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PREDICTORS OF LONG-TERM VENTRICULAR TACHYARRHYTHMIA RECURRENCE AFTER COMBINED ENDO-EPICARDIAL ABLATION IN PATIENTS WITH STRUCTURAL HEART DISEASE

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Purpose. To identify predictors of ventricular tachycardia (VT) recurrence after endo-epicardial ablation in patients with structural heart disease.

Methods. A prospective observational study included 39 patients with structural heart disease and indications for catheter ablation of ventricular tachyarrhythmia. Endo- and epicardial electroanatomical mapping of the ventricular myocardium and ablation of abnormal electrical activity areas were performed. Clinical, ECG characteristics, and voltage maps of bipolar potentials (limits of scar detection <0.5 mV, normal activity >1.5 mV) and unipolar signals (limits <5.0 and >9.0 mV, respectively) on endo- and epicardial surfaces were evaluated. Intraprocedurally, the procedure was considered effective when no VT was inducible; partially effective - when only clinical VT(s) was/were non-inducible. Scheduled patient visits or remote monitoring were performed at 6, 12 and 24 months, and then annually.

Results. The mean age of the patients was 49.5±15.7 years (34 men and 5 women). VT recurrences at 6 months were more often detected in non-ischemic cardiomyopathy patients, in subjects with non-paroxysmal atrial fibrillation (42.9% vs. 7.4%), with a lower VT-QRS amplitude in lead III (0.6 [0.4;1.07] versus 1.28 [0.99; 1.53] mV), and when epicardial "scar" area prevailed over endocardial, P<0.05 for all listed parameters. At 12-months, VT recurrence was more common in patients with partially effective ablation (33.3% vs. 5.0% in patients with effective ablation, P=0.02). The presence of electrical storm at the time of ablation was independently associated with recurrences (HR 4.32; 95% CI: 1.06-17.48; P=0.04).

Conclusion. In a heterogeneous group of patients, clinical and electrophysiological factors associated with VT recurrence have been identified at various follow-up periods after endo-epicardial ablation. Electrical storm ablation is an independent predictor of VT recurrence in the long-term, up to 5 years of follow-up.

Key words: epicardial ablation; ventricular tachycardia recurrence; subepicardial substrate; radiofrequency ablation; electrical storm

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Radiofrequency catheter ablation (RFA) is a modern, highly effective method for ventricular tachycardia (VT) management. The VANISH study has demonstrated the benefit of catheter ablation compared to extending drug therapy to prevent VT recurrences [1, 2]. As new scientific evidence emerges on the efficacy and outcomes of new approaches to the interventional treatment of VT, the classes of indications for catheter ablation are revised and the possibilities for its use are expanded [3]. However, despite the success in treating VT, in some cases endocardial ablation of the VT substrate is ineffective due to the intramural/subepicardial location of the arrhythmogenic substrate. In this case, epicardial mapping and ablation of the tachycardia substrate is performed. Epicardial access is important in patients with arrhythmogenic right ventricular cardiomyopathy (ARVC) [4-6], postmyocardial cardiosclerosis and

postinfarction scarring [7]. However, despite the combined endo-epicardial substrate classification of VT, VT recurrences are not uncommon. The issue of identifying predictors of VT recurrences, improving ablation approaches, and enhancing their efficacy is undoubtedly important.

The aim of this study was to identify predictors of recurrence of ventricular tachycardia in patients with structural heart disease after epicardial ablation and to determine the likely association between clinical and electrophysiological factors and the characteristics of monomorphic ventricular tachycardia.

MATERIAL AND METHODS

A prospective observational study included patients with VT with structural heart disease (with a history of myocardial infarction or myocarditis, ARVC) and indications for arrhythmia substrate RFA who have signed a consent form for epicardial ablation other than standard endocardial ablation. The inclusion criteria were: a proven history of myocardial infarction or myocarditis or definite/problematic ARCV (myocardial changes visualised by transthoracic echocardiography and/or magnetic resonance imaging (MRI)), presence of persistent VT paroxysms (or frequent symptomatic ventricular extrasystoles (VE)), documented by ECG and/or interview data from an implanted cardioverter defibrillator (ICD), with ineffective antiarrhythmic therapy (AAT). Exclusion criteria were: stenotic coronary artery atherosclerosis disease requiring revascularization; acute myocarditis; history of open surgery with pericardial dissection; acute or exacerbated inflammatory disease; presence of three-component antithrombotic therapy without possibility of interruption; myocardial infarction less than 3 months ago; presence of acute coronary syndrome at the time of screening. Patients underwent a standard examination: resting ECG, echocardiography, Holter monitoring, coronarography, interrogation and setting of implanted ICD/cardiac resynchronisation therapy with defibrillation function (CRT-D), cardiac MRI.

Endocardial and epicardial access, electrophysiological study and catheter ablation

In the X-ray operating room, electrophysiological examination, tachycardia substrate mapping and RFA were performed. Anesthesiological support was provided with artificial pulmonary ventilation.

Epicardial (EPI) access was gained via subxiphoid puncture; the technique is described in detail in a previous publication [8]. A Preface Multipurpose Introducer (Cordis, USA) was inserted into the pericardial space. Endocardial (ENDO) access to the right ventricle was made by puncturing the femoral vein. The ENDO access to the left ventricle (LV) was made retrograde transaortally by puncturing the femoral artery and transseptally.

Transseptal puncture of the interatrial septum was performed under fluoroscopy using a Brockenbrough BKR-1 needle (St. Jude Medical, USA). After positioning the introducer in the LV, heparin was injected intravenously at a dose of 80-100 IU per 1 kg body weight, and hypocoagulation was further maintained while controlling the activated clotting time (target value above 250 s).

The procedure was performed with the non-fluoroscopic navigation system CARTO 3. NaviStar Thermocool or SmartTouch (Biosense Webster, USA) was used for mapping and ablation; Pentaray multipole navigation electrode (Biosense Webster, USA) was used for mapping in some patients.

