https://doi.org/10.35336/VA-2022-4-08

DIAGNOSIS AND TREATMENT TACHYCARDIA-INDUCED CARDIOMYOPATHY: CASE REPORT

V.I.Steklov[1], M.B.Patsenko[2], A.V.Demyanenko[1], M.V.Lipskaya[1], S.O.Lependin[1], F.G.Rzayev[3]

[1]"Central Military Clinical Hospital named after P.V.Mandryka", Ministry of Defense of Russia, Moscow, 8A Bolsyaha Olenya str; [2]Main Military Medical Directorate of the Russian Ministry of Defense, Moscow, 14 Znamenka str; [3]City Clinic Hospital named after I.V.Davidovsky, Russia, Moscow, 11 Yauzkaya str.

The article highlights current issue of the etiology of cardiomyopathy resulting from persistent tachycardia. Clinical studies devoted to the diagnosis and treatment of tachycardia-induced cardiomyopathy and the criteria for its diagnosis are presented. The article presents a clinical case report of a 48-year-old patient who developed cardiomyopathy against the background of long-term persistent tachysystolic atrial fibrillation. After the restoration of the sinus rhythm against the background of optimal drug therapy, the clinical signs of cardiomyopathy regressed, the size of the heart chambers and the contractile function of the myocardium of the left ventricle of the heart returned to normal.

Key words: tachycardia-induced cardiomyopathy; heart failure; tachysystolic atrial fibrillation; left ventricular ejection fraction

Conflict of Interests: nothing to declare.
Funding: none.
Received: 16.03.2022 **Revision received:** 19.07.2022 **Accepted:** 27.07.2022
Corresponding author: Vladimir Steklov, E-mail: vsteklov@yandex.ru

V.I.Steklov - ORCID ID 0000-0001-6790-9401, M.B.Patsenko - ORCID ID 0000-0001-8391-1691, A.V.Demyanenko - ORCID ID 0000-0001-6099-8317, M.V.Lipskaya - ORCID ID 0000-0003-0630-928X, S.O.Lependin - ORCID ID 0000-0001-8670-8787, F.G.Rzayev - ORCID ID 0000-0002-4094-7771

For citation: Steklov VI, Patsenko MB, Demyanenko, Lipskaya MV, Lependin SO, Rzayev FG. Diagnosis and treatment tachycardia-induced cardiomyopathy: case report. *Journal of Arrhythmology.* 2022;29(4): 53-60. https://doi.org/10.35336/VA-2022-4-08.

It is known that one of the clinical symptoms of chronic heart failure (CHF) and cardiomyopathy (CMP) is various cardiac arrhythmias. Both supraventricular and ventricular tachyarrhythmias can cause or be a triggering factor for the development of non-ischaemic tachy-induced cardiomyopathy (TI CMP) [1-4]. This occurs most often in patients with late-diagnosed tachyarrhythmia or due to its inadequate treatment. In these patients, there is dilation of the ventricle, followed by the development of TI CMP [5-7]. Therefore, timely diagnosis and appropriate treatment are necessary to prevent the development of this dangerous complication of chronic tachyarrhythmias.

Definition of tachy-induced cardiomyopathy, etiopathogenesis and prevalence

The concept of TI CMP is based on the fact that prolonged tachyarrhythmia can lead to reversible disturbances in left ventricular (LV) function and was proposed by scientists A.M.Gossage and J.B.Hicks as early as 1913 [8]. Later, E. Phillips and S.A.Levine (1949) discovered the connection between reversible LV dysfunction and tachyarrhythmia [9], and in 1962 G.H.Whipple experimentally demonstrated in animal models that rapid and sustained atrial pacing leads to a significant decrease in ejection fraction (EF) and dilatation of ventricles [10].

The simplest and most comprehensive definition of TI CMP is the formulation proposed by A.Okada in 2016: a reversible form of systolic ventricular dysfunction caused mainly by a rapid ventricular response in tachyarrhythmias [11]. In the medical literature, there are different definitions of TI CMP, significant differences in terminology, definitions and criteria prevent a general understanding and early diagnosis of the disease, as there are no uniform criteria and expert recommendations for diagnosis and clinical management. For this reason, TI CMP is a disease with an understudied prevalence.

The exact mechanisms responsible for the contractile dysfunction and structural changes in tachyarrhythmia are currently unknown. Researchers have proposed several hypotheses to explain the mechanisms of the TI CMP development: 1) depletion of myocardial energy reserves and impaired energy utilisation; 2) myocardial ischaemia due to tachycardia; 3) impaired regulation of calcium homeostasis; 4) oxidative stress and extracellular matrix remodelling; 5) inflammatory process; 6) neurohormonal activation; 7) genetic causes of myocardial dysfunction (DD homozygous polymorphism of the angiotensin-converting enzyme gene) [7, 12].

