jak i jej skutkiem.Brak jest ponadto specyficznych dla tej jednostki markerów diagnostycznych, co sprawia, że szczegółowa analiza obrazu klinicznego pozostaje jak na razie jedyną drogą rozpoznania w codziennej praktyce.

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Long-term persistence of tachyarrhythmia for many years has been identified as one of the possible causes of heart failure and cardiomyopathy in the wake of the mostly dilated nature (1). In the cases of heart failure, it was noted that there is a close relationship between inhibition deterioration progress of heart muscle function and elimination of the arrhythmia or at least release frequency of arrhythmia. This involved a substantial improvement of the prognosis if arrhythmia were reduced. The actual incidence of tachycardiomyopathy is not known, it is difficult to conclusively determine whether tachyarrhythmia is the leading cause of heart

failure in the patient, or is only a secondary process to an existing, significant muscle damage.

Tachycardiomyopathy may develop on any form of supraventricular arrhythmias (2). However, it should be specified as the main atrial fibrillation, atrial flutter, and various forms of atrial paroxysmal tachycardia. Also arrhythmias such as ventricular tachycardia and ventricular premature beats may lead to tachycardiomyopathy.

Common causes of tachycardiomyopathy is described in table 1.

Table 1. Types of arrhythmia-inducing tachycardiomyopathy.

Supraventricular arrhythmias	**Ventricular arrhythmias**
– Atrial fibrillation – Atrial flutter – Atrial tachycardia – AVRT – AVNRT – Junctional tachycardia – Sinus tachycardia (pathological)	– Ventricular tachycardia originated from right ventricular outflow tract – Ventricular tachycardia bi-static beam – A number of premature ventricular beats – Idiopathic ventricular tachycardia from the left ventricle
Other (less common) causes	
Stimulation of the atria with a high frequency	Stimulation of the ventricles with a high frequency
Other causes of tachycardiomyopathy	
– Myocarditis accompanied by tachycardias and tachyarrhythmias – Thyreotoxicosis – Glucagonoma	

PATHOPHYSIOLOGY

Macroscopic changes: prolonged maintenance of arrhythmia running with high incidence of heart rate leads primarily to the progressive impairment of systolic function in both the left and right ventricle (3-5). Ventricular filling is due to the inscreased pressure, the cardiac output is reduced and elevated systemic vascular resistance is observed. The lack of increase in left ventricular muscle mass is a characteristic for this disease. Contractility of the myocardium is reduced, while reducing its contractile reserve. The lack of left ventricular muscle hypertrophy causes enlargement and the advent of usually at least moderate mitral regurgitation. These changes are observed due to dilatation of the mitral ring, changes in left ventricular geometry and lack of the mitral leaflets coaptation. As a result of the deterioration of the function of the left atrium, the increase of existing most supraventricular arrhythmias is observed. Increase rigidity and impairment of left ventricular torsion affects both systolic and diastolic function. These changes are substrate in damage to skeletal proteins, extracellular matrix proteoglycan.

Microscopic changes: changes in the course of tachycardiomyopathy include primarily the loss of myocyte contraction, significant increase in myocyte length, the disruption of connections between the basement membrane and sarcolemma. This reduces the possibility of adequate transmission of stress in the wall of the heart muscle. In addition, the reduction in the number of T-tubules type L calcium channels and beta-adrenergic receptors takes place. This affects negatively the duration of action potential of myocardial cells, leading to the development of the incorrect activation and reduces the systolic dysfunction. After heart rate normalization some of microscopic changes recede much slower than macroscopic. This mainly applies to the focal fibrosis of the heart muscle. These areas become the arrhythmia substrate and relapse.

Prolonged tachycardia affects not only the systolic impairment but also interferes with the relaxation of the heart muscle (6). The heart muscle is in so-called partial relaxation. This is due to the sustained, disproportionately elevated calcium ions contained in the tanks of sarcolemma during diastole. It can be illustrated as the functionally spasmolytic contracture of the heart muscle. This phenomenon also affects indirectly reducing flows in the sub endocardial layers of the heart muscle due to lingering high end-diastolic pressure in the left ventricle. Ischemia and hypoxia can also be the result of an increased distance between capillary and myocytes. Ischemia probably corresponds to the myocardial hibernation phenomenon. After the suppression of the arrhythmia rapid improvement is observed.

