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Predictors of tachycardia-induced cardiomyopathy in patients with first-time decompensation of chronic heart failure with reduced left ventricular ejection fraction of nonischemic etiology and persistent atrial tachyarrhythmia

Aim	To identify possible predictors of tachycardia-induced cardiomyopathy (TICMP) in patients with newly developed decompensated chronic heart failure (CHF) of nonischemic origin with reduced left ventricular ejection fraction (LV EF) and with persistent atrial tachyarrhythmias.
Material and Methods	This study included 88 patients with newly developed decompensated CHF of nonischemic origin with reduced LV EF and persistent atrial tachyarrhythmias. Resting 12-lead electrocardiography (EGC) and transthoracic echocardiography (EchoCG) were performed upon admission and following the electrical impulse therapy for all patients. Also, 24-h ECG monitoring was performed to confirm sinus rhythm stability. After recovery of sinus rhythm, outpatient monitoring was performed for three months, including repeated EchoCG to evaluate the dynamics of heart chamber dimensions and LV EF.
Results	The patients were divided into two groups based on the increase in LV EF: 68 responders (TICMP patients with a LV EF increase by >10%) and 20 non-responders (patients with an increase in LV EF by <10% during 3 months following the sinus rhythm recovery). According to results of the baseline EchoCG, LV EF did not significantly differ in the two subgroups (TICMP, 40±8.3%, 18–50% and non-responders, 38.55±7.9%, 24–50%); moreover, the incidence of cases with LV EF <30% did not differ either (9 patients TICMP and 2 non-responders, p=1.0). TICMP patients compared to non-responders, had significantly smaller left atrial dimensions (4.53±1.14 (2–7) cm and 5.68±1.41 (4–8) cm, p=0.034; 80.8±28.9 (27–215) ml and 117.8±41.3 (46–230) ml, p=0.03, respectively) and left ventricular end-systolic volume (ESV) (67.7±33.1 (29–140) ml and 104.5±44.7 (26–172) ml, p=0.02, respectively). The effect of major EchoCG parameters on the probability of TICMP development was assessed by one-factor and multifactor regression analyses with adjustments for age and sex. The probability of TICMP increased with the following baseline EchoCG parameters: end-diastolic volume (EDV) <174 ml [odd ratio (OR), 0.115, 95% confidence interval (CI): 0.035–0.371], ESV <127 ml [OR, 0.034, 95% CI: 0.007–0.181], left atrial volume <96 ml [OR, 0.08, 95% CI: 0.023–0.274], right ventricular dimension <4 cm [OR, 0.042, 95% CI: 0.005–0.389].
Conclusion	Among patients with newly developed decompensation of CHF with reduced LV EF of non-ischemic origin and persistent atrial arrhythmias, TICMP was detected in 72% of patients. The probability of TICMP did not depend on baseline EF and duration of arrhythmias, but increased with the following baseline EchoCG parameters: EDV<174 ml, ESV<127 ml, left atrial volume <96 ml, right ventricular dimension <4 cm. The multifactorial analysis showed that a right atrial volume <96 ml is an independent predictor for the development of TICMP.

Keywords	Atrial fibrillation; CHF; tachycardia-induced cardiomyopathy
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Introduction

Chronic heart failure (CHF) belongs to the main group of pathologies causing disability and mortality in the adult population. Tachycardia-induced cardiomyopathy (TIC) may be a possible unrecognized causes of CHD. It is a cardiac disease that occurs in sustained

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tachyarrhythmia and is characterized by partially or completely reversible changes in the heart cavities after rhythm restoration or heart rate control achieved [1–4]. According to some data, its prevalence ranges from 10% to 37% in patients with atrial fibrillation (AF) referred for radiofrequency ablation [5, 6].

Differential diagnosis of TIC in patients with newly diagnosed left ventricular (LV) systolic dysfunction and uncontrolled supraventricular tachycardia remains a relevant and difficult clinical issue. There are only scattered literature data on the possible use of echocardiographically measured LV dimensions or a decrease in the levels of brain natriuretic peptide as prognostic markers of TIC. There have been no large studies on this topic [7–10].