The basis of TI CMP is dilatation of the cardiac cavity and ventricular systolic dysfunction (predominantly of the LV), which develops against a background of chronic tachyarrhythmia. Thus, the following tachyarrhythmias may lead to the development of TI CMP: atrial fibrillation (AF) and/or atrial flutter, atrial tachycardia (AT), recurrent AV reentry tachycardia, paced tachycardia, high-frequency atrial pacing, persistent rapid ventricular pacing, permanent right ventricular pacing, ventricular tachycardia

and frequent ventricular extrasystoles (VE) [1-4, 13, 14]. According to the literature, AF with a poorly controlled ventricular response is the most common cause of TI CMP in adults. Some studies have shown a 25-75% association rate between AF and advanced LV dysfunction [4,14,15[. An analysis of the Framingham study confirms the association of CHF and AF: of 1733 patients with newly-onset AF, 37% had CHF, and of 1166 patients with newly-onset CHF, 57% had AF [4].

In the persistent form of atrial flutter, TI CMP is diagnosed in 15-25% of patients [3, 16]. TI CMP occurs in 10% of patients with AT, and with persistent AT it may occur in one in three patients. In continuous recurrent AV recurrent tachycardia, the range of occurrence of TI CMP is from 20% to 50% [13].

In patients with frequent VE lasting more than 15% of daily rhythm and persistent ventricular tachycardia, negative myocardial remodeling may develop, followed by formation of TI CMP in about 7-30% of patients [2, 17, 18]. The reason why, in some very similar situations, some patients develop ventricular systolic dysfunction during tachyarrhythmia and others do not, or they occur much later, is not entirely clear [1, 19].

The diagnosis of TI CMP is complicated by the lack of pathognomonic morphological and specific functional features of the myocardium. The key diagnostic task is differentiation of TI CMP and dilated cardiomyopathy (DCMP) in the presence of tachyarrhythmia. However, it is difficult to determine whether heart failure is a cause or a consequence of tachyarrhythmia.

The diagnosis is confirmed when the size of the heart cavities and the contractile function LV have normalized against a background of adequate heart rate control or recovery of sinus rhythm. After arrhythmia conversion, recovery of LV function is observed within 6-8 weeks. At the same time, the risk of sudden cardiac death is increased, especially with recurrent arrhythmias or when CMP has a mixed etiology, including coronary artery disease [15, 20].

Clinical picture, diagnosis

CMP induced by arrhythmia have a wide range of nonspecific clinical manifestations. For this reason, this disease was long considered a clinical manifestation of CHF and CMP. In the early stages of this pathological process, some patients experience severe haemodynamic disturbances, including cardiogenic shock. Elimination of the tachyarrhythmia leads to restoration of the function of LV, and if there is no recurrence of arrhythmia, the prognosis in these patients is favorable [21].

Major clinical symptoms include palpitations (29%), heart failure of functional classes III-IV (47%), and syncope/pre-syncope (12%). At the same time, some patients may have no symptoms. Patients with TI CMP have an increased risk of sudden cardiac death. Despite the treatment and resolution of CMP, sudden cardiac death is registered in 8-12% of cases [18, 20].

The most common diagnostic criteria for TI CMP are those of R.Gopinathannair, et al. (2009), distinguished by their accessibility and ease of use [6]:

- non-sinus rhythm with heart rate (HR) >100 bpm;
- frequent VE lasting more than 15% of the daily rhythm;
- exclusion of other causes of CMP;
- complete or partial normalization of LV function after elimination of tachyarrhythmia, restoration of sinus rhythm or achievement of target HR;
- rapid decrease of LV EF after tachycardia recurrence in a patient with restored LV after a previous episode of tachyarrhythmia.

Therefore, the diagnosis TI CMP is usually made retrospectively when there is a steady increase in EF against a background of rhythm recovery or when heart rate control is achieved [22, 21]. The time from onset of arrhythmia to clinical presentation or deterioration of LV function varies widely and depends on the duration of persistent arrhythmia, concomitant structural heart disease and the age of the patient. Clinical studies have shown that the time between onset of arrhythmia symptoms and the development of TI CMP varies between 3 and 120 days [19].

Management of patients with TI CMP

The characteristic feature of TI CMP is the reversibility of the reduced systolic cardiac function after elimination of the tachyarrhythmia. Therefore, the main treatment should aim to suppress the tachycardia responsible for the development of TI CMP (Table 1) with antiarrhythmic drugs and/or catheter ablation, while the treatment of pacemaker-mediated tachycardia requires its reprogramming [18, 19]. In case of hemodynamically significant tachycardia, emergency electropulse therapy is indicated.

To reverse TI CMP remodelling, initial treatment should be comprehensive and include medications to treat

Table 1.

Methods of treatment of TI CMP depending on the type of arrhythmia.