Acceleration of the rhythm also causes depletion of myocardium in high-energy compounds adequately to the higher incidence of rhythm and duration of tachyarrhythmia. Oxidative stress can also be a significant factor responsible for the diminished systolic function during atrial fibrillation. Patients with the DD genotype of angiotensin converting enzyme are characterised by elevated level of this enzyme which is responsible for the accelerated changes in the length of cardio myocytes and increase volume of cardiac cavities and the end-diastolic pressure.

Taking into account morphological changes discussed above, the irregularity of neurohormonal are very close to the classic heart failure of the other substrate (7, 8). Typically activation of the renin-angiotensin-aldosterone system occur. Increased levels of angiotensin II and endothelin are observed. An elevated level of atrial natriuretic peptide is observed due to increased pressure in atria and reduced sodium expulsion. Its concentration decreases, with the progression of heart failure. The concentration of cerebral natriuretic factor is also higher. Aldosterone – the final component of the renin-angiotensin-aldosterone system – affects the progressive fibrosis and enhances the arrhythmias and conduction in the damaged heart muscle. Expression of beta-adrenoreceptors on the surface of the cells of the myocardium is significantly reduced.

It should be noted that right ventricle reacts a little differently on the load generated by prolonged arrhythmias. Both the mass and the possibility of contraction by myocytes are increased.

DIAGNOSIS

Currently there is no strictly defined, clear criteria for diagnosis of tachycardiomyopathy. Arrhythmias

present in the patient with newly diagnosed heart failure running with a fast rhythm of the ventricles, not controlled in an appropriate manner, with a frequency of over 100/min should induce to consider this entity as the underlying disease. Coronary heart disease should be ruled out. The diagnosis of tachyarrhythmias can be considered in a situation:

- no other causes of non-ischemic events (such as: hypertension, alcohol intoxycation, drugs intoxycation, stress),
- lack of features of left ventricular hypertrophy,
- or the correct dimensions of the left ventricle (in particular its end-diastolic dimension < 5.5 cm),
- the return of normal left ventricular systolic function after restoring normal rhythm or by applying frequency control of rhythm in the period from 1 to 6 months after the start of therapy,
- rapid deterioration of heart muscle function caused by arrhythmia in patients previously treated due to tachycardiomyopathy,
- quick normalization of the previously elevated levels of natriuretic peptide after restoring the basic sinus rhythm.

Tachycardiomyopathy can reveal many months or even years after the onset of cardiac arrhythmias and cardiac arrhythmias which cause the tachycardiomyopathy may not be visible in the course of the investigation. On the other hand, heart failure secondary to rapid pacing rhythm reveals often in the first 24 hours after stimulation. The degree of progress of heart failure depends primarily on the time of duration of arrhythmia, and ventricular rate generated in the course of arrhythmia. Of great importance in the diagnosis of tachycardiomyopathy is echocardiography execution after the liquidation of the rhythm disturbances, especially paroxysmal ones.

The incidence of tachycardiomyopathy determined on the basis of the above criteria is quite wide depending on the type of arrhythmia. In the case of large extra systolic premature beats tachycardiomyopathy is diagnosed in about 10% of patients. In patients with atrioventricular nodal reentrant tachycardia in 20-50%, and in patients with atrial flutter in 25% of cases. The actual prevalence of tachycardiomyopathy in the population with atrial fibrillation is the most difficult to determine due to the additional load usually occurring in this group of patients. It is believed that patients at a younger age, with higher incidence of heart rate and chronic cardiac arrhythmias are more likely to develop tachycardiomyopathy. Recurrent arrhythmias with higher frequencies contribute to a greater extent to the development of tachycardiomyopathy than chronic arrhythmias with relatively slower rhythm of the ventricles. However the most important factor in the development of this disease, rhythm and artial fibrillation is an outstanding example for that.

