EPICARDIAL VOLTAGE MAPPING IN PATIENTS WITH POSTINFARCTION VENTRICULAR TACHYCARDIA: A PILOT STUDY

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Introduction. Radiofrequency ablation (RFA) is an established treatment of post-myocardial infarction ventricular tachycardia (VT). Endocardial VT ablation can be insufficient for VT termination when the scar is intramural/epicardial. Purpose: to assess the extent of epicardial electrophysiological VT substrate in patients with remote myocardial infarction.

Materials and methods. Thirteen patients with sustained postinfarction VT, who signed an informed consent, were included into the study. All patients underwent full clinical evaluation. Electroanatomical voltage bi- and unipolar mapping of endocardial and epicardial surfaces was performed. Maps were evaluated for the presence of low-voltage areas and local abnormal ventricular activity (LAVA). RFA was performed at LAVA sites. The end-point of the procedure was scar LAVA abolition and VT noninducibility (procedure success). VT recurrence was detected using an implantable cardioverter-defibrillator and/or ECG monitoring.

Results. Epicardial access was successful in 12 patients. Epicardial ablation was performed at a first procedure in 7 patients, 4 patients had a history of previous endocardial ablation. Epicardial LAVA sites were detected in 9 patients. Endocardial and epicardial arrhythmogenic substrate localization coincided in 8 patients. One patient had only epicardial scar, 1 patient had only septal endocardial scar. In one patient LAVA sites had different localizations on epicardial and endocardial maps. Acute ablation success was noted in 12 patients.

Conclusion. In our patient group transmural scar and epicardial electrophysiological arrhythmogenic substrate was detected in 82% of cases. Isolated endocardial ablation may be unsuccessful, in such cases epicardial mapping and ablation might be useful.

Key words: postinfarction cardiotosclerosis; ventricular tachycardia; radiofrequency catheter ablation; endocardial access; epicardial access; scar tissue; late potentials; mapping

Conflicts of interest: E.N. Mikhaylov and D.S. Lebedev report receiving speaker and consultation honoraria from Biosense Webster; other authors report no conflicts of interest.

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The prediction of arrhythmogenic substrate subepicardial location is frequently limited. Magnetic resonance imaging (MRI) is a useful tool in delineation of the depth and extent of a scar, but its usability is limited in patients with an implanted ICD/CRT-D. Moreover, ECG criteria for epicardial VT exit site prediction have less accuracy in the presence of a postinfarction scar.

Theoretically, a combined endo-epicardial approach can improve the long-term efficacy of VT catheter ablation in some patients.

The aim of our pilot study was to evaluate the prevalence of electrophysiologically mapped epicardial VT arrhythmogenic substrate, and the necessity of epicardial ablation in patients with remote myocardial infarction.

**MATERIAL AND METHODS**

Between 2015 and 2018 165 patients with structural heart disease (coronary heart disease, dilated cardiomyopathy, arrhythmogenic right ventricle cardiomyopathy, infiltrative heart disease) were referred for VT catheter ablation at the Almazov Medical Centre, and 59 had remote myocardial infarction. The study group comprised patients who signed informed consent for epicardial access (study and agreement form were established by ethical committee of the Almazov NMRC, protocol №181 14.12.2015). One patient included into study group was operated in the Sukanov Federal Centre for Cardiosurgery.

Inclusion criteria were the following: a postinfarction scar detected by transthoracic echocardiography or MRI and ECG-criteria of myocardial scar, the presence of sustained VT registered on ECG or during ICD follow-up, ineffective antiarrhythmic drug therapy, the absence of indications for revascularisation or it’s impossibility, signed informed consent for epicardial access. Exclusion criteria: myocarditis, previous cardiothoracic surgery with pericardial involvement at the Almazov Centre, and 59 had remote myocardial infarction. The study group comprised patients who signed informed consent for epicardial access (study and agreement form were established by ethical committee of the Almazov NMRC, protocol №181 14.12.2015). One patient included into study group was operated in the Sukanov Federal Centre for Cardiosurgery.