Three-dimensional reconstruction of the right ventricle and LV surfaces ENDO was performed first, followed

Table 1.

Clinical profile of patients

Parameter	Value
VT etiology, n	
- PICS	15
- ARVC	14
- myocarditis	6
- unspecified etiology	4
Hypertension, n	21
Diabetes mellitus, n	4
COPD, n	6
Atrial fibrillation, n*	6
Electric storm, n	11
ICD/CRT-D, n	29
Follow-up time, months	24 [MCD: 6; 33]
Average age, years**	50±16
Length of history of arrhythmia, months	19.5 [MCD: 5; 48]
Average VT cycle length, ms	375 [MCD: 332; 471]
Previous substrate RFA procedures	22
- 1 surgery	13
- 2 or more surgeries	9
“Acute” effect	32
Partial effect	4
Presence of LEP on the epicardial surface	27
Pseudo-delta wave width, ms	72±27
Left atrial volume index, ml/m^2	45±18
Average LV EF, %	46±14
TAPSE, mm	18±5
QTc at sinus rhythm, ms	462 [MCD: 438;513]
QRS width on sinus rhythm	115±33
Amplitude of the QRS VT in lead III	1.23 [MCD: 0,84;1,52]

Notes hereafter: VT, ventricular tachycardia; PICS, postinfarction cardiosclerosis; ARVC, arrhythmogenic right ventricular cardiomyopathy; COPD, chronic obstructive pulmonary disease; *, persistent/permanent forms; ICD, implantable cardioverter-defibrillator; CRT-D, cardiac resynchronization therapy with defibrillation function; **, at the time of surgery; RFA, radiofrequency ablation; LVP, late ventricular potentials; LV EF, left ventricular ejection fraction

by reconstruction of the cardiac surface EPI. The following limits for detection of scarring and altered zones were used in the creation of voltage maps: 0.5-1.5 mV for bipolar signal registration and 5.0-9.0 mV for unipolar signal registration. Electroanatomical maps were drawn with a color boundary of 5-10 mm when mapping with NaviStarThermocool or SmartTouch ablation catheters (Biosense Webster, USA) and 2 mm when mapping with PentaRay multipolar catheters (Biosense Webster, USA). Areas of late potentials and fragmented potentials were marked on the maps. The integrated Area Measurement module was used to calculate the registration area of altered electrical activity and low amplitude myocardium on both bipolar and unipolar maps on the ENDO and EPI surfaces.

The location of the VT was determined by activation and stimulation mapping, by the positive effect of ablation. A standard electrophysiological study protocol was performed to induce VT: programmed stimulation with 1, 2, 3 and 4 extrastimuli from the right ventricular apex and output tract or from LV, frequent volley stimulation of the ventricles. In case of induction of sustained and hemodynamically relevant VT entrainment mapping was performed.

Prior to RFA to the epicardial surface, selective coronarography was performed to assess the location of the mapped area in relation to the coronary arteries. Radiofrequency applications were performed at a distance of at least 10 mm from the coronary artery. Stimulation mapping of the anatomical projection zone of the left phrenic nerve was performed to prevent its possible damage during ablation in the lateral wall of the LV.

Radiofrequency exposure was performed with the following parameters: Power 40-50 W, duration up to 40 seconds; the electrode was flushed with 0.9% NaCl solution at a rate of 30 ml/min. Radiofrequency ablation was performed in the area of late and fragmented potentials until they disappeared or the amplitude of the potentials decreased significantly (above 85%). Ablation was also performed in these areas when mapping the VT entry/exit zones and delayed conduction channels within the

Table 2.

Endocardial and epicardial volt mapping (see continuation)

Patient No.	Endocardial surface		S ratios (bi- and unipolar) on the endocardial surface		Epicardial surface		S (bipolar) ratios on epi- and endocardial surfaces	
	% S<0.5mV×	% S<1.5 mV	<0.5 mV and<5 mV*	<1.5 mV and<9 mV**	% S<0.5 mV×	% S<1.5 mV	<0.5 mV †	<1.5mV ††
1	5	8.4	0.57	0.61	12.3	83	0.73	0.92
2	4.2	4.2	0.06	0.85	23.1	24.1	0.90	0.91
3	3	14.2	0.88	0.62	17.2	70.3	0.85	0.82
4	-	-	-	-	-	-	-	-
5	1	5,4	0.88	0.69	10.9	27.5	0.93	0.78
6	1.4	8.1	0.75	0.63	2.7	4	0.25	0.35
7	8.9	18.6	-0.44	0.12	0	4.3	-1.0	-0.10
8	0	0	1.0	1.0	8.9	14.4	1.0	1.0
9	0	0	1.0	1.0	2.3	8.5	1.0	1.0
10	5.7	6	0.49	0.50	8.4	8.4	0.65	0.63
11	4	87.3	0.73	0.05	3.9	78.1	0.39	0.35
12	14	28	0.72	0.61	17.8	25.1	0.14	0.05
13	25.6	50	0.14	0.05	13.9	86.6	0.30	0.71
14	7.9	23.6	-0.62	0.2	11.8	21.8	0.56	0.37
15	3.3	12.2	0.83	0.80	12.9	75.8	0.89	0.91
16	-	-	-	-	-	-	-	-
17	-	-	-	-	-	-	-	-
18	1.3	7.4	0.47	0.45	-	-	-	-
19	-	-	-	-	88.3	92.1	-	-
20	10.8	65.4	-	-	-		-	-
21	14	50.9	0.42	0.17	22.8	83	0.66	0.66
22	8.4	14.9	0.58	0.1	4.5	13.7	-0.19	0.08
23	13.2	62.1	1.0	1.0	19.2	56.8	1.0	1.0
24	13.1	60.9	0.53	0.17	0.3	2.9	-0.90	-0.82
25	28.8	63.3	0.51	0.20	21.6	26.9	0.27	-0.01

Table 2.

