The results of the examination and treatment of a patient with frequent ventricular ectopy are presented in the article. During ablation of an ectopic focus in the left coronary sinus of the aorta, as a result of dislocation of the ablation catheter, a spasm of the left coronary artery has been diagnosed and successfully managed.

Keywords: ventricular ectopy; radiofrequency catheter ablation; coronary sinus of aorta; ablation catheter; main left coronary trunk; intracardiac echocardiography

Conflict of Interests: nothing to declare

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Coronary artery (CA) damage during radiofrequency ablation (RFA) for cardiac arrhythmias is a dangerous complication, requiring urgent diagnosis and restoration of coronary blood flow. Nonetheless occurrence of such complication is extremely low. For example, presented data on the results of RFA in 4655 and 3357 patients, demonstrated incidence of this complication 0.09% and 0.029%, respectively [1, 2]. Literature data presented mainly by clinical examples which are difficult to systematize due to their individuality. There are no previous reports of the left CA (LCA) spasm during RFA for ventricular ectopy (VE). In this work, we demonstrate the case of complication and subsequent decisions in patient management.

Patient O., 32 years of age, was admitted to clinic with frequent VE in order to perform RFA, because of the main complaint of palpitations. Anamnesis data: arterial hypertension is not diagnosed, palpitations for two years, according to Holter ECG monitoring frequent single monomorphic VE were recorded in amount of 39 thousand with type of bi-, tri- and quadrigeminy, which served as a reason for planned intervention. The patient had not a significant concomitant pathology and had not received any antiarrhythmic therapy. An objective examination revealed arrhythmic heartbeat due to frequent ectopic beats; there were no abnormal findings in other organs and systems. The patient's ECG is shown in fig. 1. According to echocardiography, the sizes of heart chambers were within normal limits, the first degree of mitral valve prolapse without hemodynamic disturbance, left ventricular false tendon. Laboratory data without pathology.

Radiofrequency ablation procedure
the patient underwent an electrophysiological study (EPS) procedure. As a first stage an electroanatomical mapping (CARTO 3, Biosense Webster, Israel) of right ventricle was performed. Based on local activation time and pace mapping the early zone was defined in the septal region of the right ventricular outflow tract (RVOT), with local electrogram advancing of -28 ms relative to QRS onset. However, pace mapping failed to achieve fully identity of stimulated complexes and an area of 'suboptimal' pacing criteria in RVOT was wide. This area was ablated with the following parameters: temperature -43°C, power 43W in irrigated mode at rate of 17 ml/min. During the RF applications, we repeatedly observed the effect of ectopic focus 'warming up' in the form of ventricular tachycardia with QRS-complexes similar to native VE morphology. We also observed short episodes of eliminated ectopic activity by the end of RF application. The total RFA time was 6 minutes. Due to inefficiency of RFA in RV, we decided to perform left ventricle mapping. Through the right femoral artery approach left ventricle and aortic sinuses were examined using ablation electrode. An earliest activation zone was located in the left coronary sinus, where the local electrogram activation time of -42 ms, and noted positive criteria during pace mapping (Fig. 2).

Further the distal pole of the mapping electrode was advanced at the LCA ostium, which was confirmed by contrast angiogram through an external irrigation circuit. The LCA ostium was marked on the activation map. The measured distance to the ectopic focus amounted to 12 mm, which is considered as acceptable safety [3]. During application ectopic activity disappeared at 6th second, according to temperature rising. RF application was supposed to continue till the target time of 60 seconds. At 40 second of exposure, electrode displaced toward the ostium of LCA, that initially was considered as an allowable change in position of the electrode due to a respiratory excursion, which led to the continuation of the application for 5 seconds more with a further cessation. Thus, the total RF exposure time was 45 seconds. After 10 seconds of RF discontinuation, an episode of ST segment depression in leads II, III and aVF up to 2 mm was observed, which lasted at

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about 30 seconds. This episode was not accompanied by subjective manifestations and hemodynamic disturbances. Due to the presence of transient ischemic changes in the ECG and the proximity of the RFA zone to the ostium of LCA, coronary angiography (CAG) was performed, 60% stenosis of the left main trunk was detected, with negative nitroglycerin test, which was considered as LCA stenosis, but not a spasm. The right CA was without pathology. The LCA was reviewed with intravascular ultrasound (IVUS): the lumen area of LCA trunk was 5.04 mm², i.e. stenosis is interpreted as significant (Fig. 3).