The objective of this study is to identify possible predictors of tachycardia-induced cardiomyopathy in patients with new-onset decompensated CHF with reduced left ventricular ejection fraction (LVEF) of non-ischemic origin and persistent atrial tachyarrhythmias. In accordance with the above objective, the goals of this study are to determine the prognostic value of echocardiographic findings in patients with new-onset decompensated chronic heart failure with reduced left ventricular ejection fraction of non-ischemic origin in the development of TIC; assess the effect of constitutional and clinical data, including comorbidities, as predictors of TIC.

Material and methods

This is a full-design, prospective, observational study. All patients who were examined hospitalized in the cardiac department of the University Clinical Hospital No. 1 and the City Clinical Hospital No. 1 for heart failure (HF) with reduced and moderately reduced LVEF (less than 50%) associated with persistent atrial fibrillation/atrial flutter (AF/AFL) from January 2020 to May 2022 were examined.

The inclusion criteria were hospitalization for decompensated HF; atrial tachyarrhythmia with heart rate (HR) >100 bpm; reduced and moderately reduced LVEF<50% at the time of hospitalization; no history of myocardial infarction. The study also included patients without left atrial appendage thrombosis as shown by transesophageal echocardiogram and sinus rhythm restored later using electrical cardioversion, after pretreatment with amiodarone in some patients. All patients underwent 12 lead ECG at rest and transthoracic echocardiography at admission and after electrical cardioversion, 24-hour ECG monitoring was used to confirm stable sinus rhythm.

Exclusion criteria were documented ischemic origin of HF, known and documented specific origin of HF, inability to maintain sinus rhythm, primary valvular heart defects of any origin, hypertrophic cardiomyopathy, restrictive cardiomyopathy, chronic cor pulmonale, thyrotoxicosis, pregnancy, patient's unwillingness to participate in the study, mental disorders, severe comorbidities with life expectancy of not more than 12 months. Coronary artery disease (CAD) was diagnosed based on a history of myocardial infarction or in the presence of significant coronary artery atherosclerosis as shown by coronary artery angiography (CAG). Valvular disease was established by transthoracic echocardiography. Congenital heart defects, amyloidosis, hypertrophic cardiomyopathy, myocarditis, non-compaction cardiomyopathy, postpartum cardiomyopathy, arrhythmogenic right ventricular dysplasia, Fabry's disease were determined using the current Russian and international guidelines [11–13].

Selective CAG or multislice computed tomography (MSCT), CAG (contrast-enhanced coronary artery MSCT) was performed during hospitalization or within 12 months before hospitalization to exclude ischemic origin of HF. Magnetic resonance imaging (MRI) of the heart with contrast enhancement was performed after restoration of sinus rhythm and reduction of clinical manifestations of HF.

Included patients underwent repeat echocardiography within the coming three months to assess changes in dimensions of the heart chambers and ejection fraction using the Simpson method.

At the second (outpatient) stage, patients were divided into two groups: responders (patients with LVEF increased by 10% or more) and non-responders (patients with LVEF increased by less than 10% within 3 months of follow-up after restoration of sinus rhythm).

All diagnostic examinations (including CAG and cardiac MRI), drug treatment for CHF, and selection of treatment strategy were carried out in accordance with the orders of the attending physician under the current Russian and international guidelines and standards of care.

The primary endpoint was an increase in LVEF by 10% or more from the baseline levels within 3 months of follow-up. The Charlson comorbidity index was used to objectify the severity of multimorbid status [14]. Glomerular filtration rate (GFR) was calculated in adults using the CKD-EPI formula [15].

The study was approved by the local ethics committee of Sechenov University (protocol No. 31–20 dated 11.11.2020).

Statistical analysis

All quantitative variables were tested for normal distribution using the Kolmogorov–Smirnov test. Nor-

mally distributed variables were described as the means and standard deviations and spread. Non-normally distributed variables were described using the medians and interquartile ranges between the 25th and 75th percentiles and compared using non-parametric tests. The groups were compared by quantitative variables using the Student's t-test (in normal distribution) and the Mann-Whitney test (in non-normal distribution). Categorical variables were expressed as the absolute and relative values, and the chi-square test or Fisher's exact test was used for the comparison, depending on the sample parameters. All tests were two-tailed and $p<0.05$ was statistically significant.

Univariate and multivariate logistic regression analysis was performed to determine the effect of patient's clinical characteristics on the likelihood of TIC. The quality of binary classification was assessed using a ROC-curve analysis with the definition of critical values and using the Youden index. All statistical analyses were performed in SPSS version 22 (SPSS Inc., Chicago, USA).

