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Echocardiographic Predictors of Ventricular Tachyarrhythmias in Patients With Cardioverter-Defibrillator Implanted for Primary Prevention of Sudden Cardiac Death. Results From a two-Year Prospective Follow-up Study

Aim	To compare variables of transthoracic EchoCG for determining echocardiographic predictors and their prognostic role in the development of persistent paroxysmal ventricular tachyarrhythmias (VT) in patients with ischemic CHF who had been implanted with a cardioverter defibrillator (CD) for primary prevention of sudden cardiac death.
Material and Methods	This single-site prospective study included 176 patients with CHF of ischemic origin aged 58.7±7.4 years with a left ventricular ejection fraction (LV EF) of 30% [25; 34] % who had been implanted with CD. The follow-up duration was 24 months. The primary endpoint was a newly developed persistent paroxysm of VT (duration ≥30 sec) detected in the "monitored" VT area or a VT paroxysm that required electric treatment. The echocardiographic picture was evaluated by 28 variables. Statistical analysis was performed with the χ^2, Fisher's, and Mann–Whitney tests, and the one-factor logistic regression (LR). Prognostic models were developed with a multifactorial LR. The model accuracy was evaluated by 4 metrics: area under the ROC (AUC), sensitivity, specificity, and diagnostic efficacy.
Results	The primary endpoint was observed in 60 (34%) patients. Mean time to a persistent VT episode was 19.2±0.8 months (95% confident interval (CI): 17.5–20.8). Superior-inferior dimensions of the right and left atria (RA and LA, respectively) and the left atrial volume (LAv) were independent predictors for VT. The odds of VT development in patients of the study cohort increased with RAl ≥4.5 cm (odds ratio (OR), 1.6; 95% CI: 1.4–1.9; p=0.03), LAl ≥5.5 cm (OR, 2.5; 95% CI: 1.01–6.1; p=0.04), LAv ≥95 ml (OR, 3.2; 95% CI: 1.3–17.5; p=0.01). A comprehensive analysis of echocardiographic variables proved the prognostic potential of LAv that was linearly associated with the development of VT. The metrics of the best prognostic model were AUC 0.7±0.07 with 95% CI: 0.54–0.83; specificity, 20.9%; sensitivity, 95.7%; and diagnostic efficacy, 47%.
Conclusion	This study allowed evaluation of capabilities of transthoracic EchoCG for predicting the probability of VT in patients with CHF of ischemic origin and reduced LV EF. It was shown that linear and volumetric atrial dimensions could be used for stratification of risk of VT and for determining the tactics for primary prevention of sudden cardiac death in this patient category.

Keywords	Chronic heart failure; ventricular tachyarrhythmias; prognostic models; transthoracic echocardiography; ischemic heart disease
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According to the current clinical guidelines, patients with chronic heart failure (CHF) and left ventricular ejection fraction (LVEF) ≤ 35% have a high risk of sudden cardiac death (SCD), which is mainly due to ventricular tachyarrhythmias (VTs) [1]. In several large studies, implantable cardioverter-defibrillators (ICDs) were shown

to be an effective means of preventing SCD, especially in ischemic cardiomyopathy (ICM) [2–4]. Together, these proven hypotheses increased the number of ICD implantations and significantly decreased the prevalence of SCD in patients with CHF [5]. Meanwhile, it was shown that only about 20% of patients with ICDs used for primary prevention received appropriate life-saving shocks [6]. In other cases, the presence of ICD does not increase life expectancy and requires regular replacement of the device (every 5–6 years), which is associated with high costs and can be accompanied by life-threatening postoperative complications [7].

According to many experts, determination of the indications for interventional primary prevention of SCD based on LVEF alone should be revised. In this regard, it is relevant and demanded to search for new predictors of a very high risk of SCD in patients with CHF with reduced LVEF (HFrEF). The most promising solutions of these problems are the identification of electrocardiographic markers of fatal ventricular arrhythmias and the use of modern cardiac imaging techniques, such as ultrasound examinations for myocardial deformation assessment, contrast-enhanced cardiac magnetic resonance imaging [8].