Endocardial and epicardial volt mapping (continuation)

Patient No.	Endocardial surface		S ratios (bi- and unipolar) on the endocardial surface		Epicardial surface		S (bipolar) ratios on epi- and endocardial surfaces	
	% S<0.5mV×	% S<1.5 mV	<0.5 mV and<5 mV*	<1.5 mV and<9 mV**	% S<0.5 mV×	% S<1.5 mV	<0.5 mV †	<1.5mV ††
26	0	7.6	-	0.27	1.1	8.2	1	0.68
27	0	0	-	-	0	0	-	-
28	6.6	9.3	0.44	0.81	18.5	93	0.76	0.93
29	21.6	24.8	0.44	0.43	12.7	14.9	0.40	0.40
30	-	-	-	-	-	-	-	-
31	0	0.5	0.49	0.52	2.4	74.8	0.71	0.66
32	-	-	0.54	0.44	-	-	-	-
33	0	0	-	1.0	4.7	8.4	1.0	1.0
34	1.2	2.9	0.48	0.48	77.2	86.4	1.0	0.98
35	46.5	98.9	0.39	0.15	12.5	17.6	0.43	0.09
36	22.6	77.6	0.49	-0.03	17.1	18.6	0.31	-0.22
37	34	87.9	0.45	0.05	93.3	96.3	0.79	0.54
38	2.3	5.8	0.85	0.87	88.7	88.7	0.86	0.68
39	-	-	-	-	-	-	-	-
Total %	5.4 [1.3;13.4]	13.2 [5.7; 53.4]	0.51 [0.4; 0,8]	0.5 [0.2;0.8]	12.5 [4.2; 18.9]	25.1 [11.1; 80.6]	0.71 [0.3; 0.9]	0.66 [0.4; 0.9]

Note: S - signal area; × - P < 0.05; * - according to the formula (S1endo_uni-S1endo_bi)/(S1endo_uni+S1endo_ bi), where S1endo_bi is the recording area of the bipolar signals < 0.5mV on the endocardial surface, S1endo_uni - recording area of the unipolar signals < 5mV on the endocardial surface; ** - according to the formula (S2endo_uni-S2endo_bi) / (S2endo_ uni+S2endo_bi), where S2endo_bi is the area of the bipolar signals < 1.5 mV on the endocardial surface, S2endo_uni - recording area of the unipolar signals < 9 mV on the endocardial surface; † - according to the formula (S1epi_bi-S3endo_bi) / (S1epi_bi+S3endo_bi), where S1endo_bi is the area of the bipolar signals < 0.5 mV on the endocardial surface, S3epi_bi is the area of the bipolar signals < 0.5 mV on the epicardial surface; †† - according to the formula (S2epi_bi-S4endo_bi) / (S2epi_bi+S4endo_bi), where S2endo_bi is the area of the bipolar signals < 1.5 mV on the endocardial surface, S4epi_bi is the area of the bipolar signals < 1.5 mV on the epicardial surface.

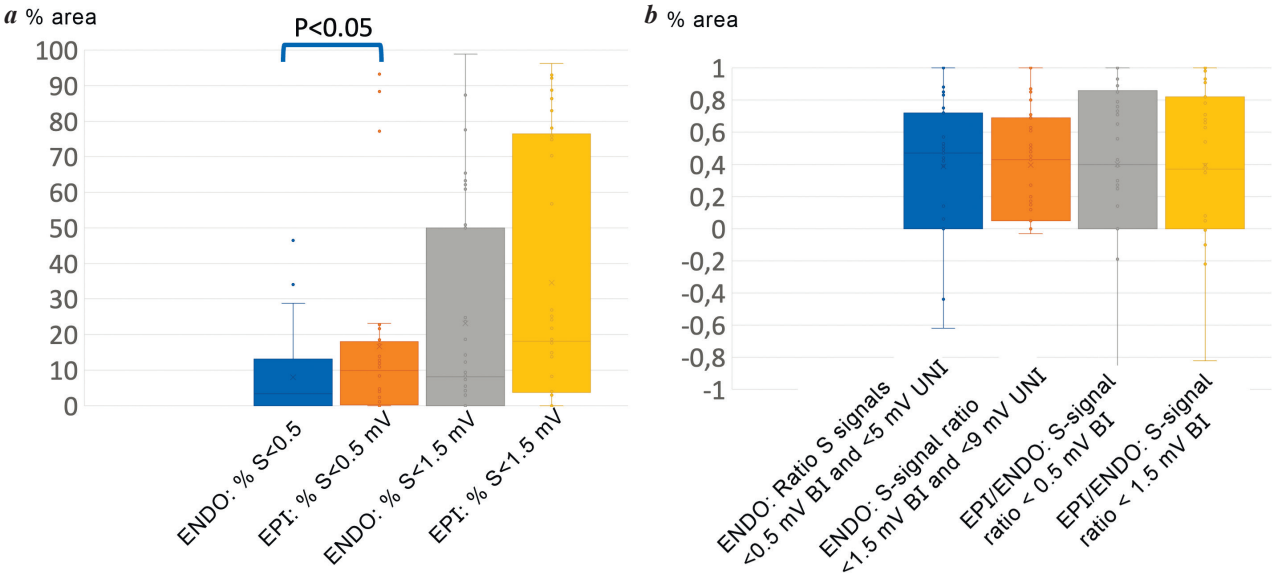

Fig. 1. Histograms characterising the relative areas of low-amplitude electrical signals in the whole group of patients: a - the relative area (as a percentage of the total area) of registration of electrical potentials < 0.5 mV and < 1.5 mV on endocardial and epicardial ventricular maps; b - normalised ratio of the areas of registration of low-amplitude potentials on endocardial and epicardial surfaces. Note: S - area, BI - bipolar signals, UNI - unipolar signals, ENDO - endocardial surface, EPI - epicardial surface.

scar. When mapping of the VT cycle was performed, RFA were performed in the area of "critical" tachycardia isthmus.