Results

A total of 88 patients with reduced and moderately reduced LVEF who met the inclusion criteria were consistently included in the study. The mean age was 65±13.5 (18–92 years) years, and female patients prevailed among them (46 patients, 52.2%). The most common comorbidities were arterial hypertension (AH; 60 patients, 68.0%) and diabetes mellitus (DM; 18 patients, 20.5%). The median Charlson comorbidity index was 4 (3; 5). AF was established in 81 (92%) patients at admission to the hospital, and the rest had atrial flutter (AFL).

After restoration of sinus rhythm, LVEF increased by more than 10% from the baseline in 68 patients, and it did not change or increased by less than 10% in 20 patients. Thus, the diagnosis corresponded to the criteria of TIC in 72% of patients. The patient characteristics are presented in Table 1. The groups did not differ significantly in sex and age (65±13 years and 64±14 years, respectively, p=0.925), and physical finding (blood pressure (BP), HR, NYHA class).

The prevalence of individual comorbidities (DM, AH) did not differ among patients, but the Charlson comorbidity index was significantly lower in the TIC group (4 versus 5 in the non-responder group, p=0.022). The mean glomerular filtration rate estimated by the CKD-EPI formula was 59.9±22.6 mL/min/1.72 m^2 (16–120), with 14 (16%) patients having CKD C3b, i.e. eGFR less than 45 mL/min/1.72 m^2. Mean eGFR did not differ between the groups [59.4±20.6 mL/min/1.72 m^2 (20–107) in the TIC group, 58.6±25.1 mL/min/1.72 m^2 (28–120) in the non-responder group, p=0.936].

The duration of arrhythmia was more than 48 hours in more than half of patients (66 patients, 78.0%), including some patients having for several years, attack lasted more than 24 hours, but less than 48 hours in 8 (9.0%) patients; 13 (14.7%) patients were hospitalized within the first 24 hours from the attack onset. The median duration of arrhythmia was 36 days (1; 3,705) (42 days (1; 2,745) in the TIC group, 39 (1; 3,705) days in the non-responder group, p=0.871).

The analysis of the baseline echocardiographic data did not show significant differences in LVEF between the two subgroups (TIC 40±8.3%,18–50% and non-responders 38.55±7.9%, 24–50%); moreover, the incidence of cases with EF<30% also did not differ (9 patients in the TIC group and 2 in the non-responder group, p=1.0). Left atrial dimensions and left ventricular end-systolic volume were significantly lower in patients with TIC than in non-responders. Right ventricular dimension and right atrial volume were also lower in patients with TIC, but the differences were not statistically significant. The severity of mitral and tricuspid regurgitation, the mean estimated pulmonary artery pressure were similar between the groups (Table 1). Beta-blockers were the most frequently ordered drugs. There were no significant differences in drug therapy between the two groups.

The ROC curves were constructed to clarify the critical values of the baseline echocardiographic parameters that allow differentiating TIC. The critical value of LVESV (using the Youden index) was less than 127 mL with 96.7% sensitivity and 50% specificity, the critical value of the LVEDV was less than 174 mL with 89.7% sensitivity and 50% specificity, the critical value of the left atrial volume was less than 96 mL with 75.8% sensitivity and 80% specificity, the critical right ventricular dimension was less than 4 mm with 97% sensitivity and 30% specificity.

The effects of the main echocardiographic parameters on the likelihood of TIC was evaluated in the univariate (Table 2) and multivariate regression analyses with age and sex adjustments. The multivariate analysis showed that left atrial volume of less than 96 mL was an independent predictor of TIC (Table 3).

Discussion

Virtually any high-rate arrhythmia can cause the development of TIC: AF, AFL, supraventricular tachycardia, ventricular tachycardia, and frequent ventricular extrasystole [16]. TIC is diagnosed retrospectively due to complex detection, and its prevalence in patients with HF is unknown. The main

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Table 1. Characteristics of patients with TIC and patients with left ventricular ejection fraction increased by less than 10 % after sinus rhythm restoration (non-responders)