Perhaps, the value of transthoracic echocardiography is not limited in the SCD risk stratification to providing information about LVEF. This hypothesis was tested in this study.

Objective

Conduct a comparative analysis of the transthoracic echocardiographic findings, establish echocardiographic predictors and their prognostic role in the onset of sustained VT episodes or VT episodes requiring electrotherapy (anti-tachycardia stimulation or shock therapy) in patients with ischemic CHF and LVEF ≤ 35% without syncope or a history of sustained ventricular arrhythmias.

Material and Methods

The study was conducted following the Good Clinical Practice and the Declaration of Helsinki. The study design was approved by the local ethics committee of the Astrakhan State Medical University (Russian Federation). All observed patients signed the informed consent to be included in the study.

Inclusion criteria:

Patients were enrolled from 2013 to 2021. Initially, 540 patients with CHF of NYHA functional class 3–4 and LVEF ≤ 35% were included in the study.

Exclusion criteria:

Patients with documented sustained VT episodes, data on a history of SCD, hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, valvular heart disease, and verified inherited channelopathies were excluded from the study.

ICD was implanted in 189 patients complying with the inclusion/exclusion criteria. A total of 176 patients completed the postoperative per-protocol follow-up (Figure 1).

The main clinical and demographic characteristics of the patients included in the study are presented in Table 1.

Table 1. Clinical and demographic characteristics of the included patients

Clinical parameter	All patients (n=176)	Patients without VT (n=116)	Patients with VT (n=60)	p_{3-4}
Age, years, M ± SD	58.7 ± 7.4	58.7 ± 6.4	58.7 ± 9.2	0.991
Male, n (%)	146 (83)	96 (83)	50 (83)	0.282
PICS, n (%)	130 (74)	86 (74)	44 (73)	0.864
AH, n (%)	118 (67)	82 (71)	36 (60)	0.521
Diabetes mellitus, n (%)	36 (20)	22 (19)	14 (23)	0.512
Obesity, n (%)	70 (40)	48 (41)	22 (37)	0.863
Stroke, n (%)	8 (5)	8 (7)	0	0.154
CKD, n (%)	98 (56)	70 (60)	28 (47)	0.761
Anemia, n (%)	8 (5)	6 (5)	2 (3)	0.744
AF (paroxysmal/persistent), n (%)	52 (30)	38 (33)	14 (23)	0.462
AF (permanent), n (%)	10 (6)	6 (5)	4 (7)	0.721
Drug therapy				
Beta blockers, n (%)	176 (100)	116 (100)	60 (100)	0.992
ACE inhibitors/ARB, n (%)	120 (68)	80 (69)	40 (67)	0.901
ARNI, n (%)	56 (32)	36 (31)	20 (33)	0.822
MRAs, n (%)	158 (90)	103 (88)	52 (92)	0.212
Loop diuretics, n (%)	174 (99)	114 (99)	60 (100)	0.891
Sotalol, n (%)	26 (15)	14 (12)	12 (20)	0.190
Amiodarone, n (%)	70 (40)	51 (44)	19 (32)	0.110
History of surgical revascularization, n (%)	167 (95)	109 (94)	58 (97)	0.351
Implanted two-chamber ICD, n (%)	112 (64)	72 (62)	40 (67)	0.523
Implanted CRT-D, n (%)	64 (36)	44 (38)	20 (33)	0.382
Remote ICD monitoring systems, n (%)	92 (52)	64 (55)	28 (47)	0.131

VT, ventricular tachyarrhythmia; ICD, implantable cardioverter-defibrillator; PICS, postinfarction cardiosclerosis; CKD, chronic kidney disease; AF, atrial fibrillation; CRT-D, cardiac resynchronization therapy defibrillator; ACE, angiotensin-converting enzyme, ARB, angiotensin II receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor.

