https://doi.org/10.35336/VA-2022-1-07

PERCUTANEOUS EPICARDIAL MAPPING AND ABLATION OF THE VENTRICULAR TACHYCARDIA SUBSTRATE IN A PATIENT AFTER PERICARDIOTOMY: CASE REPORT

K.A.Simonova[1], M.A.Naymushin[1], A.V.Kamenev[1], T.A.Lyubimtseva[1], V.S.Orshanskaya[1], Yu.V.Miller[1], D.S.Lebedev[1, 2], E.N.Mikhaylov[1, 2]

[1]Almazov National Medical Research Centre, Russia, Saint-Petersburg, 2 Akkuratova str;
[2]Saint-Petersburg Electrotechnical University, Russia, Saint-Petersburg, 5 Professor Popov str.

We present a case of successful percutaneous epicardial access in patients with non-ischemic cardiomyopathy with limited mapping and ablation of the ventricular tachycardia substrate on the epicardial surface.

Key words: ventricular tachycardia; epicardial mapping; epicardial ablation; pericardiotomy; pericardial adhesions

Conflict of Interests: nothing to declare.
Funding: this work was supported by the Ministry of Healthcare of the Russian Federation (registration number 121031100306-6).
Received: 23.10.2021 **Revision Received:** 01.12.2021 **Accepted:** 09.12.2021
Corresponding author: Simonova Kseniia, E-mail: simonova_elphys@mail.ru

K.A.Simonova - ORCID ID 0000-0003-2423-1041, M.A.Naymushin - ORCID ID 0000-0003-2685-623X, A.V.Kamenev - ORCID ID 0000-0001-8780-2601, T.A.Lyubimtseva - ORCID ID 0000-0002-8651-7777, V.S.Orshanskaya - ORCID ID 0000-0003-4287-9597, Yu.V.Miller - ORCID ID 0000-0003-3416-0910, D.S.Lebedev - ORCID ID 0000-0002-2334-1663, E.N.Mikhaylov - ORCID ID 0000-0002-6553-9141

For citation: Simonova KA, Naymushin MA, Kamenev AV, Lyubimtseva TA, Orshanskaya VS, Miller YuV, Lebedev DS, Mikhaylov EN. Percutaneous epicardial mapping and ablation of the ventricular tachycardia substrate in a patient after pericardiotomy: case report. *Journal of Arrhythmology.* 2022;29(1): 45-49. https://doi.org/10.35336/VA-2022-1-07.

Epicardial access is used for mapping and ablation of ventricular tachycardia (VT) after an ineffective endocardial procedure or initially when there is evidence of a subepicardial VT substrate (which may be indicated by the etiology of the heart disease, ECG criteria for clinical VT, subepicardial contrast latency on magnetic resonance imaging, endocardial bipolar and unipolar mapping results). In patients with a history of pericardiotomy, epicardial access is extremely difficult and mapping and ablation options are limited due to adhesions in the pericardium.

The aim of our work is to present a case of successful percutaneous epicardial access in a patient with nonischemic cardiomyopathy with limited mapping and ablation of the VT substrate on the epicardial surface.

A 54-year-old patient with no history of structural heart disease was admitted for repeat catheter ablation of the substrate of paroxysmal VT (Fig. 1 a,b), which was refractory to medical therapy and accompanied by unstable hemodynamics.

Examination revealed no coronary artery changes, diagnosed dilatation of both atria, normal left (LV) and right ventricular (RV) systolic function. ECG showed prolongation of PQ interval, atrial fibrillation (CHA_2DS_2-VASc - 1 point, HAS-BLED - 0 point) for more than a year ambulatory, for which the patient received anticoagulant therapy with apixaban at a dose of 5 mg b.i.d. A clinical VT with a ventricular contraction rate of 200 bpm recorded on ECG did not fully meet the criteria for epicardial localization: 2 of 4 possible criteria in the stepwise algorithm (QS in leads II, III, avF, maximum deflection index, internal deviation time index - 0.81, pseudo-delta wavelength 90 ms, no q in lead I) were met [1]. Delayed contrast magnetic resonance im-