Special attention should also be paid to patients who have been already diagnosed with tachycardiomyopathy, and recurrent arrhythmias (9). Relapse occurs very quickly and the deterioration of health status may even lead to death (10-12).

Atrial fibrillation is the most common disorder of the rhythm and frequency of its prevalence in the general population may reach as much as 1.5 percent. This type of arrhythmia causes systolic impairment as a result of too high a frequency of rhythm (typically above 120/min), loss of function of the atria, irregularity and, of course, the loss of atrioventricular synchrony (13). A minute cardiac output is reduced by approximately 20%. In this situation, it seems highly desirable to restore as soon as possible the proper basic rhythms – sinus rhythm – or at least precise control of frequency of ventricular rate during atrial fibrillation. Previous clinical observations indicate possible worse prognosis in patients in whom it is impossible to maintain sinus rhythm. This phenomenon may be associated with the use of antiarrhythmic drugs, which may have proarrhythmic side effects. In this situation, the ablation can be a solution without adverse events of antiarrhythmic drugs (14). In most cases, the cause of induction of atrial fibrillation is a pathological impulse activity from the muscle of the pulmonary veins. Isolation of pulmonary veins in such cases can result in a complete cure of the patient, but in selected cases it can be necessary to perform more than one ablation treatment. Isolation of pulmonary veins in selected patients is beneficial compared to the ablation of the atrioventricular connection followed by synchronising pacing. Rhythm control in addition to ablation seems to be superior compared to single control of the incidence of rhythm. The special method of treatment is the rythm or rate control with atrio-ventricular node ablation followed by pacemaker implantation. Of course, this strategy leads to permanent atrial fibrillation and is reserved for older patients with multiple diseases. Right ventricle pacing influent on the previously impaired myocardial systolic function through the induction of interventricular and intraventricular asynchrony. Accordingly in such situations, it is desirable to perform cardiac resynchronizing pacing. Its application in selected cases significantly reduces mortality.

Tachycardiomyopathy can also develop among patients with other disorders of the supraventricular rhythm. Particularly, atrial flutter and paroxysmal and permanent supraventricular tachycardia. Ablation significantly improves the prognosis for these patients, especially in cases when sources of the arrhythmia are located within the atria or pulmonary veins. The effectiveness of ablation is very high and reaches 97%.

Ventricular tachycardia and ventricular premature beats with similar QRS morphology may also contribute to the formation of tachycardiomyopathy (15, 16). This applies to patients without prior structural abnormality of the heart muscle and idiopathic ventricular tachycardia originated from right and left ventricular outflow tract. In these cases, the most effective solution is ablation. The similar QRS morphology, male, younger age and incessant tachycardias are the main risk factors for

the development of tachycardiomyopathy. Longer observation of these patients indicates the need to search for them the other causes of arrhythmias. The number of premature beats seems to correlate with severity of left ventricular systolic dysfunction. One-third of the patients with more than 10 000 ventricular premature beats per day were at risk of tachycardiomyopathy development. The established reasons for tachycardiomyopathy in patients with ventricular arrhythmias are: high frequency heart rate, premature beats with short feedback time with longer extrapotentialization which leads to the accumulation of intracellular calcium and increase in oxygen consumption. QRS widening, presence retrograde P waves and subepicardial-originated ventricular premature beats indicate a higher risk of developing heart failure among patients with tachycardiomyopathy. In the case of this type of arrhythmias ablation seems to be an optimal treatment although pharmaceuticals such as beta-blockers, amiodarone and others can treat and prevent the development of heart failure (17).

Tachycardiomyopathy, caused by persistent supraventricular or ventricular tachyarrhythmia, is a seldom but potentially curable form of dilated cardiomyopathy. Over the last years new risk and etiology factors, as well as recent advances in ablation treatment have been described. Because there are no specific markers available to precise diagnose tachycardiomyopathy, clinical features still remains the only available tool to diagnose. It should be noticed that all patients with tachyarrhythmia and heart failure are at risk of tachycardiomyopathy. Clinical observations suggest also that the maintenance of sinus rhythm or heart rate control is crucial in the prevention and treatment of tachycardiomyopathy. Future research need to be directed towards risk factors, diagnostic tools and new methods of treatment.