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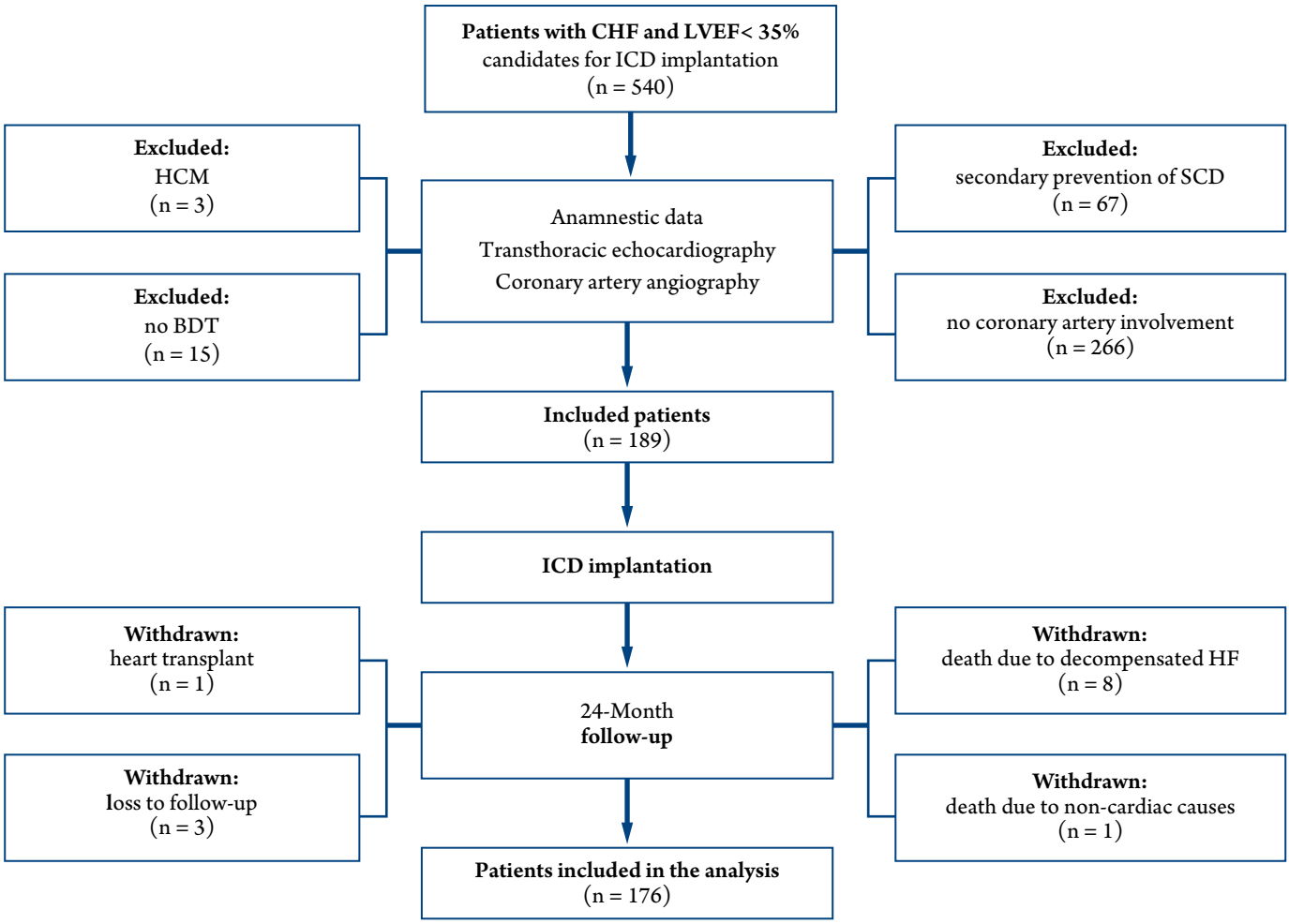
Figure 1. Study design



LVEF, left ventricular ejection fraction; ICD, implantable cardioverter-defibrillator; HCM, hypertrophic cardiomyopathy; BDT, best possible drug therapy; SCD, sudden cardiac death.

Cardioverter-defibrillator implantation and programming

All patients included in the study had ICDs implanted for primary prevention of SCD [9, 10]. Included patients received optimal medical therapy for heart failure and were followed for 2 years. The protocol of ICD programming and endpoint observation and registration has been previously described by the authors [11].