Treatment was effective if repeat ablation mapping showed no late and fragmented potentials and no seizure (amplitude 10 V, stimulus duration 1 ms, cycle 500 ms). At the end of the procedure, a repeat stimulation protocol was performed to induce VT. The procedure was considered completely effective if no induction of VT was observed; ineffective if induction of clinical VT persisted; the absence of induction of clinical VT was assessed as a partial effect. Induction of ventricular fibrillation was not considered in the acute effect evaluation.

At the end of the procedure, the pericardial introducer was removed or replaced with a drain, which was removed after 12-24 hours. Before removal of the pericardial introducer, triamcinolone was injected into the pericardial cavity at a dose of 2 mg/kg to prevent pericardial adhesions.

Table 3.

Clinical characteristics and electrophysiological substrate characteristics in patients with and without recurrent VT after index ablation

	Recurrence of VT (6 months follow-up)		Recurrence of VT (12 months follow-up)		Recurrence of VT (24 months follow-up)	
	Yes	No	Yes	No	Yes	No
Number of patients[@]	7	27	6	14	6	18
Gender (m/w)	4/3*	25/2*	5/1	11/3	5/1	17/1
Coronary cardiomyopathy, n (%)	1 (14)	10 (37)	0	8 (57)	0††	9 (50)††
Hypertension, n (%)	5 (71)	14 (52)	3 (50)	11 (79)	3 (50)	11 (61)
Diabetes mellitus, n (%)	2 (29)	2 (7)	1(17)	3 (21)	1 (17)	2 (11)
COPD, n (%)	2 (29)	3 (11)	2 (33)	3 (21)	1 (17)	3 (17)
Atrial fibrillation, n (%)	3 (43)*	2 (7)*	1 (17)	3 (21)	1 (17)	1 (6)
ES in the anamnesis, n (%)	1 (14)	4 (15)	3 (50)	5 (36)	2 (33)	1 (6)
Average age[$], years (Me [MCD])	48 [30;62]	53 [38;61]	34 [25;45]	53 [39; 61]	39 [29; 50]	54 [44;64]
DAA, months (Me [MCD])	37 [18;73]	20 [5;48]	37 [27;54]	20 [5;61]	61 [55; 78]	9 [3; 25]
LV EF, % (Me [MCD])	38 [32; 49]	48 [38; 59]	40 [29;50]	52[38;60]	49 [46; 53]	55 [42; 60]
TAPSE, mm (Me [MCD])	19 [16; 21]	18 [15; 21]	17 [16;21]	18 [14;21]	21 [20; 22]	18 [14; 20]
QRSav, ms (Me [MCD])	111 [85;151]	104 [92;126]	106 [89; 138]	97 [88; 117]	93 [81; 152]	98 [90; 121]
QTcav, ms (Me [MCD])	484 [436;547]	457 [434;485]	411 [402; 488]	461 [443; 518]	488 [425;557]	460 [438;487]
QRSvt, ms (Me [MCD])	250 [223;250]	203 [199;240]	223 [202;250]	204 [194;243]	200 [195;214]	208 [202;250]
VT cycle length, ms (Me [MCD])	399 [398;400]	361 [319;482]	355 [299;426]	379 [356;556]	399 [380;455]	379 [332;571]
Width of the PDV, ms (Me [MCD])	76 [67;88]	66 [57;89]	67 [66;76]	65 [56;84]	56 [50; 60]	68 [58; 89]
Partial acute ablation effect, n (%)	1 (14)	3 (11)	2 (33)†	1 (7)†	1 (17)	2 (11)
Presence of LEP on epicardial surface, n (%)	7 (100)	18 (67)	5 (83)	13 (93)	5 (83)	9 (50)
Amplitude of QRSvtin the third lead, (Me [MCD])	0.6 [0.4;1.1]*	1.3 [1.0;1.5]*	0.8 [0.4;1.5]	1.2 [0.8;1.4]	0.9 [0.5; 1.9]	0,6 [0.4; 0.8]
Ratio of scar[#] areas (Me [MCD])	0.87 [0.8;0.9]*	0.4 [0.2;0.7]*	0.79 [0.65;0.86]	0.5 [0.2;0.8]	0.8 [0.7;0.9]	0.5 [0.3; 0.9]
Beta-blocker dose (as % of target dose) (Me [MCD])	28.1 [25;100]	25 [6.25;62.5]	100 [62.5; 106.25] †	25 [15.6;50.0] †	50 [40.63; 87.5]	43.75 [34.38; 62.5]
History of substrate VT RFA[&] (Me [MCD])	1 [0.5; 1.5]	1 [0; 1]	1 [0.25; 1]	1 [0; 1]	2 [1; 3.8]††	0 [0; 1] ††

Note: [@] - under observation; ICMP - ischemic cardiomyopathy; ES - electrical storm; [$] - at the moment of surgical intervention; AHD - arrhythmia history duration; SR - sinus rhythm; PDV - pseudo delta wave; [#] - bipolar signals <0.5 mV on epi- and endocardial surfaces (mean); [&] - number of previous attempts; * - P<0.05 between group with relapse and group without relapse by 6 months; † - P <0.05 between the group with relapse and the group without relapse by 12 months; ††† - P <0.05 between the group with relapse and the group without relapse by 24 months.

Ventricular voltage map analysis

The area of the mapped epicardial surface naturally outweighs the endocardial surface, as both the left and right ventricular surfaces were mapped. When comparing the area of the low-amplitude myocardium, the ratio to the total area of the corresponding surface was considered rather than the absolute value. In the case of selective mapping of the area of suspected VT performance, such a voltage map was excluded from the calculation. The proportion (in %) of low amplitude myocardium in bi- and unipolar mapping on ENDO and EPI surfaces was assessed. In some cases, low amplitude zones were detected isolated on ENDO or EPI surfaces. Therefore, the ratio of areas of low-amplitude myocardium on two surfaces ranging from -1.0 to +1.0 was estimated using the formula (S1-S2)/(S1+S2), where S is the area of the low-amplitude zone on the corresponding surface.

ECG analysis

ECG analysis was performed in sinus rhythm and VT. The QRS complex width, QTc interval duration in sinus rhythm, QRS complex amplitude in standard leads, pseudo-delta wave width, internal tilt time in V1, and clinical VT cycle length were considered.