Parameters	TIC group (n=68)	Non-responder group (n=20)	p
Age, years	65±13 (18–92)	64±14 (29–83)	0.925
Male, n (%)	34 (50)	8 (40)	0.457
Arterial hypertension, n (%)	49 (72)	11 (55)	0.506
Diabetes mellitus, n (%)	14 (20)	4 (20)	0.744
CKD C3b, n (%)	9 (10.2)	5 (5.6)	0.263
Charlson Comorbidity Index	4 (3;5)	5 (3;5)	0.022
Atrial fibrillation, n (%)	64 (94)	17 (85)	0.609
Atrial flutter, n (%)	4 (8)	3 (15)	0.609
HR at admission, bpm	121.1±20.0 (101-181)	120.2±20.8 (100–179)	0.94
Systolic BP, mm Hg	129.7±22.9 (90-214)	127.6±27.2 (100–214)	0.845
Diastolic BP, mm Hg	81.8±14.1 (60-141)	81.3±19.6 (60–141)	0.962
Echocardiography at first screening			
LVEF, %	40±8.3 (18-50)	38.55±7.9 (24-50)	0.446
LVESV, mL	67.7±33.1 (29-140)	104.5±44.7 (26-172)	0.02
LVEDV, mL	109.2±41.5 (45-192)	104.5±44.8 (26-172)	0.203
LVEDD, mm	5.03±0.795 (4-7)	5.6±1.14 (4-7)	0.745
LA dimension, cm	4.53±1.14 (2-7)	5.68±1.41 (4-8)	0.034
LA volume, ml	80.8±28.9 (27-215)	117.8±41.3 (46-230)	0.003
RA volume, mL	59.6±24.3 (17-125)	71.95.6±27.8 (25-118)	0.183
RV dimension, cm	3.26±0.78 (2-7)	4.04±1.12 (2-6)	0.184
Mitral regurgitation, moderate-to-severe, %	6 (9)	1 (5)	0.76
Tricuspid regurgitation, moderate-to-severe, %	6 (9)	2 (10)	0.38
Pulmonary artery pressure, mm Hg	40.66±14.4 (20-95)	46.7±15.64 (23-70)	0.165
Heart rate monitoring			
History of radiofrequency catheter ablation, n (%)	9 (13)	2 (10)	0.588
History of cardioversion, n (%)	38 (56)	9 (45)	0.762
Treatment during follow-up			
Beta-blockers, n (%)	26 (38)	9 (45)	1.0
ACE inhibitors/ARBs, n (%)	25 (36)	4 (20)	0.85
Amiodarone, n (%)	7 (10)	6 (30)	0.06
Digoxin, n (%)	5 (7)	1 (5)	0.55

The data are expressed as the medians and interquartile ranges (Me (25 %;75 %)), the means and standard deviations (M±SD);
*p<0.05 versus the first screening; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; LVEF, left ventricular ejection fraction; LVESV, left ventricular end-systolic volume; LVEDV, left ventricular end-diastolic volume; LVESD, left ventricular end-systolic dimension; LVEDD, left ventricular end-diastolic dimension; LA, left atrium; LV, left ventricle; RV, right ventricle; RA, right atrium; TIC, tachycardia-induced cardiomyopathy.

diagnostic criterion is an increase in LVEF after rhythm restoration or achievement of HR control. According to some studies, TIC develops in 10% of patients with continuously recurrent supraventricular tachycardia (> 90% of the rhythm within 24 hours) [17]. Several experimental studies also showed that the higher risk of developing TIC was associated with constant frequent ventricular stimulation [1, 18–21]. Although the mechanism of tachycardia-induced cardiomyopathy is not exactly known, myocardial energy depletion, myocardial ischemia, abnormal cardiac calcium regulation, and remodeling of myocytes and/or extracellular matrix, are potential mechanisms of this biventricular dysfunction [20]. The analysis of a large register of patients with AFL revealed an initial decrease in LVEF in 15% of patients with TIC. The prevalence of TIC is likely to be 8–10% in all patients with persistent atrial tachyarrhythmias. Since AF contributes to the development of HF, and HF predisposes to AF, it is difficult to determine the exact percentage of TIC among

these patients [22]. According to our findings, 10% of patients hospitalized for HF associated with irregular heart rhythm met the diagnostic criteria of TIC, which is consistent with previously published works [23].

According to the largest available TIC register, AF is the most frequent arrhythmia substrate of TIC (84%) [24]. In our cohort, TIC developed as a result of AF in 98% of patients.