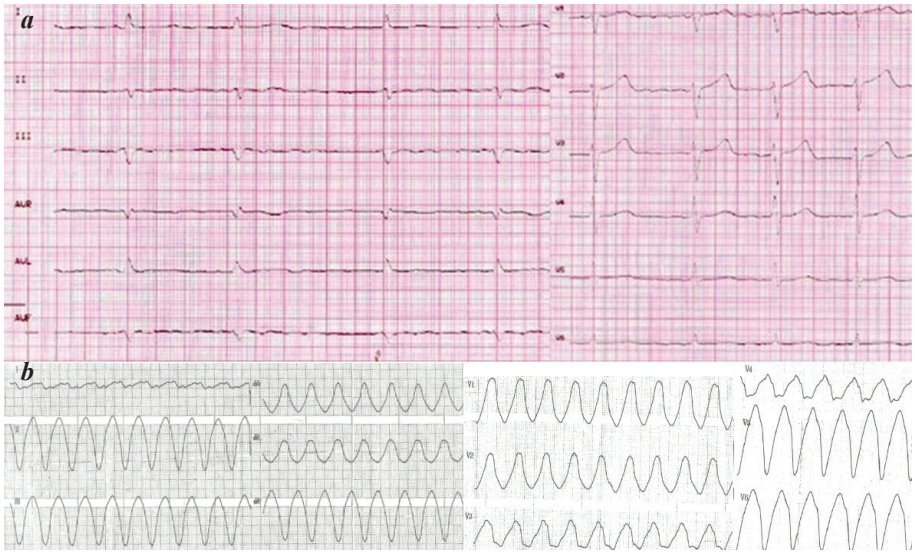

Fig. 1. ECG of the patient: a - atrial fibrillation, b - ventricular tachycardia.

aging confirmed atrial dilatation in both atria and showed increased trabecularity in the apex and medial-apical regions of the lateral and posterior LV walls. There was no evidence of subepicardial localization of the arrhythmia substrate.

The first attempt at substrate ablation from VT was performed 2 months ago. Endocardial mapping of the LV and RV myocardium showed no zones of low amplitude or fragmented activity on analysis of bipolar and unipolar voltage maps (CARTO 3 navigation system, Biosense Webster: SmartTouch ThermoCool Ablation and Cartridge Catheter, Biosense Webster, USA). For LV access, atrial transseptal puncture was performed, and mapping was anterograde. Programmed ventricular stimulation induced a clinical VT with a cycle of 375 ms accompanied by a drop in blood pressure that prevented detailed activation mapping. Stimulation mapping of the LV revealed the greatest overlap of the stimulated QRS complex with the clinical VT at the border between the apex and the lateral wall of the LV (95% overlap, PASO module used, Biosense Webster), which did not correspond to the 'ideal' tachycardia exit point mapping. With evidence of a likely subepicardial VT output, radiofrequency applications were performed in this area on the endocardial surface (40W; up to 90s). Further mapping attempts resulted in mechanical perforation of the LV wall with the ablation catheter. Because of the nature of the perforation, a decision was made to correct it surgically and perform a median sternotomy, pericardiotomy, removal of the ablation catheter that had penetrated the myocardium, and suturing of the LV defect. Surgical ablation was not possible during the emergency sternotomy because no electrophysiological equipment was available for open-heart radiofrequency (RF) or cryoablation. The patient was discharged on amiodarone saturation therapy without spontaneous episodes VT.

Two months later, on treatment with amiodarone in combination with a beta-blocker, VT recurred and the patient was referred for repeat catheter ablation of the arrhythmia substrate.

After cardiologists, electrophysiologists, and cardiovascular surgeons discussed intervention tactics with the patient, it was decided to attempt epicardial ablation under general anesthesia via a punctured subxiphoid approach in the presence of a cardiovascular surgeon and to be prepared for open surgery should complications arise. Despite a history of pericardiotomy, a minimally invasive approach to epicardial mapping seemed warranted.

Percutaneous access to the pericardial space was achieved by subxiphoid puncture. The method of access was described in detail in a previous publication [2].

Attempted pericardial punctures showed adhesions between the parietal and visceral pericardial leaflets, and inadvertent puncture of the RV was observed (without consequences in follow-up). On repeated puncture attempts on the diaphragmatic surface of the heart, limited guidance with a diameter of 0.035 inch (Emerald, Cordis, USA) between the pericardial leaflets was observed against a background of bolus injection of a small amount of contrast medium (Omnipack-300, GE Healthcare, Ireland). Partial separation of the pericardial leaflets was achieved by careful movements of the guidewire under fluoroscopic control and then by alternating 5-10 ml boluses of contrast medium and saline through the soft dilator of a 6F vascular introducer (Avanti+, Cordis, USA) (Figs. 2, 3). An unguided 8F multipurpose introducer (Cordis, USA) was then inserted into the nonadherent area, and a 3.5 mm NaviStar