Standard clinical evaluation included: 12-lead rest ECG, TTE, 24-hour Holter monitoring, coronary angiography, ICD/CRT-D check-up. Heart MRI was performed in all patients (cut-off values 0.5-1.5 mV for bipolar signals), then epicardial electroanatomical mapping was performed with the same cut-off values. For unipolar maps cut off values 5.0-9.0 were used. Sites with local registration of late potentials, fragmented and double potentials were tagged on maps. When a scar was localized in the interventricular septum and VT remained inducible despite endocardial ablation, then RV mapping and substrate ablation was performed. Electroanatomical voltage mapping was performed during sinus rhythm or RV pacing. A scar was defined when myocardial signal amplitude was <0.5 mV, the intact myocardium was defined when myocardial signal amplitude was >1.5 mV.

The identification of VT exit site location was performed by both, activation and pace-mapping, and according to effective ablation. Arrhythmogenic substrate surface area was evaluated on bipolar and unipolar voltage maps. Protocol of EP study for VT induction included programmed stimulation up to three extrastimuli from the RV apex, RV outflow tract, from LV, and overdrive burst pacing was performed when VT was not induced by programmed stimulation. Pace-mapping of conduction channels in the scar, entry and exit zones VT was performed. When induced VT was haemodynamically stable, entrainment-mapping performed. Selective coronary angiography used before epicardial ablation in order to de-

**Endocardial and epicardial accesses, electrophysiological study, catheter ablation**

Procedure was provided in an EP laboratory under general anesthesia. Vascular accesses were performed using the Seldinger technique: to the right femoral artery and right femoral vein. Percutaneous pericardial access was obtained by a subxyphoid puncture. The access technique was described in detail previously [13]. The long Preface Multipurpose sheath (Cordis,USA) was introduced into the pericardial space. Then interatrial septum punctured under fluoroscopic guidance using the Brockenbrough needle (BKR-1, St. Jude Medical, USA). The transseptal sheath (Preface Multipurpose, Cordis, USA) was positioned in the left atrium and left ventricle. Therefore, a double endocardial access to LV was used. Heparin was administered as intravenously 80-100ME/kg with further ACT monitoring, with a target value >250 sec. A quadripolar diagnostic catheter (Webster, Biosense Webster, USA) was positioned in the RV apex. The procedure was performed under the guidance of the three-dimensional navigation system (Carto 3, Biosense Webster, USA). The NaviStar Thermocool or SmartTouch (Biosense Webster, USA) ablation catheters were used for mapping and ablation. The multipolar Pentaray catheter (Biosense Webster, USA) was used for epicardial mapping in two cases. Endocardial three-dimensional LV reconstruction and electroanatomical voltage mapping was performed in all patients (cut-off values 0.5-1.5 mV for bipolar signals), then epicardial electroanatomical mapping was performed with the same cut-off values. For unipolar maps cut off values 5.0-9.0 were used. Sites with local registration of late potentials, fragmented and double potentials were tagged on maps. When a scar was localized in the interventricular septum and VT remained inducible despite endocardial ablation, then RV mapping and substrate ablation was performed. Electroanatomical voltage mapping was performed during sinus rhythm or RV pacing. A scar was defined when myocardial signal amplitude was <0.5 mV, the intact myocardium was defined when myocardial signal amplitude was >1.5 mV.

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**Figure 1. Patient №5. Electroanatomical voltage map with the endocardial scar only in the interventricular septum. RAO projection. The bipolar substrate area dominates over the unipolar map. Bipolar map cut off values 0.5-1.5 mV; unipolar cut off values 5.0-9.0 mV. Red color indicates the myocardial signal amplitude <0.5 mV; purple color indicates the myocardial signal amplitude >1.5 mV.**
fine the proximity of coronary artery and to prevent their damage. Safety distance for ablation from the coronary artery was considered about 10 mm. RF energy was delivered at areas with late potentials, fragmented potentials until their abolishment or decreased amplitude by 85%. Entry and exit VT sites and conducting channels were ablated. When a VT cycle was mapped, the critical isthmus of tachycardia was ablated.