BIBLIOGRAPHY

1. Shinbane JS, Wood HAS, Jensen DN et al.: Tachycardia-induced cardiomyopathy: a review of animal models and clinical studies. J Am Coll Cardiol 1997 Mar 15; 29(4): 709-715.
2. Khasnis A, Jongnarangsin K, Veerareddy S et al.: Tachycardia-induced cardiomyopathy: a review of literature. Pacing Clin Electrophysiol 2005 Jul; 28(7): 710-721.
3. Gupta S, Figueredo VM: Tachycardia mediated cardiomyopathy: pathophysiology, mechanisms, clinical features and management. Int J Cardiol 2014 Mar 1; 172(1): 40-46.
4. Noszczyk-Nowak A, Skoczyński P, Gajek J: Tachycardiomyopathy in human and animals – patophysiology, treatment and prognosis. ADV Clin Exp Med 2010; 19(2): 245-249.
5. Mohamed HA: Tachycardia-induced cardiomyopathy (rachycardiomyopathy). Libyan J Med 2007 Mar 1; 2(1): 26-29.
6. De Selby, Palmer BM, LeWinter MM, Meyer M: Tachycardia-induced diastolic dysfunction and resting tone in myocardium from patients with a normal ejection fraction. J Am Coll Cardiol 2011 Jul 5; 58(2): 147-154.
7. Morris Robinson PD, Channer KS: Reversible heart failure: toxins, tachycardiomyopathy and mitochondrial abnormalities. Postgrad Level Med J 2012 Dec; 88(1046): 706-712.
8. Penela D, Van Huls Van Taxis C, Aguinaga L et al.: Neurohormonal, structural, and functional recovery pattern after premature ventricular complex ablation is independent of structural heart disease status in patients with depressed left ventricular ejection fraction: a prospective multicenter study. J Am Coll Cardiol 2013 Sep 24; 62(13): 1195-1202.
9. Dandamudi G, Rampurwala Mahenthiran J, Miller JM, Das MK: Persistent left ventricular dilatation in tachycardia-induced cardiomyopathy patients after appropriate treatment and normalization of ejection fraction. Heart Rhythm 2008 Aug; 5(8): 1111-1114.
10. Taillandier, S Brunet Bernard A, Lallemand B et al.: Prognosis in patients hospitalized with permanent and nonpermanent atrial fibrillation in heart failure. Am J Cardiol 2014 Apr 1; 113(7): 1189-1195.
11. Curtis AB: Practice implications of the Atrial Fibrillation Guidelines. Am J Cardiol 2013 Jun 1; 111(11): 1660-1670.
12. Nerheim P, Birger-Botkin S, Piracha L, Olshansky B: Heart failure and sudden death in patients with tachycardia-induced cardiomyopathy and recurrent tachycardia. Circulation 2004 Jul; 110(3): 247-252.
13. Hasdemir C, Simsek E, Yuksel A: Premature atrial contraction-induced cardiomyopathy. Europace 2013 Dec; 15(12): 1790.
14. Medi C, Kalman JM, Haqqani H et al.: Tachycardia mediated cardiomyopathy-secondary to focal atrial tachycardia: long-term outcome after via Stereokiller ablation. J Am Coll Cardiol 2009 May 12; 53(19): 1791-1797.
15. Yarlagadda RK, Iwai S, Stein KM et al.: Simply stated of cardiomyopathy in patients with repetitive monomorphic ventricular ectopy originating from the right ventricular outflow tract. Circulation 2005 Aug 23; 112(8): 1092-1097.
16. Hasdemir C, Yavuzgil O, Ulucan C et al.: Tachycardia-induced cardiomyopathy in patients with idiopathic ventricular arrhythmias: the incidence, clinical and electrophysiologic characteristics, and the predictors. J Cardiovasc Electrophysiol 2011 Jun; 22(6): 663-668.
17. Bogun F, Crawford T, Reich S et al.: Radiofrequency ablation of frequent ventricular premature idiopathic, complexes: comparison with a control group without intervention. Heart Rhythm 2007 Jul; 4(7): 863-867.

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