Echocardiogram analysis

Two experts performed transthoracic echocardiography before or immediately after implantation in normal heart rate using the following techniques: two-dimensional echocardiography (M-mode), Doppler echocardiography (color Doppler flow mapping). Standard transthoracic views and sections were received in all patients. Electrocardiogram synchronization was used to determine the phases of the cardiac cycle.

The following dimensions were determined: left atrial anteroposterior (LAap), medial-lateral (LAwidth) and superior-inferior (LAlength), LA volume (LAV); right atrial medial-lateral (RAwidth) and superior-inferior (RAlength); right ventricular; interventricular septal (IVS) thickness; LV posterior wall (LVPW); linear LV dimensions: end-systolic (LVESD) and end-diastolic (LVEDD); LV volumes: end-systolic (LVESV) and end-diastolic (LVEDV); LV linear and volumetric indexes: LVESDI, LVEDDI, LVESVI, LVEDVI; LVEF [12].

Mean LV wall thickness (LVWT), relative LV wall thickness index (RLVWTI), LV mass (LVM), and LVM relative to body surface area (LVMI) were calculated. The type of LV remodeling was determined based on LVMI and RLVWTI [12].

Study endpoints

The primary endpoint was new-onset sustained VT episode (≥ 30 s) detected in the monitoring zone of VT, or VT episode requiring electrotherapy (anti-tachycardia stimulation or shock therapy).

Statistical analysis

The study materials were statistically processed using the parametric and non-parametric analysis methods in

Table 2. Echocardiographic parameters of interest depending on the endpoint achievement

Echocardiographic parameter	All patients (n = 176)	Patients without VT (n = 116)	Patients with VT (n = 60)	p_{3-4}
LVEDV, mL (Me [Q1–Q3])	215 [190; 278]	219 [190; 277]	211 [190; 278]	0.991
LVEDVI, mL/m² (Me [Q1–Q3])	110 [89; 132]	108 [90; 134]	112 [89; 132]	0.992
LVESV, mL (Me [Q1–Q3])	150 [127; 198]	150 [128; 198]	156 [127; 190]	0.911
LVESVI, mL/m² (Me [Q1–Q3])	77 [62; 95]	77 [61; 95]	79 [64; 93]	0.784
LVEDD, cm (Me [Q1–Q3])	6.5 [6.1; 7.2]	6.5 [6; 7.2]	6.5 [6.3; 7]	0.792
LVEDDI, cm/m² (Me [Q1–Q3])	3.2 [2.9; 3.6]	3.3 [2.9; 3.6]	3.2 [2.9; 3.6]	0.561
LVESD, mm (M ± SD)	5.6 ± 0.8	5.6 ± 0.8	5.6 ± 0.8	0.844
LVESDI, cm/m² (M ± SD)	2.8 ± 0.5	2.8 ± 0.5	2.8 ± 0.5	0.983
IVS thickness, cm (Me [Q1–Q3])	1 [0.8; 1.1]	1 [0.9; 1.1]	1 [0.9; 1.1]	0.283
LVPWT, cm (Me [Q1–Q3])	1.1 [1; 1.1]	1.1 [1; 1.1]	1 [1; 1.2]	0.541
Mean LVWT, cm (Me [Q1–Q3])	1.05 [0.95; 1.15]	1.05 [0.9; 1.15]	1 [0.95; 1.1]	0.182
RLVWTI, cm (M ± SD)	0.31 ± 0.07	0.32 ± 0.07	0.3 ± 0.06	0.484
EF (Simpson), % (Me [Q1–Q3])	30 [25; 34]	30 [26; 33]	30 [23; 35]	0.673
LVM, g (M ± SD)	305 ± 88	309 ± 90	298 ± 85	0.563
LVMI, g/m² (Me [Q1–Q3])	144 [120; 181]	146 [125; 181]	136 [110; 176]	0.442
LAlength, cm (M ± SD)	6 ± 0.8	5.9 ± 0.8	6.4 ± 0.7	0.004
LAwidth, cm (M ± SD)	4.6 ± 0.6	4.5 ± 0.5	4.7 ± 0.6	0.081
LAap, cm (M ± SD)	4.7 ± 0.6	4.7 ± 0.6	4.8 ± 0.6	0.341
LAV, mL (Me [Q1; Q3])	97 [83; 112]	93 [83; 100]	110 [91; 138]	0.014
RAlength, cm (Me [Q1–Q3])	5.3 [4.7; 5.9]	5.2 [4.6; 5.8]	5.6 [5; 6]	0.041
RAwidth, cm (Me [Q1–Q3])	4 [3.6; 4.7]	4 [3.4; 4.7]	4.2 [3.7; 4.5]	0.252
PASP, mm Hg (Me [Q1; Q3])	40 [32; 51]	40 [32; 50]	39 [35; 57]	0.692
Concentric LVH, n (%)	10 (6)	8 (7)	2 (3)	0.432
Eccentric LVH, n (%)	166 (94)	108 (93)	58 (97)	0.441
AR, n (%)	4 (2)	4 (3)	0	0.321
MR, n (%)	68 (39)	46 (40)	22 (37)	0.483
TR, n (%)	40 (23)	24 (21)	16 (27)	0.442