RF energy 40-50W was used, application duration - 10-40 sec, ablation catheter tip irrigation was 30 ml/min. Ablation was considered effective if late potentials and fragmented potentials vanished and loss of stimulation capture was achieved (amplitude of stimulation 10 V; time duration 1 ms, cycle length - 500 ms). After substrate ablation programmed stimulation for VT induction was performed. Acute procedure effectiveness was considered when VT was non-inducible.

The mean follow-up period was 20.2±16.1 months (from 2 to 46 months). VT recurrences were documented by ICD/CRT-D regular check-ups, and 24-hour Holter monitoring.

Statistical analysis
Continuous data with normal distribution were reported as mean ± standard deviation, compared by T-test. Categorical variables were expressed using non-parametric statistics, median with interquartile range (IQR). Mann-Whitney test and Fischer exact test were used for comparison non-parametric variables. Results were considered significant with a P-value <0.05. Statistical analysis was provided using STATISTICA 6.0 (StatSoft, Tulsa, USA).

RESULTS

Patient clinical characteristics
The study group included 13 patients, the mean age 58.1±9.8 years (12 men). Clinical characteristics are presented in table 1. Antiarrhythmic drug (AAD) therapy with a combination of amiodarone and beta-blockers was used in 9 patients, in 3 patients amiodarone was discontinued because of complications, and only beta-blockers were prescribed. In one patient without ICD AAD therapy was limited by symptomatic sinus bradycardia (nebivolol 2.5 mg per day). In nine patients the combined endo-epicardial access was used as a first-line approach. Endocardial ablation of post-infarction VT was previously performed in four patients, procedures were ineffective or with temporary effect (1-2 attempts). In one patient with two previous ineffective attempts of endocardial VT ablation three consecutive epicardial procedures were performed due to clinical VT recurrence. Final ablation was successfully performed using bipolar ablation because of intramural location of the tachycardia critical zone. Transmural postinfarction scar location was detected by cardiac MRI in two patients. Epicardial access was obtained in 12 of 13 patients, in one patient it was unsuccessful, presumably because of pericardial adhesions on diaphragmatic surface of the heart. In one case comparative analysis of endocardial and epicardial electroanatomical voltage maps was not performed because of a technical failure. Thus, comparative analysis of endo- and epicardial voltage maps was performed in 11 patients.

Mapping and ablation
Electroanatomical voltage maps were created with the color threshold 5-10 mm (when an ablation catheter was used for mapping) and the color threshold 2 mm (when the multielectrode catheter was used for mapping). The mean epicardial mapped surface area prevailed above endocardial surface area because epicardial mapping included evaluation of both ventricles.

The prevalence of a median substrate surface area of unipolar voltage maps over bipolar voltage maps by 3.7 times was noted on the endocardial surface (45.8

Figure 2. Patient №1. Endocardial and epicardial maps (bipolar and unipolar). Posterior projection. Bipolar map cut off values 0.5-1.5 mV, unipolar cut off values 5.0-9.0 mV. Pink tags represent sites with late potentials. The epicardial abnormal electrogram area prevails above the endocardial area. The unipolar low voltage area is more extensive than bipolar. The wide inferior myocardial involvement is seen on the unipolar map, while the bipolar map shows mainly lateral wall involvement.
(IQR:17.1;86.5) cm² vs 11.8 (IQR: 2.0;31.6) cm²; p=0.035) (Table 2). Only in one patient the bipolar substrate area dominated over the endocardial unipolar map (by 2.5 times). There was no any abnormal electrical activity or low voltage signals registered on the epicardial surface. In one case there was no low-voltage substrate on the endocardial bipolar map, and was hardly represented on the unipolar map.