According to available data, TIC can develop within the first 24 hours after the onset of tachycardia attack or in several years after the onset of arrhythmia [25]. In the study cohort, TIC was diagnosed later than 48 hours after the onset of AF in the vast majority of patients (78.0%), and some patients have AH for a long time (more than 6 months).

There is no reliable and universally accepted parameter that can differentiate TIC from other forms of non-ischemic cardiomyopathies. There are only scattered study results demonstrating that LV dimension measured by echocardiography was smaller in patients with TIC than in non-responder patients [7, 8]. Jeong et al. showed that LV diastolic dimension ≤ 61 mm can predict TIC with sensitivity of 100% and specificity of 71.4% [8]. Similar results were obtained in a study evaluating bivetricular dysfunction in patients with new-onset HF using contrast-enhanced MRI of the heart (gadolinium test). Smaller LV volume and mass were shown to distinguish patients with TIC from non-responders [26]. Our study also supports the fact that the more preserved dimensions the left heart can also predict the development of TIC, but their sensitivity and specificity are relatively smaller compared to the above studies. This may be due to the peculiarities of sampling (left atrial volume OR 0.08, 95% CI: 0.023–0.274, p=0.0001, 75.8% sensitivity and 80% specificity; LVESV OR 0.034, 95% CI: 0.007–0.181, p=0.001, 96.7% sensitivity and 50% specificity). Our findings are the evidence that more pronounced LA dilation is characteristic of non-responders.

Research data have been previously presented showing that TIC develops at any age, including children and adolescents, and is more common in younger patients [6]. Fenelon et al. also demonstrated that age is a risk factor for TIC [27]. We did not obtain reliable data on the effect of age on the development of TIC. The persistently high prevalence of AF in elderly patients with comorbidities emphasizes the prospects of further research in this direction.

Limitations

This is a single-center, prospective, observational study with a relatively small cohort of patients. Patients were selected for cardioversion by the attending physicians. The obtained data should be confirmed in further large-scale clinical trials.

Table 2. Relationship between the echocardiographic parameters and their effects on the development of TIC in patients with decompensated chronic heart failure with reduced left ventricular ejection fraction

Parameter	Odds ratio	Confidence interval (95 %)	p value in the model
LVESV less than 127 mL	0.034	0.007–0.181	0.001
LVEDV less than 174 mL	0.115	0.035–0.371	0.001
Left atrial volume less than 96 mL	0.08	0.023–0.274	0.0001
Right ventricular dimension less than 4 mm	0.042	0.005–0.389	0.005

LVESV, left ventricular end-systolic volume; LVEDV, left ventricular end-diastolic volume; TIC, tachycardia-induced cardiomyopathy

Table 3. Multivariate analysis of TIC predictors

Parameter	Odds ratio	Confidence interval (95 %)	p value in the model
LVESV less than 127 mL	0.08	0.003-1.921	0.119
LVEDV less than 174 mL	1.354	0.084-21.803	0.831
Left atrial volume less than 96 mL	0.175	0.034-0.889	0.036
Right ventricular dimension less than 4 mm	0.331	0.0165-6.795	0.296

LVESV, left ventricular end-systolic volume;
LVEDV, left ventricular end-diastolic volume;
TIC, tachycardia-induced cardiomyopathy.

Conclusions

Tachycardia-induced cardiomyopathy was detected in 72% of patients with new-onset decompensated chronic heart failure with reduced left ventricular ejection fraction of non-ischemic origin and persistent atrial arrhythmias. The likelihood of tachycardia-induced cardiomyopathy was independent of baseline ejection fraction and duration of arrhythmia. However, it increased with the following baseline echocardiographic findings: end-diastolic volume of less than 174 mL [OR 0.115, 95% CI: 0.035–0.371], end-systolic volume of less than 127 mL [OR 0.034, 95% CI: 0.007–0.181], left atrial volume less than 96 mL [OR 0.08, 95% CI:

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0.023–0.274], right atrial dimension less than 4 mm [OR 0.042, 95% CI: 0.005–0.389].

The multivariate analysis showed that left atrial volume of less than 96 mL was an independent predictor of tachycardia-induced cardiomyopathy.