LVEDV, left ventricular end-diastolic volume; LVEDVI, left ventricular end-diastolic volume index; LVESV, left ventricular end-systolic volume; LVESVI, left ventricular end-systolic volume index; LVEDD, left ventricular end-diastolic dimension; LVEDDI, left ventricular end-diastolic dimension index; LVESD, left ventricular end-systolic dimension; LVESDI, left ventricular end-systolic dimension index; IVS, interventricular septum; LVPWT, left ventricular posterior wall thickness; LVWT, left ventricular wall thickness; RLVWTI, relative left ventricular wall thickness index; EF, ejection fraction; LVM, left ventricular mass; LVMI, left ventricular myocardial mass index; LVap, anterior-posterior left atrial dimension; LAwidth, medial-lateral left atrial dimension; LAlength, superior-inferior left atrial dimension; LAV, left atrial volume; RAwidth, medial-lateral right atrial dimension; RAlength, superior-inferior right atrial dimension; AR, aortic regurgitation; MR, mitral regurgitation grade II or more; TR, tricuspid regurgitation grade II or more.

IBM SPSS Statistics 23. The applied statistical methods have been previously described by the authors [11]. If the distribution was confirmed normal, the data were described using the means (M), standard deviations (SD). Non-normally distributed data were expressed using the medians (Me) and the lower and upper quartiles (Q1–Q3). Relationships between the variables were analyzed using the Spearman's correlation analysis. The critical significance threshold for the statistical hypotheses was set as $p = 0.05$.

Multivariate prediction models were constructed using the binary logistic regression method. The independent variables were selected by stepwise inverse selection using the Wald test as an exclusion criterion. Statistical significance of the model was determined by the χ^2 test. The Nagelkerke R^2 was used as a measure of definiteness showing the part of the dispersion explainable using the logistic regression. ROC analysis was carried out and the area under the curve (AUC) was calculated to estimate the predictive value of the model and find the threshold value of the obtained function at the cut-off point.

Results

The studied cohort of patients was characterized by increased LV linear and volumetric dimensions and a significant decrease in LVEF (Table 2). Pathological LV remodeling with signs of eccentric hypertrophy was predominant (n = 166, 94%).

The primary endpoint was registered in 60 (34%) patients within the two-year follow-up. Mean time to the de-

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velopment of sustained VT episode was 19.2±0.8 (95% CI 17.5–20.8) months.

The groups formed based on the fact of achieving the endpoint did not differ in the main clinical and demographic characteristics (Table 1). The analysis of the echocardiographic parameters of interest showed statistically significant differences in LAlength (p=0.004), RAlength (p = 0.04), LAV (p = 0.014).

The ROC curves were used to find the optimal cut-off values of LAlength, RAlength, and LAV, which allowed classifying patients according to the risk of registering the endpoint (Table 3).