The median epicardial arrhythmogenic substrate area on the unipolar map prevailed over the same on the bipolar map by 2.3 times: 107.7 (IQR: 84.3; 168.9) cm² versus 46 (IQR: 15.9; 55.5): p=0.041 (Figure 1). Low-voltage areas were not found on the epicardial surface, and was hardly represented on the endocardial unipolar map. In one case there was no low-voltage substrate on the epicardial bipolar map, and was hardly represented on the unipolar map.

Area of epicardial low voltage registration on the epicardial surface was wider than on the endocardial unipolar map dominated over the endocardial unipolar map (by 2.5 times). Only in one patient the bipolar substrate area (IQR:17.1;86.5) cm² prevailed over the same on the unipolar map. We found no correlation between the substrate area on the endocardial and epicardial surfaces.

Late potentials and fragmented potentials were registered in nine patients: in seven cases - on the endocardial surface, and in six cases- on the epicardial surface. The areas of late potential registration in study patients is presented in table 2.

Two VT morphologies were induced before ablation in 4 patients, in 4 patients only one clinical VT morphology was induced, and in 4 patients VT was non-inducible.

Endocardial and epicardial scar location coincidence was noted in 8 cases. In one patient a scar was detected on the epicardial surface only. In one patient the postinfarction scar was identified endocardially only in the interventricular septum. In one case, there were different endocardial and epicardial scar localizations: an endocardial scar was identified on the inferior and septal walls, an epicardial low voltage activity was detected on the lateral RV wall.

Fragmented potentials and late potentials were detected in 9 patients, RF ablation was performed in these areas in all cases.

The mean procedure time duration was 228±62 minutes, the mean fluoroscopy time duration was 45±21 minutes. In 12 patients VT was non-inducible at the end of procedure, in two of them ventricular fibrillation was induced by an aggressive stimulation protocol. Thus, in 12 of 13 cases complete acute effect was achieved. In one patient the procedure was discontinued because of haemopericardium occurrence.

**Complications**

In one patient with unsuccessful epicardial access (there were adhesions on the inferior wall of LV), an attempt of transseptal puncture was performed with cardiac perforation and haemopericardium, which required surgical correction. There were no complications, associated with the epicardial access itself.

**Long-term results**

The mean follow-up period was 19.3±17.6 months. Two of 11 patients, who were operated using the epicardial access, were lost to follow-up. In three patients, the follow up period was less than 6 months; VT recurrence was not evident during this period of observation. In one patient with multiple ablation sesions (6 procedures in total) VT recurrence was not registered during 3-years follow-up. In one patient VT recurrence registered 1 year after the procedure: 11 VT paroxysms with ATP therapy registered and 1 shock because of acceleration VT to VF after ATP therapy at two years. Two of four patients with previous ineffective endocardial VT ablation were free from VT recurrence after combined endo-epicardial VT ablation.

**DISCUSSION**

VT substrate can be localized on both endocardial and epicardial surfaces in patients with remote myocardial infarction. In our pilot study among 11 patients with sustained postinfarction VT, electrophysiological VT substrate was identified epicardially in 82% of cases. Some patients with previous ineffective attempts endocardial ablation VT substrate epicardial mapping and