Type of arrhythmia	Method of treatment
Sinus tachycardia/ thyrotoxicosis	Beta-blockers/sinus node lf-channels inhibitors + treatment of underlying disease
Atrial fibrillation, tachysystole	Sinus rhythm conversion (catheter ablation of pulmonary vena cava + AAT) or frequency control (AAT or catheter ablation of AVS + pacemaker/BVS)
Atrial flutter	Catheter ablation
Atrial tachycardia	Catheter ablation
AV reciprocal tachycardia, including AV nodal reciprocal tachycardia	Catheter ablation
Frequent ventricular extrasystoles, sustained ventricular tachycardia	Catheter ablation/AAT
Pacemaker-mediated tachycardia	Reprogramming the pacemaker

Note: AAT - antiarrhythmic therapy; AVS - atrioventricular connection; BVS - biventricular stimulation.

heart failure and LV systolic dysfunction (beta blockers, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, diuretics, and aldosterone blockers).

Treatment of persistent tachysystolic AF includes the use of heart rate-control agents and, if the latter are ineffective, the implantation of a permanent pacemaker/biventricular stimulation followed by catheter ablation of the AV junction to create an artificial AV block. Catheter ablation of AV node in combination with cardiac resynchronization therapy reduced mortality in patients with persistent AF and narrow QRS complex (≤110 ms), who were hospitalized for CHF, compared with pharmacological control of ventricular rate, regardless of their initial EF [23, 24].

We present below a clinical case showing TI CMP in a 48-year-old patient with long-standing persistent tachysystolic AF, with complete recovery of LV contractile function after conversion to sinus rhythm.

On December 16, 2020, Patient B. of 1972 year of birth was admitted to the Interventional Arrhythmia and Pacing Department with complaints of irregular rapid heartbeat and decreased tolerance to physical exertion. From the medical history it is known that for 15 years he has been regularly disturbed by short attacks (lasting up to 30 seconds) of palpitations during intense physical activity. No cardiac rhythm abnormalities were registered on the surface ECG. Deterioration in the form of heart palpitations, irregular heartbeat, and decreased exercise tolerance occurred in September 2019. The ECG first recorded tachysystolic AF; and on echocardiographic examination (Echo) LV EF was preserved at 60%. After discharge from hospital, the patient did not take the prescribed therapy and did not seek medical help. He did not comply with the hospital's recommendation for re-hospitalisation for restoration of sinus rhythm after optimal anticoagulation with oral anticoagulants and performance of a transoesophageal Echo to rule out thrombotic inclusions in the heart cavities. The patient is consulted by a cardiologist one month before the actual hospital admission. After 3 weeks of therapy with anticoagulants and beta-blockers, the patient was admitted for inpatient treatment.

On re-admission to hospital, the patient's condition was considered satisfactory. Skin of normal color and moisture. There are no peripheral edemas. Oxygen saturation in atmospheric air is 98%. Pulse averaged 98 per min, irregular, weakly filled. A pulse deficit of 10 beats per minute. Blood pressure (BP) 120/70 mmHg. Percrutorally, the heart is dilated to the left 1 cm outward from the left median clavicular line. Heart tones muffled, arrhythmic, 1 tone attenuated at the apex. Frequency of breathing movements - 16 per minute. There is no percussion sound dulling over the lung fields, breathing is vesicular. The abdomen is soft, not enlarged. The liver is not enlarged, the spleen is not palpable. ECG on admission - AF, tachysystole with HR 117 per min (Fig. 1a).