The likelihood of VT in the study cohort patients increased nearly twofold in RAlength ≥ 4.5 cm (OR 1.6; 95% CI 1.4–1.9; p=0.03). LAlength ≥ 5.5 cm in patients with ICM and reduced LVEF increased 2.5-fold the likelihood of the arrhythmogenic scenario of SCD (OR 2.5; 95% CI 1.01–6.1; p=0.04). LAV ≥ 95 mL was prognostically significant (OR 3.2; 95% CI 1.3–17.5; p=0.01).

The univariate logistic regression revealed seven factors of the greatest prognostic potential (p<0.01), which were linearly associated with the onset of VTs. They included LAlength, LAwidth; LAV; RAlength; LAlength ≥ 5.5 cm; LAV ≥ 95 mL; RAlength ≥ 4.5 cm (Table 4). A correlation matrix was constructed to address possible multicollinearity. LAlength was found to be closely correlated with other factors: RAlength (r=0.77; p<0.01) and LAlegth ≥ 5.5 cm (r=0.71; p<0.01).

LAwidth; LAV; RAlength; LAlength ≥5.5 cm were included in the multivariate logistic regression analysis. The obtained statistically significant prediction models differed little in the Nagelkerke coefficient of determination and did not differ in the metrics (Table 5).

The metrics of the best model were the following: AUC=0.7±0.07, 95% CI 0.54–0.83; specificity – 90.7%, sensitivity – 30.4%; diagnostic efficiency – 69.7% (p=0.002). Based on the results of the ROC analysis, the threshold function value of 0.211 was selected. After adjusting the classification threshold based on the ROC-curve analysis results, the diagnostic efficacy of the best prediction model (model 4) was 47% (sensitivity – 95.7%, specificity – 20.9%).

Discussion

There is no doubt that LVEF is nonlinearly correlated with the risk of SCD in CHF [13],but is likely to lack sufficient sensitivity [14]. This is due to the available data that sustained VTs occur in only 20–25% of patients with LVEF <35% [6, 15]. The data obtained in this study indicate that this echocardiographic parameter loses its

Table 3. Predictive power of echocardiographic parameters for the likelihood of VTs

Parameter	LAlength	LAV	RAlength
Area under the ROC curve	0.69	0.685	0.619
Standard error	0.07	0.072	0.071
p	0.005	0.011	0.095
95 % confidence interval	0.559–0.822	0.543–0.826	0.479–0.758
Cut-off threshold	5.5 cm	95 mL	4.5 cm
Sensitivity, %	91.3	73.9	91.3
Specificity, %	79.1	46.5	81.4

VT, ventricular tachycardia; LAlength, superior-inferior left atrial dimension; LAV, left atrial volume; RAlength, superior-inferior right atrial dimension.

Table 4. Correlation between the factors of interest and the endpoint

Factor	Univariate analysis			Multivariate analysis		
	OR	95% CI	p	OR	95% CI	p
LAlength	2.4	1.3–4.4	0.006	–	–	–
LAwidth	2.2	0.9–5.1	0.081	0.6	0.2–2.5	0.521
LAV	1.03	1.01–1.05	0.008	1.03	1.01–1.05	0.083
RAlength	1.7	0.99–3.03	0.052	1.1	0.5–2.5	0.782
LAlength ≥ 5.5 cm	2.5	1.01–6.1	0.042	1.6	0.3–9.3	0.623
LAV ≥ 95 mL	3.2	1.3–17.5	0.013	–	–	–
RAlength ≥ 4.5 cm	1.6	1.4–1.9	0.031	–	–	–
RAwidth	1.2	0.7–2.1	0.441	–	–	–

OR, odds ratio; CI, confidence interval; LAlength, superior-inferior left atrial dimension; LAwidth, medial-lateral left atrial dimension; LAV, left atrial volume; RAlength, superior-inferior right atrial dimension; RAwidth, medial-lateral right atrial dimension; LVEDD, left ventricular end-diastolic dimension; LVPWT, left ventricular posterior wall thickness; LVM, left ventricular mass.