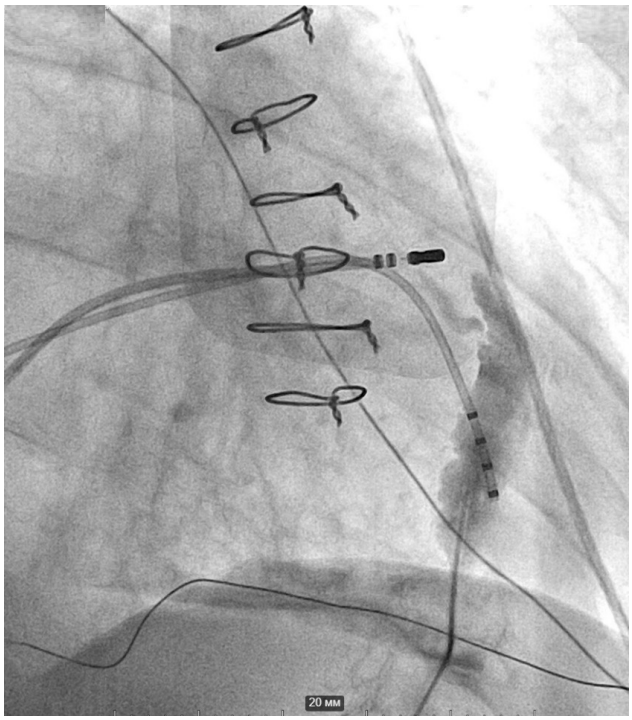

Fig. 2. Subxiphoid approach to the pericardial 'space'. Direct projection, 0°. A 6F intraductal dilator is inserted and a small bolus of contrast through the dilator separates the pericardial leaflets.

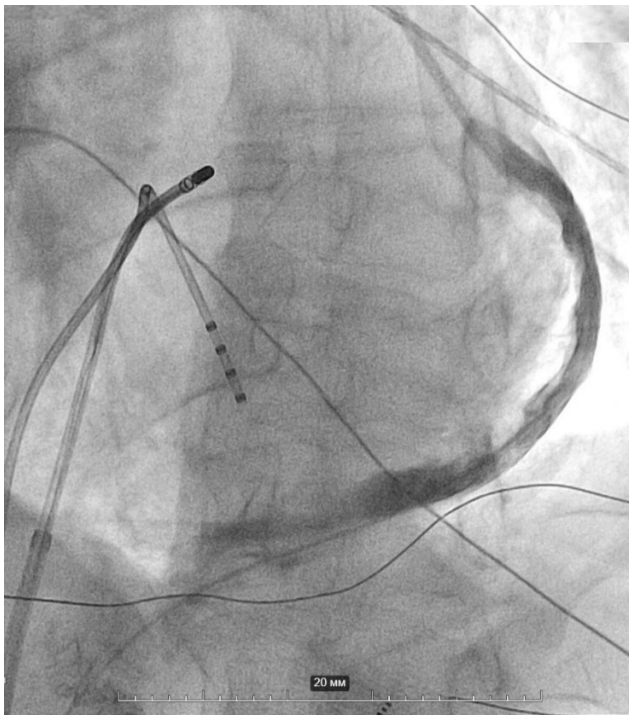

Fig. 3. Contrasting a limited area between the pericardial leaflets after partial separation of the adhesions. Left oblique projection, 30°.

Termocool irrigated tip ablation catheter (Biosense Webster, USA) was inserted through it with the irrigation turned off. The ablation catheter is used in a flexion-extension method and movements in the released space to further separate the pericardial sheets on the lateral wall of the LV, partially on the inferior wall and in the cardiac apex. During separation of the adhesions, limited bleeding into the pericardial cavity (approximately 10 ml) occurred, and spontaneous hemostasis was achieved within 5 minutes.

The epicardial mapping area was limited to the area of adhesion separation and was opposite the area of greatest correspondence between the morphology of the stimulated QRS and the spontaneous clinical VT at the previous procedure.

Endocardial mapping was performed retrograde via transarterial access. Electroanatomic mapping was performed using the CARTO 3 nonfluoroscopic 3D navigation system with the Confidense module (Biosense Webster) with the following settings: LAT Stability - 10 ms, Position Stability - 6 ms, Density - 1 mm, Color Threshold - 10 mm. Endo- and epicardial voltages were mapped with preset limits of 0.5-1.5 mV (bipolar) and 5.0-9.0 mV (unipolar). For endocardial cardiac surface mapping, 1198 and 638 points were recorded for LV and RV, respectively. Epicardial mapping yielded 682 points.