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REFERENCES

1. Gopinathannair R, Etheridge SP, Marchlinski FE, Spinale FG, Lakkireddy D, Olshansky B. Arrhythmia-Induced Cardiomyopathies: Mechanisms, recognition, and management. Journal of the American College of Cardiology. 2015;66(15):1714–28. DOI: 10.1016/j.jacc.2015.08.038
2. Hindricks G, Potpara T, Dagres N, Arbelo E, Bax JJ, Blomström-Lundqvist C et al. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). Russian Journal of Cardiology. 2021;26(9):234–329. DOI: 10.15829/1560-4071-2021-4701
3. Hindricks G, Potpara T, Dagres N, Arbelo E, Bax JJ, Blomström-Lundqvist C et al. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association of Cardio-Thoracic Surgery (EACTS): The Task Force for the diagnosis and management of atrial fibrillation of the European Society of Cardiology (ESC) Developed with the special contribution of the European Heart Rhythm Association (EHRA) of the ESC. European Heart Journal. 2020;42(5):373–498. DOI: 10.1093/eurheartj/ehaa612
4. Stronati G, Guerra F, Urbinati A, Ciliberti G, Cipolletta L, Capucci A. Tachycardiomyopathy in Patients without Underlying Structural Heart Disease. Journal of Clinical Medicine. 2019;8(9):1411. DOI: 10.3390/jcm8091411
5. Dandamudi G, Rampurwala AY, 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;5(8):1111–4. DOI: 10.1016/j.hrthm.2008.04.023
6. Medi C, Kalman JM, Haqqani H, Vohra JK, Morton JB, Sparks PB et al. Tachycardia-Mediated Cardiomyopathy Secondary to Focal Atrial Tachycardia: Long-term outcome after catheter ablation. Journal of the American College of Cardiology. 2009;53(19):1791–7. DOI: 10.1016/j.jacc.2009.02.014
7. Fujino T, Yamashita T, Suzuki S, Sugiyma H, Sagara K, Sawada H et al. Characteristics of Congestive Heart Failure Accompanied by Atrial Fibrillation With Special Reference to Tachycardia-Induced Cardiomyopathy. Circulation Journal. 2007;71(6):936–40. DOI: 10.1253/circj.71.936
8. Jeong Y-H, Choi K-J, Song J-M, Hwang E-S, Park K-M, Nam G-B et al. Diagnostic Approach and Treatment Strategy in Tachycardia-induced Cardiomyopathy. Clinical Cardiology. 2008;31(4):172–8. DOI: 10.1002/clc.20161
9. Nia AM, Gassanov N, Dahlem KM, Caglayan E, Hellmich M, Erdmann E et al. Diagnostic accuracy of NT-proBNP ratio (BNP-R) for early diagnosis of tachycardia-mediated cardiomyopathy: a pilot study. Clinical Research in Cardiology. 2011;100(10):887–96. DOI: 10.1007/s00392-011-0319-y
10. Bubeshka D.A., Snezhitskiy V.A. On the mechanism of tachycardia-induced cardiomyopathy in patients with atrial fibrillation. Grodno State Medical University Journal. 2015;2(50):24–9. [Russian: Бубешко Д.А., Снежицкий В.А. К вопросу о механизмах развития тахииндуцированной кардиомиопатии у пациентов с фибрилляцией предсердий. Журнал Гродненского Государственного медицинского университета. 2015;2(50):24-9]
11. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L et al. Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Journal of the American Society of Echocardiography. 2015;28(1):1-39.e14. DOI: 10.1016/j.echo.2014.10.003
12. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K et al. Guidelines for the Echocardiographic Assessment of the Right Heart in Adults: A Report from the American Society of Echocardiography. Journal of the American Society of Echocardiography. 2010;23(7):685–713. DOI: 10.1016/j.echo.2010.05.010
13. Zoghbi W. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and doppler echocardiography. Journal of the American Society of Echocardiography. 2003;16(7):777–802. DOI: 10.1016/S0894-7317(03)00335-3
14. Hemmelgarn BR, Manns BJ, Quan H, Ghali WA. Adapting the charlson comorbidity index for use in patients with ESRD. American Journal of Kidney Diseases. 2003;42(1):125–32. DOI: 10.1016/S0272-6386(03)00415-3
15. Cattran DC, Feehally J, Cook HT, Liu ZH, Fervenza F, Mezzano SA et al. Kidney Disease: Improving Global Outcomes (KDIGO) Glomerulonephritis Work Group. KDIGO Clinical Practice Guideline for Glomerulonephritis. Kidney International Supplements. 2012;2(2):139–274. DOI: 10.1038/kisup.2012.9
16. Berdibekov B.Sh., Aleksandrova S.A., Mrikaev D.V., Golukhova E.Z. Premature ventricular contraction-induced cardiomyopathy. Creative Cardiology. 2020;14(2):125–37. [Russian: Бердибеков Б.Ш., Александрова С.А., Мрикаев Д.В., Голухова Е.З. Кардиомиопатия, индуцированная желудочковыми экстрасистолами. Креативная кардиология. 2020;14(2):125–37]. DOI: 10.24022/1997-3187-2020-14-2-125-137
17. Cruz FES, Cheriex EC, Smeets JLRM, Atié J, Peres AK, Penn OCKM et al. Reversibility of tachycardia-induced cardiomyopathy after cure of incessant supraventricular tachycardia. Journal of the American College of Cardiology. 1990;16(3):739–44. DOI: 10.1016/0735-1097(90)90368-Y
18. Wilson JR, Douglas P, Hickey WF, Lanoce V, Ferraro N, Muhammad A et al. Experimental congestive heart failure produced by rapid ventricular pacing in the dog: cardiac effects. Circulation. 1987;75(4):857–67. DOI: 10.1161/01.CIR.75.4.857
19. Spinale FG, Hendrick DA, Crawford FA, Smith AC, Hamada Y, Carabello BA. Chronic supraventricular tachycardia causes ventricular dysfunction and subendocardial injury in swine. American Journal of Physiology. 1990;259(1):H218–29. DOI: 10.1152/ajpheart.1990.259.1.H218
20. Shinbane JS, Wood MA, Jensen DN, Ellenbogen KA, Fitzpatrick AP, Scheinman MM. Tachycardia-Induced Cardiomyopathy: A Review of Animal Models and Clinical Studies. Journal of the American College of Cardiology. 1997;29(4):709–15. DOI: 10.1016/S0735-1097(96)00592-X
21. Chow E, Woodard JC, Farrar DJ. Rapid ventricular pacing in pigs: an experimental model of congestive heart failure. American Journal of Physiology. 1990;258(5):H1603–5. DOI: 10.1152/ajpheart.1990.258.5.H1603
22. Batul SA, Gopinathannair R. Atrial Fibrillation in Heart Failure: a Therapeutic Challenge of Our Times. Korean Circulation Journal. 2017;47(5):644–62. DOI: 10.4070/kcj.2017.0040
23. Atabegashvili M.R., Shchekochikhin D.Yu., Gromyko G.A., Pershina E.S., Bogdanova A.A., Nesterov A.P. et al. Tachicardia-induced cardiomyopathy. Case report. Therapeutic Archive. 2021;93(4):465–9. [Russian: Атабегашвили М.Р., Щекочихин Д.Ю., Громыко Г.А., Першина Е.С., Богданова А.А., Нестеров А.П. и др. Кардиомиопатия, индуцирован-