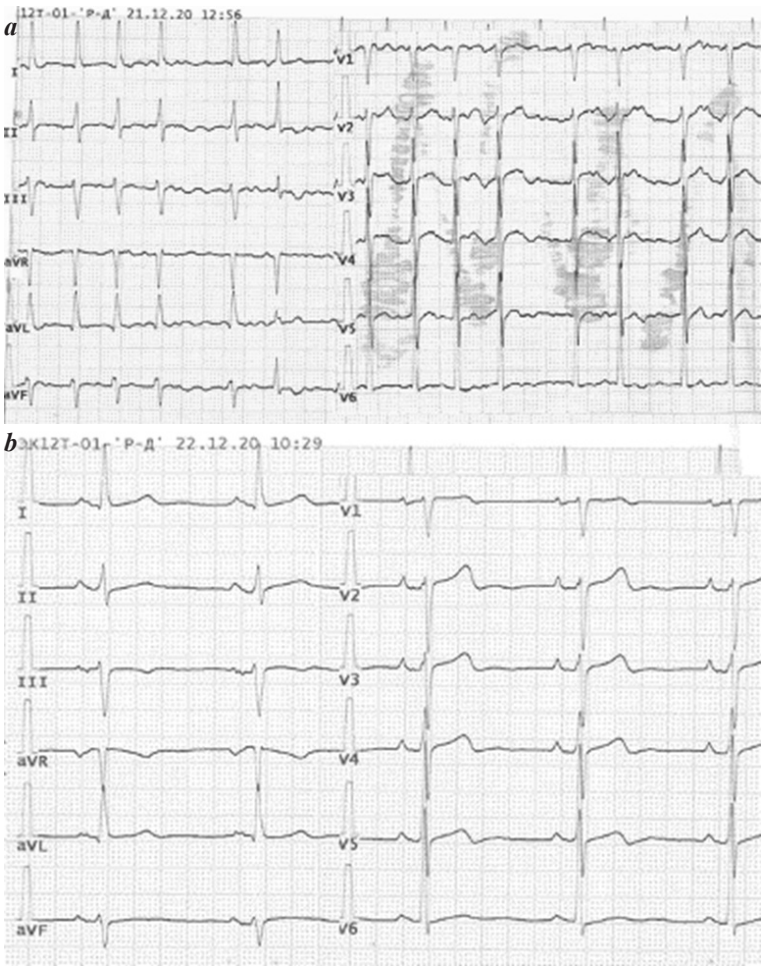

Fig. 1. Electrocardiogram of the patient: a - from December 21, 2020 (before cardioversion - tachysystolic atrial fibrillation with heart rate 117 bpm, b - from December 22, 2020 (after cardioversion - sinus bradycardia with heart rate 52 bpm).

Transthoracic Echo on December 17, 2020 revealed changes characteristic of DCMP: significant LV dilatation (LV end-systolic dimension = 65 mm, LV end-diastolic dimension (EDD) = 76 mm, EDD index = 40.9 ml/m2, LV end-systolic volume = 210 ml, LV end-diastolic volume (EDV) = 307 ml, EDV index =165 ml/m2, stroke volume = 98 ml, LV myocardial mass index = 163 g/m2) with significant decrease in LV systolic function (EF = 21%), moderate right ventricular (RV) dilatation, significant left atrial (LA) dilatation (LA = 70 mm, LA volume = 105 ml, LA volume index = 56 ml/m2), significant right atrial dilation. The difference between LV and RV preload periods was 35 ms, LV end-diastolic pressure was 18 mmHg. A slight decrease in LV longitudinal strain and RV systolic function (GLS = 17%, TAPSE = 14 mm) was detected. Thickening of aortic walls, aortic and mitral valve cusps. No aortic regurgitation was detected. Severe mitral regurgitation, moderate tricuspidal regurgitation. Signs of moderate pulmonary hypertension. Calculated systolic pulmonary artery pressure = 31 mmHg. Pericardium - the sheets are thickened (Fig. 2a).

On laboratory examination, biochemical, blood and urine tests were within normal limits. Thyroid hormone levels are within normal limits. C-reactive protein, anti-streptolysin-O, circulating immunocomplexes, rheumatoid factor - within reference values.

Based on the patient's complaints, medical history, and investigations, the following clinical diagnosis was made: first-onset AF, long-persistent form, tachysystolic variant. EHRA 2a cm. CHA_2DS_2-Vasc - 1. HAS-BLED - 0. Tachy-induced cardiomyopathy. CHF 2a, II FC NYHA.

The patient was prescribed therapy with amiodarone, eplerenone, continued therapy with dabigatran 150 mg b.i.d. After exclusion of thrombotic inclusions by transesophageal Echo and correction of electrolyte disturbances, electrical synchronised cardioversion with a discharge capacity of 150 J was performed on December 22, 2020, with restoration of sinus rhythm with a heart rate of 52/min (Fig. 1b).

After restoring sinus rhythm, the patient noted an improvement in his general condition in the form of increased exercise tolerance and the disappearance of heart palpitations. The control Echo revealed a decrease in the size of heart cavities, an increase in EF from 21% to 31% (Fig. 2b).

The patient was discharged on December 29, 2020, with recommendations to continue therapy with dabigatran 150 mg b.i.d., amiodarone 200 mg 5 days a week, eplerenone 25 mg in the morning, panangin 1 tab 3 times a day for 2 weeks and perindopril 2.5 mg in the evening.

The patient had no complaints during follow-up examinations on March 05, 2021 and July 27, 2021. Condition is satisfactory. Pulse is rhythmic, 64-68 per min, of satisfactory filling. BP 110/70 mm Hg. Clear, rhythmic heart tones. Breathing is vesicular. The patient fully complies with the functional responsibilities assigned to him. ECG showed a correct sinus rhythm with heart rate of 64-68 per min. Echo from March 05 and July 27, 2021 showed cardiac chambers within normal limits, with preserved LV EF of 54-60%. The dynamics of cardiac chamber size is shown in the graph (Fig. 3). After restoring sinus rhythm over a period of 2.5 months, the heart chambers have shrunk and the contractile function of the heart has been restored. Thus, the preservation of sinus rhythm resulted in complete recovery of myocardial contractility and performance capacity, meeting the criteria for a diagnosis of tachy-induced cardiomyopathy.

Seven months later, a magnetic resonance imaging (MRI) scan of the heart with contrast was performed to rule out myocardial structural abnormalities, which did not reveal any pathological changes. No delayed myocardial contrast indicated the absence of post-inflammatory fibrosis and post-ischaemic myocardial scarring, which also confirms the predominantly electrical nature of TI CMP (Fig. 4).