Table 5. Comparative characteristics of the multivariate prediction models

Parameter	Equation variables	R^2	Se	Sp	p
Model 1	RAlength; LAwidth; LAV; LAlength ≥5.5 cm	0.21	30.4	90.7	0.031
Model 2	LAwidth; LAV; LAlength ≥ 5.5 cm	0.2	30.4	90.7	0.014
Model 3	LAwidth; LAV	0.2	30.4	90.7	0.006
Model 4	LAV	0.19	30.4	90.7	0.002

RAlength, superior-inferior right atrial dimension; LAwidth, medial-lateral left atrial dimension; LAV, left atrial volume; LAlength, superior-inferior left atrial dimension; R^2, Nagelkerke coefficient of determination; Se, sensitivity; Sp, specificity.

prognostic potential and cannot be used to determine the likelihood of VTs in patients with HFrEF.

According to our findings, there were no statistically significant correlations of LVM and LV wall thickness with the study endpoint, although these indicators can be used as independent predictors of VT [16] and in multivariate prediction models [17]. The obtained data showed that the process of cardiac remodeling was initiated with significantly disturbed local wall motion in four or more segments of LV and significantly reduced LVEF in all patients with HFrEF included in the study, which reduces the prognostic significance of identifying a myocardial scar as a prediction factor for VTs in patients with ICM and severe systolic dysfunction.

The prognostic significance of LAV is confirmed by the results of multivariate analysis. The results of univariate analysis, which indicate the independent prognostic value of other atrial linear measurement (RAlength, LAlength) and LAV, are even more convincing. The cut-off values were proposed for each of these factors with an equal or higher values indicating a high VT risk of (RAlength ≥4.5 cm; LAlength ≥ 5.5 cm; LAV ≥ 95 mL).

LA is a determinant in LV filling from the pulmonary veins and optimizing cardiac output. Atrial cardiomyopathy, which acts as a mechanism of CHF initiation and progression, develops in regular volume overload [15]. In the context of interest, manifestation of AF is the main clinical manifestation of LA reservoir dysfunction. In a large meta-analysis, AF increased the risk of SCD in CAD (OR 1.56; 95% CI 1.17–6.25; p<0.001) and in the presence of CHF (OR 1.75; 95% CI 1.4–2.19; p<0.001) [18]. We have previously confirmed the presence of the correlation between AF and VT [15].

LA functions are not limited to hemodynamics, this heart chamber is involved in the endocrine and neurovegetative processes affecting the cardiovascular system [19]. The neuroendocrine effects accompanying this condition, such as increased sympathetic activity, release of vasopressin, hypersecretion of natriuretic peptides, significantly increase the likelihood of VT manifestation [20, 21].

Highly sensitive (95.7%) multivariate prediction model was constructed in the study, which allows determining the high risk of VTs in patients with ICM and LVEF ≤35%. The significant weaknesses of the model are low specificity (20.9%) and low coefficient of determination (0.19) indicating the variance of the dependent variable explained by the model in question. The model metrics can be significantly improved by including additional factors that have no correlations with the identified echocardiographic predictors, e.g., clinical anamnestic data, findings of electrocardiography and cardiac magnetic resonance imaging. The data presented are intermediate results of the ongoing single-center prospective study, which will include at least 450 patients with HFrEF. Increasing the number of observations as new patients are included can undoubtedly increase the significance of future findings.

Limitations

The limitations of the study include the relatively small number of subjects and the lack of analysis of the effect of resynchronization therapy on the endpoint registration.

The identification of VT episodes was limited by the programmed detection interval, which limited the diagnosis of arrhythmias with a lower heart rate.

Conclusion

The data obtained suggest that left ventricular ejection fraction, left ventricular mass, and left ventricular wall thickness have no significant correlations with the frequency of detecting sustained ventricular tachycardias in patients with ischemic chronic heart failure and left ventricular ejection fraction ≤ 35%, and pathological atrial remodeling may increase the likelihood of ventricular tachycardias. Left atrial linear and volumetric measurements can be

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used to stratify the risk of ventricular tachycardias and determine the strategy of primary prevention of sudden cardiac death in patients of this category.

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