ная тахикардией. Клиническое наблюдение. Терапевтический архив. 2021;93(4):465–9]. DOI: 10.26442/00403660.2021.04.200803
24. Mueller KAL, Heinzmann D, Klingel K, Fallier-Becker P, Kandolf R, Kilias A et al. Histopathological and Immunological Characteristics of Tachycardia-Induced Cardiomyopathy. Journal of the American College of Cardiology. 2017;69(17):2160–72. DOI: 10.1016/j.jacc.2017.02.049
25. Bozkurt B, Colvin M, Cook J, Cooper LT, Deswal A, Fonarow GC et al. Current Diagnostic and Treatment Strategies for Specific Dilated Cardiomyopathies: A Scientific Statement From the American Heart Association. Circulation. 2016;134(23):e579–646. DOI: 10.1161/CIR.0000000000000455
26. Oka S, Kai T, Hoshino K, Watanabe K, Nakamura J, Abe M et al. Rate Versus Rhythm Control in Tachycardia-Induced Cardiomyopathy Patients with Persistent Atrial Flutter. International Heart Journal. 2021;62(1):119–26. DOI: 10.1536/ihj.20-266
27. Fenelon G, Wijns W, Andries E, Brugada P. Tachycardiomyopathy: Mechanisms and Clinical Implications. Pacing and Clinical Electrophysiology. 1996;19(1):95–106. DOI: 10.1111/j.1540-8159.1996.tb04796.x

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