Surveillance and recording of arrhythmia recurrence, ablation efficacy criteria

Recurrent VT was recorded by the ICD/CRT-D survey, the results of Holter monitoring and the telephone interview. Patients from remote areas emailed protocols of implanted device interrogation and adjustment and Holter monitoring results. Interrogation and adjustment of implanted devices was routinely performed once a year or ahead of schedule in case of ICD activation or recurrence of arrhythmias. In the early postoperative period, a Holter monitoring was performed in the first three days, after which a Holter monitoring was recommended at 1 year or earlier if there were any complaints.

The effectiveness of ablation (no recurrence of sustained VT lasting more than 30 s and/or no shock to the implanted defibrillator) was evaluated at 6-, 12- and 24-month follow-up, and the absence of arrhythmia recurrence throughout the follow-up period after catheter ablation was also evaluated. VE ablation was also considered effective if the number of extrasystoles with dominant morphology per day was reduced by 90% of the baseline number of VEs according to Holter monitoring results.

Statistical analysis

Quantitative data are expressed as mean ± standard deviation and compared by t-test with normal distribution. For non-normal distributions, variables were reported as median with interquartile range (IQR) and compared using non-parametric tests (Mann-Whitney U, Fisher test). Categorical variables were expressed as percentages and absolute values. Kaplan-Meier curves were compared with Kraskell-Wallis or Cox tests. Proportional regression analysis was performed in two stages: univariate, assessing each of the clinical and electrophysiological parameters studied; then multivariate, including the parameters whose P values were closest to statistical significance (≤0.07) in the univariate analysis. The analysis was performed using STATISTICA 12.0 software (StatSoft, Tulsa, USA).

RESULTS

Clinical characteristics of the patients

The study group included 39 patients (mean age 49.5±15.7 years, 34 men and 5 women). In two patients, ventricular arrhythmias were detected by clinically significant VE. Both patients underwent MRI of the heart which showed no subepicardial/intramural fibrosis foci. However, based on the mapping results, the ineffectiveness of previous attempts at endocardial RFA and the presence of ECG evidence of epicardial ectopic outcome, the likely subepicardial location of the arrhythmogenic substrate was inferred. The clinical characteristics of the patients are listed in Table 1.

Twenty-one patients received amiodarone as AAT, 15 patients received amiodarone in combination with beta-blockers; 11 patients received beta-blockers only; 4 patients had taken amiodarone in the past but were discontinued because of the development of side effects. All patients were receiving beta-blockers at the time of the intervention. In one patient, the possibility of AAT was limited by clinically significant sinus bradycardia on a background of minimal doses of beta-blockers (nebivolol 2.5 mg/day); in one patient, therapy with a target dose of a beta-blocker was limited by the severe course of bronchial asthma. Eleven patients had a history of electrical storm, with 5 patients undergoing RFA for the substrate VT within 1-6 days of electrical storm development, 4 patients within 2 weeks and 2 patients within 2 months of electrical storm development.

In 16 patients, the combined endo-epicardial approach was used initially. In 22 patients, endocardial catheter ablation of VT had previously been performed with no or only transient effect (1 to 4 procedures in the history). EPI access was performed in 37 of 39 patients; subxiphoid puncture was unsuccessful in 2 patients. In only 5 patients was it not possible to perform endo- and epicardial ventricular analysis due to technical difficulties (no epicardial map, targeted mapping of the area of intended tachycardia exit point). Thus, the endo- or epicardial maps were analysed in 34 patients.

Mapping and ablation

Late potentials were recorded in 27 patients at EPI and ablation of these sites was performed. Effective ab-

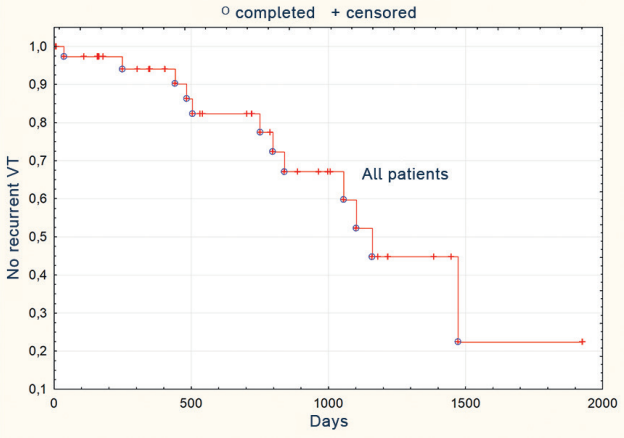

Figure 2. Kaplan-Meyer survival curve: freedom from ventricular tachycardia recurrence after endo-epicardial ablation in the total patient group.

lation in the acute phase was observed in 32 patients (no induction of any VT), and the procedure was partially successful in 4 patients. In 1 case, the procedure was discontinued due to the development of tamponade after an unsuccessful transseptal puncture. The results of volumetric chart analysis are shown in Table 2.

In 6 cases, the arrhythmogenic substrate was isolated to the surface EPI and in 3 patients the substrate was detected only on the ENDO surface. The relative "scar area" (amplitude < 0.5mV) on the epicardial surface predominated over the endocardial surface: 12.5% [IQR:4.2 - 18.9] versus 5.4% [IQR:1.3-13.4], P=0.04 (Figure 1a). Furthermore, myocardial areas with an amplitude of less than 0.5 mV predominated on the unipolar signal map compared to the bipolar signal map on the ENDO surface: only 3 patients had a calculated coefficient with a negative value (Figure 1b).

Complications

There were 2 cardiac tamponades: one as a result of failed transseptal puncture after epicardial mapping (required surgical correction) and the other a hemopericardium after endomyocardial biopsy at the end of the procedure (percutaneous drainage was performed). There were no complications directly related to epicardial access or ablation.