Endocardially, both bipolar and unipolar voltage mapping showed no areas of low amplitude signals (scarring/fibrotic changes) or altered electrical activity (fragmented, late potentials). Epicardially, areas of myocardium with low signal amplitude were identified, located at the cardiac apex (Fig. 4).

Endocardial stimulation and activation mapping identified the most satisfactory criteria for localization of the VT substrate in the apex of the RV in the septal wall (the morphological agreement of the stimulated QRS with the clinical VT when assessed by PASO was 0.978). When stimulation was mapped from LV to this point - PASO correlation was 0.89. Programmed stimulation induced clinical VT with a cycle length of 470 ms mapped in the background of VT - the earliest activation was detected in the apex of the RV endocardially. A series of 40-50 W RF applications (lasting up to 60 seconds at an electrode irrigation rate of 30 ml/min) was applied in this area, resulting in a transient exacerbation of VT. Based on the above, it was decided that RF applications to the epicardial surface of the cardiac apex were necessary.

A series of epicardial RF applications (40-50 W, flush 17 ml/min, ablation time 40-60 seconds) was applied apically at the cardiac apex, at the border between the RV and LV. Subsequent programmed pacing from the RV and LV (up to 5 extrastimuli) and increasing pacing from different parts of the RV and LV did not elicit tachycardia.

Catheher was removed from the femoral vessels, and a drain was left in the pericardial cavity for 6 hours and then removed without sequelae. The patient was discharged on continued antiarrhythmic therapy.

Given the lack of evidence of structural myocardial damage and the absence of a history of circulatory arrest, the decision to implant a cardioverter-defibrillator is made only after an evaluation of the efficacy of the surgical procedure. The patient had no recurrence of VT within one year of epicardial ablation, even after discontinuation of amiodarone.

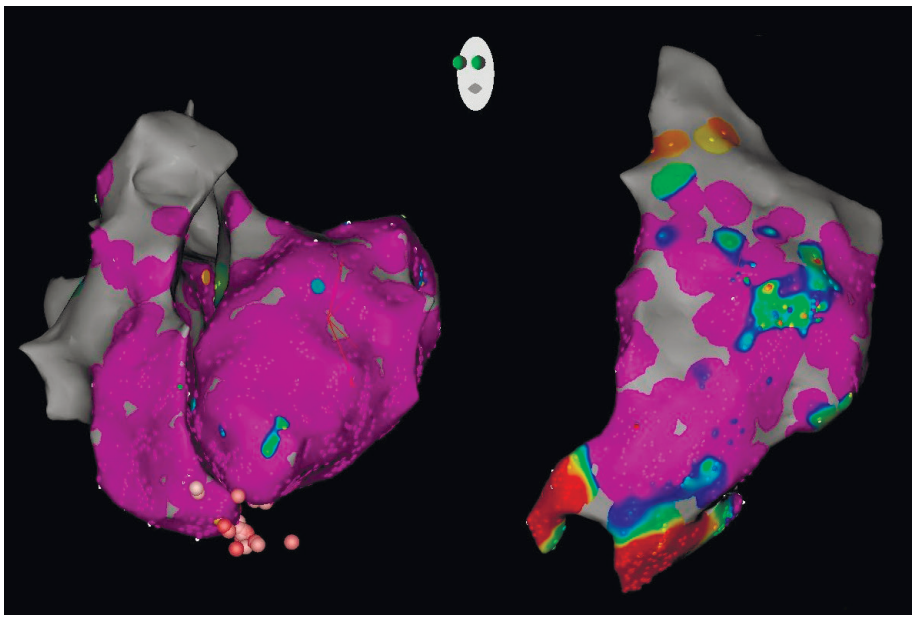

Fig. 4. Electroanatomical mapping, left oblique projection 30º. Left is a bipolar voltage map of the endocardial surface of the left and right ventricles; right is a bipolar voltage map of the epicardial surface of the lateral wall of the left ventricle and the apex of the heart. The detection limit of the scar zones is 0.5-1.5 mV. Purple indicates unchanged myocardium with normal signal amplitude and red indicates myocardium with low signal amplitude. The pink dots are projections of the applied RF applications on the epicardial surface, located on the endocardial map opposite the cardiac apex, where no reduction in signal amplitude or recording of altered potentials was detected.

DISCUSSION

This clinical case demonstrates successful epicardial mapping and ablation of the VT substrate in a patient with postoperative pericardial adhesions.