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**Table 1.**

| Parameter                        | Value          |
|----------------------------------|----------------|
| Remote myocardial infarction, n (%) | 13 (100)       |
| Hypertension, n (%)              | 10 (76.9)      |
| Diabetus melitus, n (%)          | 3 (23)         |
| COPD, n (%)                      | 1 (7.7)        |
| Atrial fibrillation, n (%)       | 6 (46.2)       |
| ICD, n (%)                       | 9 (69.3)       |
| CRT-D, n (%)                     | 2 (15.4)       |
| Patients with remote SCD, n (%)  | 10 (76.9)      |
| ICD shock, n (%)                 | 4 (30.8)       |
| External shock, n (%)            | 7 (53.8)       |
| Mean LV EF, %                    | 38.8±10.6      |
| Mean LV EDV, ml                  | 193.8±73.7     |
| Mean LV ESV, ml                  | 125.3±54.9     |
| Coronary angiography             |                |
| Without HSS, n (%)               | 9 (69.3)       |
| PTCA and stenting, n (%)         | 5 (38.5)       |
| Repeated PCI, n                   | 2 of 5 patients|
| CABG, n (%)                      | 0              |
| TTE, scar localization           |                |
| Inferior wall, n (%)             | 10 (76.9)      |
| Lateral wall, n (%)              | 7 (53.8)       |
| Apex, n (%)                      | 2 (15.4)       |
| Septum, n (%)                    | 4 (30.8)       |
| Anterior wall, n (%)             | 2 (15.4)       |

Description. COPD - chronic obstructive pulmonary disease, ICD - implantable cardioverter-defibrillator, CRT-D - cardiac resynchronization therapy defibrillator, LV EF - left ventricle ejection fraction, LV EDV - left ventricle end-diastolic volume, LV ESV - left ventricle end-systolic volume, HSS - hemodynamically significant stenosis, PTCA - percutaneous transluminal coronary angioplasty, PCI - percutaneous coronary intervention, SCD - sudden cardiac death, TTE - transthoracic echocardiography, CABG - coronary artery bypass grafting.
Voltage mapping

| Patient number | Endocardial surface |   | Epipcardial surface |   |
|----------------|---------------------|---|---------------------|---|
|                | Bipolar signals     | Unipolar signals | Bipolar signals | Unipolar signals |
|                | <0.5 mV | >0.5-<1.5 mV | LP | <0.5 mV | >0.5-<1.5 mV | LP | <0.5 mV | >0.5-<1.5 mV |
| 1  | 8.6 | 6.0 | 25.2 | 45.8 | 14.6 | 54.6 | 315.2 | 0 | 417.1 | 23.7 |
| 2  | 2.3 | 0 | 0 | 2.6 | 25.0 | 46.0 | 2.0 | 49.8 | 77.5 | 64.4 |
| 3  | 1.7 | 7.5 | 4.1 | 27.8 | 22.9 | 49.9 | 26.1 | 19.8 | 122.1 | 45.0 |
| 4  | 11.8 | 12 | 44.1 | 81.6 | 23.7 | 19.7 | 29.7 | 0 | 574.1 | 45.2 |
| 5  | 21.9 | 23.8 | 0.7 | 8.6 | 49.6 | 0 | 37.3 | 0 | 0 | 17.1 |
| 6  | 0 | 0 | 3.5 | 2.0 | 7.6 | 56.4 | 35.0 | 47.2 | 125.0 | 40.1 |
| 7  | 66.8 | 63.5 | 18.3 | 88.9 | 55.9 | 124.5 | 650.7 | 29.3 | 212.8 | 569.7 |
| 8  | 344.5 | 91.2 | 22.4 | 84.1 | 93 | 166.8 | 439.4 | 30.5 | 91.1 | 487.9 |
| 9  | 28.6 | 22.3 | 0 | 109.2 | 195.7 | 19.5 | 39.7 | 0 | 99.6 | 19.4 |
| 10 | 47.3 | 172.6 | 52.7 | 152.2 | 159 | 2.4 | 19.5 | 19.5 | 107.5 | 491.8 |
| 11 | 0 | 0 | 0 | 25.5 | 64.4 | 12.3 | 32.1 | 9.3 | 35.4 | 42.9 |
| Median (1 и 3 quartile) | 11.8 | 12.0 | (2.0; 31.6) | 4.1 (0.4; 43.7) | 46.8 (17.3; 86.5) | 49.6 (23.3; 78.7) | 19.6 (12.3; 53.8) | 14.4 (0; 64.4) | 95.4 (52.2; 120.6) | 54.8 (40.8; 460.5) |

Description. LP - late potentials, * - p<0.05.
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