The long-term results

In the long-term postoperative period, a number of factors and their correlation with VT recurrence at 6, 12 and 24 months were assessed (table 3). Figure 2 shows the Kaplan-Meier curve for freedom from ventricular tachyarrhythmias after index ablation. Of note, after 1200 days of follow-up (40 months), recurrence of ventricular arrhythmia occurred in more than half of the patients.

When analysing the risk of recurrence of ventricular arrhythmias in patients with different etiologies VT, a significantly lower number of recurrences was observed in patients with postinfarction cardiosclerosis (figure 3a). A significantly higher risk of recurrence VT was observed in patients with electrical storm (figure 3b).

During the first 6 months, recurrent VT was reported in 7 (18%) patients (6 of whom had non-ischemic cardiomyopathies), with a higher number of recurrent VT in patients with an accompanying non-paroxysmal form of atrial fibrillation (AF) (43% recurrence versus 7% recurrence in patients without a persistent form, P=0.03).

Patients with recurrent VT had a higher value of normalized area EPI ratio < 0.5 mV to ENDO than in patients without relapse (figure 4), and the amplitude of the QRS complex on the background of VT in the third derivative was also smaller (table 3). In addition, the relative area of the "scar" on the EPI surface tended to be larger in the patients with relapse than in the group without relapse (median 18% versus 12.6%, at the edge of statistical significance, P=0.05).

By month 12 of follow-up, VT recurrence was more common in patients with partial acute effect than in those with complete effect (33% versus 7% of patients with relapse, P=0.02). Although bordering on statistical significance, there was a trend towards more frequent relapse VT in patients with reduced ejection fraction (EF). There was a trend that a higher dose of beta-adrenoblockers (as a percentage of the target dose at both study inclusion and 12-month follow-up) was associated with recurrence of VT at one year. At 24 months post-intervention, VT occurred more frequently in patients with the greatest number of previous RF substrate ablation in their history (table 3). There was also a significant direct correlation between clinical VT cycle length and the prevalence of scarring on the epicardial versus the endocardial surface (ρ=0.58).

Group of patients with postinfarct VT

In the group of patients with ischemic genesis of VT (15 patients), the mean age was 59.5±10.0 years, LV EF was 38% [IQR: 31-44%], VT cycle length was 400 ms [IQR: 368-474 ms], left atrial volume index was 51 ml/m^2 [IQR: 40-63].

There was a direct correlation between clinical VT cycle length and the ratio of scar area on unipolar and bi-

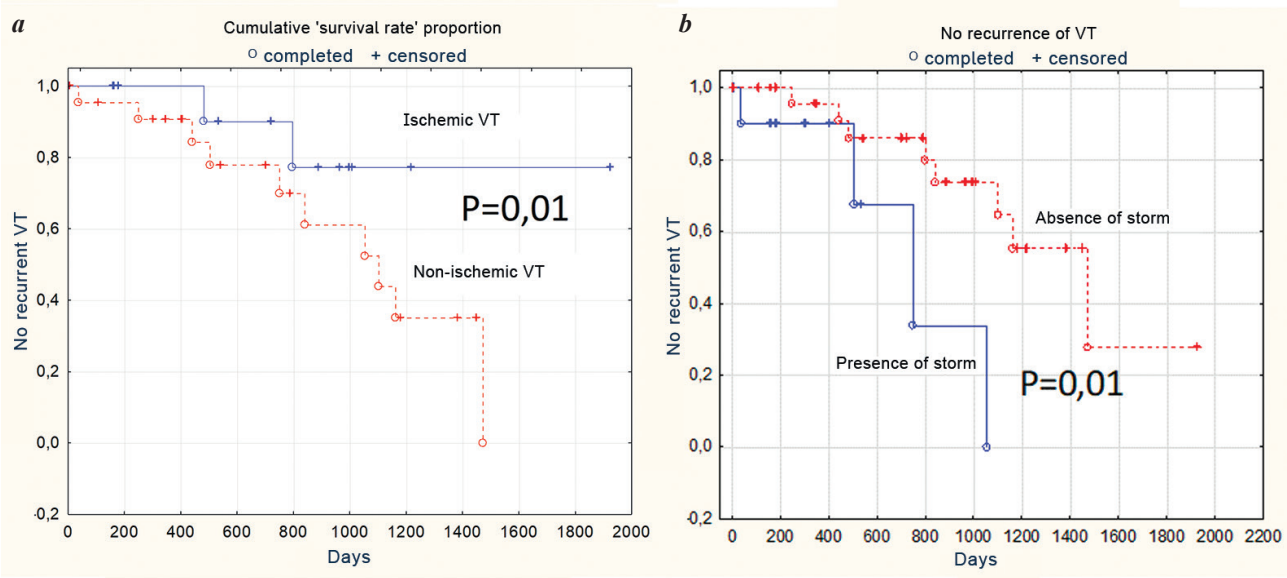

Figure 2. Kaplan-Meyer survival curve: a - risk of tachycardia recurrence in the group of patients with ischemic (postinfarction) and non-ischemic VTs (on the background of non-ischemic cardiomyopathy), Cox F-test - P=0.01; b - risk of tachycardia recurrence in the group of patients with and without electrical storm at the time of VT ablation, Cox F-test - P=0.01.

polar maps on the ENDO surface (ρ=0.57), and a significant inverse relationship between QRS complex width in sinus rhythm and the ratio of "scar" area on unipolar and bipolar maps on the ENDO surface (ρ=-0.66). There was a clear direct relationship between LV EF and the ratio of the "scar" area on EPI and the ENDO surface (ρ=0.58).

Group of patients with VT and non-ischemic cardiomyopathy

In the group of patients with VT and non-ischemic cardiomyopathy (15 patients), the mean age at the time of surgery was 45±15 years, LV EF 51±13%, VT cycle length 360 [IQR: 332-450] ms. There was a significant direct correlation between the VT cycle length and the ratio of scar area on EPI and END (ρ=0.57) and a significant inverse correlation between the width of the VT QRS complex and the ratio of scar area on EPI and END (ρ=-0.54). There was also a direct correlation between age at surgery, pseudo-delta wave width QRS, left atrial volume index and the ratio of "scar area" and transition zone on uni- and bipolar maps on the ENDO surface (ρ=0.67, ρ=0.64 and ρ=0.65, respectively). There was a significant inverse correlation between the amplitude of the QRS complex in the III lead in sinus rhythm and the ratio of the "scar" area of the uni- and bipolar maps on the ENDO surface (ρ=-0.7). Thus, the smaller the amplitude of the QRS complex in the third derivative in sinus rhythm, the larger the scar area was on the unipolar map compared to the bipolar map on the ENDO surface.