DISCUSSION

Currently, the issue of differential diagnosis of TI CMP with primary dilated, ischemic, alcoholic, dyshormonal CMP, sarcoidosis, diffuse myocarditis remains relevant. The correct diagnosis determines the further tactics of the patient's care. During the examination we ruled out the following diseases:

- idiopathic DCMP - retrospectively, after normalization of heart chamber sizes and EF;
- ischemic CMP - no history of previous focal changes in the myocardium, the clinic of angina or its equivalents, and long-standing arterial hypertension;
- alcoholic CMP - no history of alcohol abuse;
- thyrotoxic CMP - normal levels of thyroid hormones;
- sarcoidosis of heart - normal size of interventricular septum thickness (its basal section), absence of alternation of affected and normokinetic segments in LV, LV aneurysm, as well as local intracardiac masses on Ech, absence on ECG of signs of grade II-III AV block, pathological Q wave, ventricular arrhythmias, bundle branch block;
- diffuse myocarditis - no history of previous acute myocarditis, benign course of the disease, as well as absence of ventricular arrhythmias and elevation of cardiospecific enzymes in blood tests.

Thus, the diagnosis of TI CMP in our patient at admission to the clinic was legitimate. The strategic goal of treatment was 'rhythm control tactics'. In view of the first discovered AF, which was complicated by TI CMP, and the young age of the patient, it was therefore decided, after ruling out thrombotic inclusions in the heart cavities, to convert sinus rhythm immediately. This led to normalization of Echo parameters and regression of heart failure.

However, since the patient's hospitalization coincided with another wave of new coronavirus infections, it was not possible to perform coronarography, computed tomography and cardiac MRI prior to sinus rhythm conversion. Also at that time, we encountered significant difficulties in performing blood tests for various biomarkers.

Persistent atrial and ventricular tachyarrhythmias are common in patients with CMP and CHF. They should be considered as potential risk factors for the development of CMP and CHF [2-4, 13]. Today, it is not entirely clear why some patients with prolonged tachysystole (HR >100 beats per minute) or with frequent VE (daily VE frequency ≥10-15%) do not develop TI CMP [6, 7, 16]. Nevertheless, such patients should be referred to a high-risk group, requiring dynamic monitoring with ECG and transthoracic Echo every 6-12 months or less when symptoms of CHF appear [19, 25, 26]. We presented a clinical case of a patient with a long-period persistent tachyarrhythmia with HR over 100 bpm, which resulted in TI CMP.

In our work, we used the algorithm for diagnosing TI CMP developed by R. Gopinathannair [6] due to its practical accessibility. Other algorithms are also described

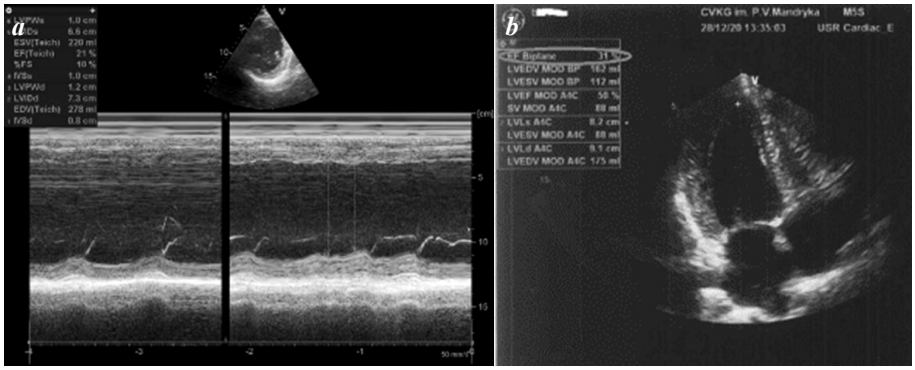

Fig. 2. Echocardiography of a patient: a - on admission to the hospital, b - after restoration of sinus rhythm.

in the literature. Thus, in 2014 S.Gupta, et al. [25] have developed an algorithm for diagnosing left ventricular dysfunction occurring for the first time against the background of chronic or recurrent tachycardia with HR over 100 beats per minute when other causes of CMP are excluded. This algorithm, as well as the algorithm developed by R. Gopinathannair et al. (2009) [6] consists of 5 items, which are listed below:

- no other causes of non-ischemic CMP (e.g., hypertension, alcohol or drug use, etc.);
- absence of LV hypertrophy;
- relatively normal LV dimensions (LV EDD < 5.5 cm);
- recovery of left ventricular function after tachycardia resolution (HR control, cardioversion or catheter ablation) within one to six months;
- rapid decrease of LV EF after tachycardia recurrence in patients with a history of LV EF restoration episode when normosystole is achieved or sinus rhythm is restored.