The issue of safe epicardial access in patients with previous cardiothoracic surgery is very important. Often, patients requiring epicardial mapping/ablation have a history of myopericarditis or cardiac surgery associated with adhesions that complicate access and manipulation between the pericardial leaflets. Dissection of adhesions may be associated with hemorrhage because they may contain new blood vessels or fuse closely with the myocardium or small epicardial vessels. Dissection of adhesions may also be associated with coronary artery damage, as described in the literature [3, 4].

Previously, a history of cardiothoracic surgery was considered an absolute contraindication

for percutaneous epicardial access. However, in 2004, E. Sosa et al. published the first experience with epicardial ablation in patients with a history of cardiothoracic surgery [4].

More recently, in 2013, observations on epicardial ablation in patients with a history of pericarditis and cardiac surgery (without coronary artery bypass grafting) were published: 10 patients were found to have dense adhesions requiring blunt separation [5].

There are now several ways to release pericardial leaflets in patients with adhesions in the pericardial cavity. These include administration of fluids (saline, radiopaque contrast agent), administration of carbon dioxide, and positioning of a balloon catheter in the area of concern may be an additional tool to separate the leaflets [6-8].

In our clinical case, access to the pericardial space was performed after a recent pericardiotomy (2 months later). On the one hand, the short postoperative time may be associated with the active formation of a large number of fresh adhesions; on the other hand, unformed "soft" adhesions allow separation of pericardial prostheses without risk.

Therefore, in patients who have recently undergone cardiothoracic surgery that does not involve coronary artery disease bypass grafting and pericardiotomy, percutaneous epicardial access may be feasible but should be performed in the hospital with the possibility of emergency cardiac surgery after weighing the benefits and risks of potential complications.

REFERENCES

1. Condori Leandro HI, Lebedev DS, Mikhaylov EN. Discrimination of ventricular tachycardia and localization of its exit site using surface electrocardiography. *J Geriatr Cardiol.* 2019;16(4): 362-377. https://doi.org/10.11909/j.issn.1671-5411.2019.04.008.

2. Simonova KA, Lebedev DS, Mikhaylov EN. Epicardial mapping and ablation in management of ventricular tachycardia. *Complex Issues of Cardiovascular Diseases*. 2017;6(4): 138-145. (In Russ.) https://doi.org/y10.17802/2306-1278-2017-6-4-138-145.

3. Killu AM, Asirvatham SJ. Percutaneous pericardial access for electrophysiological studies in patients with prior cardiac surgery: approach and understanding the risks. *Expert Review of Cardiovascular Therapy.* 2018;17(2): 143-150. https://doi.org/:10.1080/14779072.2019.1561276.

4. Yamada T. Transthoracic Epicardial Catheter Ablation. *Circulation Journal.* 2013;77(7): 1672-1680. https://doi.org/:10.1253/circj.cj-13-0510.

5. Sosa E, Scanavacca M, D'Avila A, et al. Nonsurgical transthoracic epicardial approach in patients with ventricular tachycardia and previous cardiac surgery. *Journal of Interventional Cardiac Electrophysiology.* 2004;10(3): 281-288. https://doi.org/:10.1023/B:JICE.0000026925.41543.7c.

6. Tschabrunn CM, Haqqani HM, Cooper JM, et al. Percutaneous epicardial ventricular tachycardia ablation after noncoronary cardiac surgery or pericarditis. *Heart Rhythm.* 2013;10(2): 165-169. https://doi.org/:10.1016/j.hrthm.2012.10.012

7. Verrier RL, Waxman S, Lovett EG, et al. Transatrial Access to the Normal Pericardial Space A Novel Approach for Diagnostic Sampling, Pericardiocentesis, and Therapeutic Interventions. *Circulation*. 1998;98: 2331-2333. https://doi.org/:10.1161/01.cir.98.21.2331.

8. Rogers T, Ratnayaka K, Schenke WH, et al. Intentional Right Atrial Exit for Microcatheter Infusion of Pericardial Carbon Dioxide or Iodinated Contrast to Facilitate Sub-Xiphoid Access. *Catheterization and Cardiovascular Interventions.* 2015;86: E111-E118. https://doi.org/:10.1002/ccd.25698.

9. Leyton-Mange JS, Sze EY, Conley C, et al. Epicardial access facilitated by carbon dioxide insufflation for redo ventricular tachycardia ablation in a patient with arrhythmogenic right ventricular dysplasia and dense adhesions. *Heart Rhythm*. 2021;7(4): 197-202. https://doi.org/:10.1016/j.hrcr.2020.12.008.