Predictors of long-term VT recurrent

The mean follow-up time was 22.8±15.2 months and ranged from 3 months to 5 years. Univariate Cox proportional hazards regression analysis identified the following parameters associated with VT recurrence with the highest statistical significance: LV EF < 35% (at the margin of statistical significance, P=0.07) and electrical storm (P=0.04) before ablation. Both parameters were tested in a multivariable model, with electrical storm being the only factor independently associated with VT recurrence at the time of ablation (hazard ratio 4.32; 95% CI: 1.06-17.48; P=0.04) (Table 4).

DISCUSSION

In a mixed group of patients undergoing combined ENDO and EPI mapping of the VT substrate, recurrent arrhythmias occurred predominantly in patients with non-ischemic etiology of disease, which may be explained by the large area of myocardial lesion and progressive myocardial remodelling in cardiomyopathies [9]. It is important to note that patients with an electrical storm, i.e. 3 or more episodes of VT within 24 hours that required cardioversion or ICD electrotherapy, have the highest risk of tachycardia recurrence over a median follow-up period of 22 months.

We hypothesize that this may be due to emergency ablation, which is often limited to a change in the tachycardia trigger or substrate. Subsequently, recurrences may be due to substrate areas outside the primary location of VT. Previous publications have demonstrated the high efficacy of catheter ablation in controlling electrical storms [10,11]; however, in this paper, the characteristics of remote VT recurrence were presented.

The differential efficacy of RFA in treating "substrate" VTs (in structural myocardial lesions) is in many ways due to the etiology of a substrate. For example, the recurrence rate after substrate ablation of postinfarct and non-ischemic VT is comparable in the acute phase but significantly different at 1 year - 57% and 40.7%, respectively [12]. The advantage of combined endo-epicardial ablation for non-ischemic cardiomyopathy over isolated endocardial ablation has been demonstrated in a number of studies, but the effectiveness of ablation is still far from ideal, ranging from 59 to 85% [4, 13, 14].

In our study, an association was found between persistent/permanent AF and recurrent VT within the first 6 months. This could be due to both higher sympathetic nervous system tone and the degree of progression of cardiomyopathy involving the atrial myocardium in the pathological process. The association of AF with an increased risk of AF has been reported in the literature as an independent predictor of death in patients with AF [15, 16].

Interestingly, a higher risk of AF recurrence during the first 6 months after surgery was found in patients with a lower QRS amplitude compared to AF in the third lead. The other leads showed no significant difference in QRS ampli-

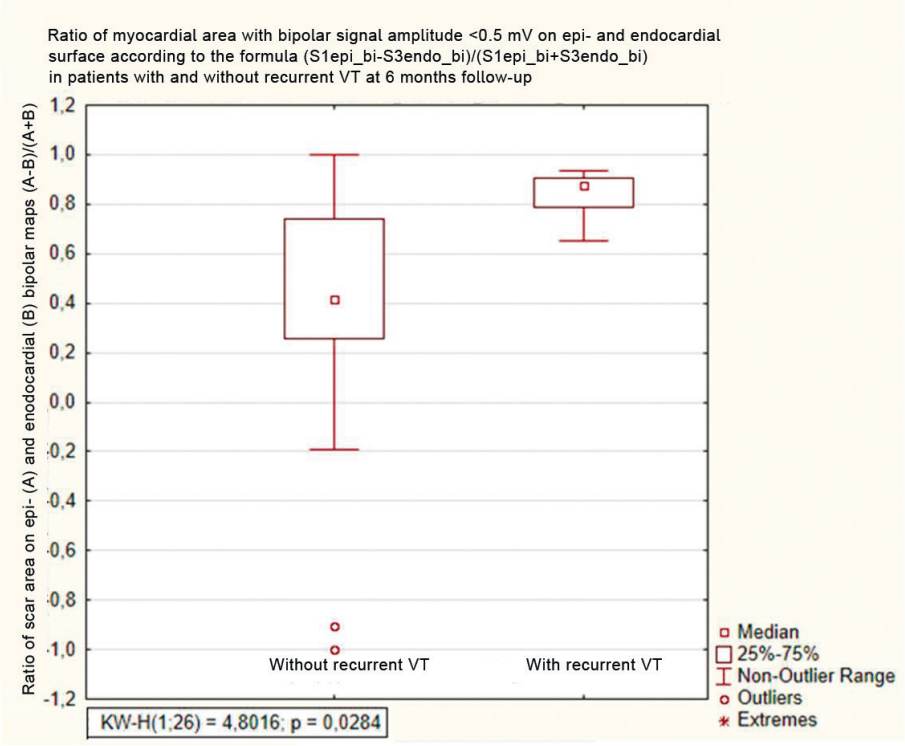

Figure 3. Graph showing the differences in the normalised ratio of low-amplitude signal (<0.5 mV) at epi- and endocardial surface in patients with recurrent VT after 6 months and without it; Kruskal-Wallis test, P=0.02.