According to the algorithms presented in our clinical example, the following clinical signs were the significant factors that allowed us to make the diagnosis TI CMP:

- exclusion of other forms of CMP (ischemic and non-ischemic);
- non-sinus rhythm with HR > 100 bpm;
- recovery of left ventricular function after tachycardia resolution within 2.5 months.

Regarding the point of relatively normal LV size (LV EDD < 5.5 cm). The long-term persistence (more than 1 year) of tachysystole in our patient appears to have led to a marked dilatation of the heart chambers with a significant reduction in inotropic function. Regarding the rapid decrease in LV EF after a relapse - the patient has not had a recurrent arrhythmia during the follow-up, so we cannot judge this today.

The current clinical challenge is to diagnose TI CMP as a potentially reversible cause of CHF with a DCMP phenotype in patients with persistent tachyarrhythmia. The presence of a dissociation between the degree of reduction in LV EF and LV EDD is likely in these patients. The study comparing the group of patients with TI CMP with the group of patients with idiopathic DCMP revealed differences in echocardiographic indices. Thus, patients with TI CMP usually have lower EDD LV end-systolic size, and myocardial mass index compared to patients with idiopathic DCMP and concomitant tachycardia. In this study, EDD was an independent factor of TI CMP: LV EDD less than 61 mm enables prediction of this disease with sensitivity of 100% and specificity of 71.4%. And in patients with EF of less than 30%, LV EDD of less than 66 mm was a prognostic criterion, with a sensitivity of 100% and specificity of 83.4%. Our patient developed a marked reduction in EF (21%) with a significant increase in LV EDD (76 mm) against a background of long persistent tachysystole, which is not very characteristic of TI CMP. The most important confirmation of TI CMP in our patient was the rapid recovery of inotropic cardiac function (after just one week of sinus rhythm, EF had increased to 31%) with a reduction in heart chamber size. And patients with idiopathic DCMP show an increase in EF of only up to 5%. In addition, patients with TI CMP were found to have a lower functional class of CHF (according to NYHA) and less severe symptoms of CHF. In our patient, clinical symptoms were also scarce and the functional class of CHF was low [26].

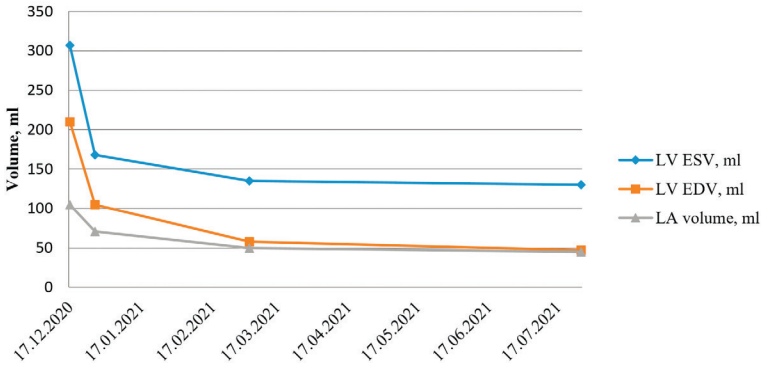

Fig. 3. Dynamics of cardiac chamber sizes before and after sinus rhythm recovery.

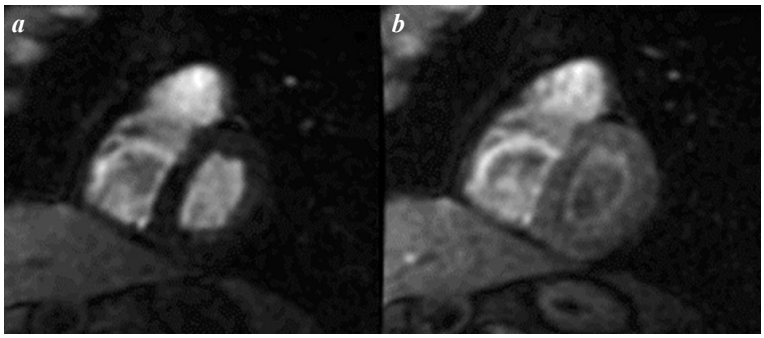

Fig. 4. Magnetic resonance imaging with contrast at 6 months after restoration of sinus rhythm: a - contrast in the heart cavities, b - uniform distribution of contrast in the left ventricular tissues with no signs of contrast accumulation (cross-sectional view of the basal areas of the heart).

The absence of delayed contrast in the LV myocardium on cardiac MRI may also indicate a predominantly 'electrical' genesis of CMP, rather than its inflammatory genesis [27,28].

This case study demonstrates the benefit of a rhythm control strategy in a patient with persistent AF, especially in the first-onset form. There is currently no consensus among practitioners on the management of patients with first-onset persistent and long-onset AF. Chamber dilatation and reduced LV EF are often interpreted as a relative contraindication to restoring sinus rhythm because of the low chance of preserving it. Often 'promising' patients with regard to recovery and retention of 'low effort' sinus rhythm are classified by the treating physicians as 'chronic AF' without any attempt at rhythm conversion. The choice of treatment depends entirely on the opinion of the attending physician, who often simply has no officially recognized benchmark by which to identify those in need of sinus rhythm restoration. In this situation, the physician chooses the 'heart rate control' tactic based on data from well-known studies such as AFFIRM, RACE, PIAF, STAFF, which showed no difference between alterna-

tive management tactics. However, the results of recent multicenter studies confirming the advantage of rhythm control tactics are already known. For example, results from the EAST-AFNET multicenter study (135 centrers, 2789 patients), published in 2020, demonstrated significant benefits of an early rhythm control strategy: early rhythm conversion with antiarrhythmic drugs or catheter ablation of AF was associated with a lower risk of adverse cardiovascular outcomes than rhythm rate control with management of symptoms associated with AF and cardiovascular disease [29]. In patients with initially diagnosed AF, restoration of sinus rhythm is advisable if there are no contraindications [5].

Given the rather short timeframe for the development of TI CMP, tachysystole can lead to severe complications and disability. This group of patients will already require new, more costly treatments to prevent sudden cardiac death including implantation of defibrillators and cardiac resynchronisation devices, especially when antiarrhythmic drugs or catheter ablation are not feasible or unsuccessful. Late diagnosis of TI CMP thus leads to increased healthcare costs, while its timely diagnosis and treatment will avoid this [4, 6, 20-22].

CONCLUSION

1. Persistent forms of atrial or ventricular tachycardia are potential risk factors for TI CMP.
2. The main screening methods for diagnosing TI CMP are ambulatory ECG monitoring and transthoracic Echo.
3. Restoration of myocardial contractile function after tachyarrhythmia elimination not only confirms the diagnosis of TI CMP, but also significantly improves the outcome of the disease.
4. In patients with persistent and long-onset AF, restoration of sinus rhythm, regardless of the duration of the paroxysm, is advisable if there are no contraindications.

REFERENCES

1. Bockeria LA, Shengeliya LD. Changes in the heart associated with atrial fibrillation. Part I. Cardiopathy of atrial fibrillation: new dilemmas and old problems. *An Arrhythmology*. 2016;3(2): 138-47 (In Russ.). https://doi.org/10.15275/annaritmol.2016.3.2.
2. Hasdemir C, Ulucan C, Yavuzgil O, et al. Tachycardia-induced cardiomyopathy in patients with idiopathic ventricular arrhythmias: the incidence, clinical and electrophysiologic characteristics, and the predictors. *J Cardiovasc Electrophysiol.* 2011;22(6): 663-8. https://doi.org/10.1111/j.1540-8167.2010.01986.x.
3. Pizzale S, Lemery R, Green MS, et al. Frequency and predictors of tachycardia induced cardiomyopathy in patients with persistent atrial flutter. *Can J Cardiol*. 2009;25(8): 469-72. https://doi.org/10.1016/s0828-282x(09)70119-9.
4. Santhanakrishnan R, Wang N, Larson MG, et al. Atrial fibrillation begets heart failure and vice versa: temporal associations and differences in preserved versus reduced ejection fraction. *Circulation*. 2016;133(5): 484-92. https://doi.org/10.1161/01.CIRCULATIONAHA.115.018614.
5. Steklov VI, Morozov DA, Lependin SO, et al. Newly diagnosed atrial fibrillation: choice of tactics. IX Russian congress of arrhythmologists. Saint Petersburg-2021. Theses. 119-20. (In Russ.).
6. Gopinathannair R, Sullivan R, Olshansky B. Tachycardia-mediated cardiomyopathy: recognition and management. *Curr Heart Fail Rep.* 2009;6(4): 257-64. https://doi.org/10.1007/s11897-009-0035-3.
7. Mueller Karin AL, Heinzmann D, Klingel K, et al. Histopathological and Immunological Characteristics of Tachycardia-Induced Cardiomyopathy. J Am Coll Cardiol. 2017;69(17): 2160-72. https://doi.org/10.1016/j.jacc.2017.02.049
8. Gossage AM, Hicks JB. On auricular fibrillation. QJM An International Journal of Medicine. 1913;6: 435-440.
9. Phillips E, Levine SA. Auricular fibrillation without other evidence of heart disease. *Am J Med*. 1949;7(4): 478-89. https://doi.org/10.1016/0002-9343(49)90397-6.
10. Whipple GH. Reversible congestive heart failure due to chronic rapid stimulation of the normal heart. *In Proceedings of the New England Cardiovascular Society*. 1962;20: 39-40.
11. Okada A, Nakajima I, Morita Y, et al. Diagnostic value of right ventricular dysfunction in tachycardia-induced cardiomyopathy using cardiac magnetic resonance imaging. *Circ J*. 2016;80: 2141-2148.
12. Elliott P. Defining Tachycardia-Induced Cardiomyopathy: Life in the fast lane. *J Am Coll Cardiol*. 2017;69: 2173-2174.
13. Medi C, Kalman JM, Haqqani H, et al. Tachycardia-mediated cardiomyopathy secondary to focal atrial tachycardia: long-term outcome after catheter ablation. *J Am Coll Cardiol.* 2009; 53(19): 1791-7. https://doi.org/10.1016/j.jacc.2009.02.014.
14. Redfield MM, Kay GN, Jenkins LS, et al. Tachycardia-related cardiomyopathy: a common cause of ventricular dysfunction in patients with atrial fibrillation referred for atrioventricular ablation. *Mayo Clin Proc.* 2000;75(8): 790 - 795. https://doi.org/10.4065/75.8.790.
15. Ardashev AV, Belenkov YuN, Matsiukevich MCh, et al. Atrial Fibrillation and Mortality: Prognostic Factors and Direction of Prevention. *Kardiologiia*. 2021;61(2): 91-98. (In Russ.). https://doi.org/10.18087/cardio.2021.2.n1348.
16. Brembilla-Perrot B, Ferreira JP, Manenti V, et al. Predictors and prognostic significance of tachycardiomyopathy: insights from a cohort of 1269 patients undergoing atrial flutter ablation. *Eur J Heart Fail*. 2016;18(4): 394-401. https://doi.org/10.1002/ejhf.482.
17. Lee GK, Klarich KW, Grogan M, et al. Premature ventricular contraction-induced cardiomyopathy: a treatable condition. *Circ Arrhythm Electrophysiol.* 2012;5(1): 229-36. https://doi.org/10.1161/CIRCEP.111.963348.
18. Yokokawa M, Good E, Crawford T, et al. Recovery from left ventricular dysfunction after ablation of frequent premature ventricular complexes. *Heart Rhythm.* 2013;10(2): 172-5. https://doi.org/10.1016/j.hrthm.2012.10.011.
19. Watanabe H, Okamura K, Chinushi M, et al. Clinical characteristics, treatment, and outcome of tachycardia induced cardiomyopathy. *Int Heart J.* 2008;49(1): 39-47. https://doi.org/10.1536/ihj.49.39.
20. Nerheim P, Birger-Botkin S, et al. Heart failure and sudden death in patients with tachycardia induced