tude between the subgroups with and without recurrence VT. We suggest that low QRS amplitude may indicate the origin of VTs from septal areas that are more refractory to ablation and may also be the result of widespread ventricular myocardial damage predisposing to multiple VTs. The association between QRS width of VT and recurrence of VTs after ablation has been shown previously, highlighting the prognostic importance of surface ECG parameters [17] in patients with structural myocardial damage. Of note, in agreement with previous studies in our cohort, recurrence of VT at 6 months was more frequent in patients with longer QTc in sinus rhythm [18]. We also found correlations between ECG signs and features of arrhythmogenic substrate. For example, we found a negative correlation between the internal deviation time in V5 in sinus rhythm and the ratio of scar area on the ENDO surface on nonpolar and bipolar maps; and a negative relationship between the amplitude of the QRS complex in the I lead on the background of VT and the ratio of scar area to transition zone on the ENDO surface. In our opinion, this finding can be explained by a higher degree of myocardial damage. In the group of patients with non-ischemic cardiomyopathy, there was a pronounced direct correlation between the width of the pseudo-delta waves and the ratio of scar area on the ENDO surface, which is consistent with previously proposed algorithms for diagnosing EPI VT [19]. We found a correlation between the length of the VT cycle and the width of the QRS complex in sinus rhythm and the area of arrhythmogenic substrate on the EPI surface. Thus, the greater the width of the QRS complex or the greater the subepicardial myocardial lesion, the shorter the cycle length of VT, i.e. the "faster" the tachycardia. Based on the results of the correlation analysis, it can be assumed that patients with a larger area of subepicardial myocardial lesions are characterized by a lower amplitude of the QRS complex of ventricular tachycardia and a higher probability of developing a VT recurrence within the first 6 months after the procedure.

At the same time, in the group of patients with postinfarction VT the opposite relationship was observed: with transmural/subepicardial scar there was a tendency for a longer VT cycle length, i.e. the tachycardia was "slower", which correlates with the findings of other investigators [20]. These differences could be due to the different "geometry" of the scar, the different homogeneity, and the area of the "border zone", which influences the length of the re-entry loop.

Also noteworthy was the fact that 24 months after ablation of the arrhythmogenic substrate, more recurrent VT were recorded in patients with a greater number of previous RFA attempts in their history. This observation could be due to a more severe progressive course of the disease and greater damage to the myocardium, which in turn necessitated repeated ablation of the arrhythmogenic substrate.

The area of the mapped scar on the bipolar and unipolar map on the ENDO surface is considered as a criterion for assessing the prevalence of the arrhythmogenic substrate and as a criterion for its probable deeper location. Muessigbrodt et al [21] showed that the ratio of low-amplitude myocardial areas on bipolar and unipolar maps was ≥0.58 was a predictor of VT recurrence; they also found a correlation between scar area on the unipolar EPI map and on the bipolar EPI map. A.Berruezo et al [22], in a study of a larger group of patients, showed that patients with a ratio of low amplitude myocardium on the ENDO surface on bipolar and unipolar maps > 0.23 have a small substrate area on the EPI surface and predominantly require endocardial ablation.

The area of low-amplitude myocardium on the unipolar ENDO map is a strong independent predictor of VT recurrence [23], which we also considered when calculating areas. The presence of low-amplitude myocardium on the unipolar ENDO map, while absent on the bipolar map, again suggests an intramural/subepicardial location of the arrhythmogenic substrate [24]. In our work, we used the original area ratio formula because cases of isolated ENDO or EPI localization of low-amplitude myocardium were observed. A simple ratio would have led to the problem of dividing the area by zero and thus excluding part of the observations from the analysis. Therefore, the prevalence of low-amplitude areas was estimated in a normalized range of -1 to +1. In our study, patients with a predominant epicardial arrhythmia substrate compared with the endocardial substrate had a higher probability of recurrent VT within the first 6 months after surgery, which correlates with the results of the above-mentioned publications.

In our study, recurrent VT at 12 months occurred more frequently in patients with partial or no acute ablation effect and in patients with non-ischemic cardiomyopathy, which is consistent with the results of the published papers [25, 26, 27]. A meta-analysis of 24 observational studies found that non-inducible postoperative VT is a predictor for the absence of VT recurrence in patients with non-ischemic cardiomyopathy, and combined endo-epicardial ablation also reduces the risk of VT recurrence in this patient group. Thus, our results confirm the data available in the literature [12, 28, 29].

We hypothesise that the observed correlation between higher doses of beta-blockers and recurrence VT after 12 months is due to a more severe disease course in these patients, with frequent recurrence VT before and after ablation.

In the group of patients with non-ischemic cardiomyopathy, more

Table 4.

Univariate and multivariate analysis of VT predictors

	Effect under study	Risk ratio	Lower level 95% CI	Upper level 95% CI	P
Univariant model					
Electric storm	Yes	4.70	1.20	18.35	0.03
LV EF <35%	Yes	3.88	0.87	17.27	0.07
Multivariate model					
Electric storm	Yes	4.32	1.06	17.48	0.04
LV EF <35%	Yes	3.39	0.71	16.02	0.12

Note: CI is the confidence interval.

recurrences occurred 12 months after ablation in patients with reduced LV EF as well as with reduced right ventricular contractility. In 2017, the results of a prospective multicentre trial of combined endo-epicardial ablation in patients with ARVC were published, with left ventricular myocardial damage being a predictor of VT recurrences [22].

Limitations of the study

Our observational study was limited primarily by the size of the study population and by the duration of follow-up of patients in the postoperative period. Among the patients with ischemic VT studied, some had previously undergone coronary bypass surgery, which precluded epicardial access. Another major limitation was the impossibility of performing MRI in preoperative patients because they were already wearing implanted devices (ICD/CRT-D). Assessment of VT morphology and determination of epicardial exit criteria was also not always possible because VT was terminated by ICD electrotherapy. Another limitation of the work is that some patients had no history of VT, clinically significant arrhythmia was represented by frequent VE. The analysis of voltage maps excluded maps with incomplete reconstruction of the surface of the examination chamber. Another limitation of the study was the lack of analysis of the localization of low-amplitude signal zones; therefore, no analysis of the relationship between myocardial zonality and recurrent VT was performed.

CONCLUSION

Thus, clinical and electrophysiological factors associated with the recurrence of ventricular tachyarrhythmias at different follow-up periods after endo-epicardial ablation were identified in a heterogeneous group of patients. The presence of an electrical storm is an independent predictor of recurrence of ventricular tachyarrhythmia in follow-up periods up to 5 years after ablation.

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