cardiomyopathy and recurrent tachycardia. *Circulation.* 2004;110: 247-52. https://doi.org/10.1161/01.CIR.0000135472.28234.
21. Huizar JF, Ellenbogen KA, Tan AY, et al. Arrhythmia-Induced Cardiomyopathy. *J Am Coll Cardiol*. 2019;73(18): 2328-44. https://doi.org/ 10.1016/j.jacc.2019.02.045.
22. Donghua Z, Jian P, Zhongbo X, et al. Reversal of cardiomyopathy in patients with congestive heart failure secondary to tachycardia. *J Interv Card Electrophysiol*. 2013;36(1): 27-32. https://doi.org/10.1007/s10840-012-9727-9.
23. Brignole M, Pentimalli F, Palmisano P, et al. AV junction ablation and cardiac resynchronization for patients with permanent atrial fibrillation and narrow QRS: the APAF-CRT mortality trial. *European Heart Journal.* 2021; ehab569. https://doi.org/10.1093/eurheartj/ehab569.
24. Brignole M, Pokushalov E, Pentimalli F, et al. A randomized controlled trial of atrioventricular junction ablation and cardiac resynchronization therapy in patients with permanent atrial fibrillation and narrow QRS. *European Heart Journal.* 2018;39: 3999-4008. https://doi.org/10.1093/eurheartj/ehy555.
25. Gupta S, Figueredo VM. Tachycardia mediated cardiomyopathy: pathophysiology, mechanisms, clinical features and management. *Int J Cardiol.* 2014;172(1): 40-6. https://doi.org/10.1016/j.ijcard.2013.12.180.
26. Jeong YH, Choi KJ, Song J-M, et al. Diagnostic Approach and Treatment Strategy in Tachycardia-induced *Cardiomyopathy. Clin Cardiol.* 2008;31(4): 172-8. https://doi.org/10.1002/clc.20161.
27. Stukalova OV, Gupalo EM, Chumachenko PV, et al. The value of cardiovascular magnetic resonance in myocarditis with different clinical presentation. *Ter Arkh.* 2019; 91(4): 28-36. (In Russ.). https://doi.org/10.26442/00403660.2019.04.000078
28. Prabhu S, Taylor AJ, Costello BT, et al. Catheter ablation versus medical rate control in atrial fibrillation and systolic dysfunction: The CAMERA-MRI study. *J Am Coll Cardiol*. 2017;70(16): 1949-61. https://doi.org/10.1016/j.jacc.2017.08.041.
29. Kirchhof P, Camm AJ, Goette A, et al. Early Rhythm-Control Therapy in Patients With Atrial Fibrillation. *N Engl J Med.* 2020;383: 1305-1316. https://doi.org/10.1056/NEJMoa2019422.