The IVUS results indicated to the presence of edema or persistent spasm. Based on the above data, decision was made in favor to conservative strategy of management. Overseeing in an intensive care unit and double antiplatelet therapy with aspirin and clopidogrel were assigned. The noticeable increase of troponin T up to 1.4 ng/l, creatinkinase and its MB fraction up to 135 and 26 U/l, respectively, was regarded as a manifestation of radiofrequency myocardial damage. Later, laboratory parameters returned to normal within 2 days. The patient’s condition remained stable; there were no ischemic changes in the ECG. On the 5th day after RFA, control coronarography was performed. No data were found for stenosis of the LCA trunk (Fig. 4).

On the 7th day after RFA, with conditions of stable hemodynamics, no complaints and ischemic ECG changes, patient was discharged from the department. The VE were absent for the entire postoperative period.

With further observation, no complaints regard to cardiovascular system were noted. Three months later, bicycle ergometer exercise testing was performed: at a load of 100W, the test was terminated according to the criterion for achieving a submaximal heart rate of 160 beats/min with no ischemic changes. According to the results of Holter ECG monitoring, ventricular ectopic activity was absent. Control coronary angiography was performed 4 months after RFA; no pathology was detected. One year after, the patient’s condition remained stable, which allowed us not to carry out follow-up examinations.

**DISCUSSION**

One of the first description of CA injury during RFA was made by P. Chatelain et al. in 1995 in the case of the left-sided accessory pathway ablation. [4]. Further publications, as mentioned above, indicated an extremely rare frequency of such complications. The damages of CA were represented as an acute occlusion of branches of the right CA and trunk of the LCA, which required immediate stenting of the corresponding arteries, or conservative strategy was adopted due to insignificant stenosis of the narrow ar-

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**Fig. 1.** Patient’s electrocardiogram: sinus rhythm, frequent ventricular extrasystoles.

**Fig. 2.** Results of EPS. On the left panel the local electrogram activation time is -42 ms before the onset of QRS complex; on the right panel the identity of stimulated and spontaneous QRS complexes. I, II, III, aVR, aVL, aVF, V1, V3, V6 - surface ECG leads (leads V2, V4, V5 were not evaluated as less informative); CS 1-2 and CS 3-4 - bipolar electrograms from the coronary sinus; Abl 1-2 and abl Uni - bipolar and monopolar electrograms from the distal pole of ablation electrode.
tery [1, 5]. As well described cases of delayed development of CA stenosis in the ‘normal’ state of the artery immediately after RFA, which required stenting later [6]. With all this, anatomical data demonstrates the proximity of the coronary arteries and, in particular, the LCA trunk to the site of RF-application [3]. According to C. Hasdemir et al., the distance between endocardium of the area of cavotricuspid isthmus and the posterolateral branch of the right CA may be about 5 mm, and the minimum distance between the coronary sinus and circumflex artery - 2 mm, which theoretically should suggest a higher coronary vessels complication rate [7]. Therefore, according to some authors, the low number of such complications due to RFA can be explained by the presence of undiagnosed cases [8]. For the most part, the absence of CA lesions in the ablation zone is associated with the protective effect of intracoronary blood flow on the vessel wall, which provides convective cooling of the area adjacent to the site of RF application [9, 10].

In our description, the LCA trunk damage could occur during RFA in the right ventricular outflow tract or, most likely, during ablation in the left sinus of Valsalva and directly at the ostium of the LCA. The damage of LCA at the RVOT ablation theoretically is possible to suppose, as the last were applied close to location of the ectopic focus, as indicated by the subsequent mapping of the aortic sinuses, the presence of a response of ectopic focus and the temporary elimination of activity. Nevertheless, this assumption is unlikely due to the absence of ischemic changes in the ECG at the time of RF applications, and anatomical data suggest, as a rule, a quite remote relative position of the RVOT and the LCA trunk [3]. It is more likely that the damage occurrence is a result of an inadvertent dislocation of the ablation electrode at the ostium of LCA and the continued application for 5 seconds with direct contact of the tip of electrode with the endothelium of CA. Upon termination of exposure, the electrode was immediately withdrawn to the ascending aorta, therefore, the assumption of its dislocation to the ostium of LCA is based on navigation data. At the same time, the exposure was short, so it did not lead to a rupture of endothelium or a concentric narrowing of the LCA, which was documented during IVUS. The follow-up of the patient was predetermined by the duration of the scar formation after RFA, which is about 8 weeks [11]. At the end of this period, the examination, including coronaryography revealed no abnormalities.

**CONCLUSION**

The main goal that we set when describing this complication is to demonstrate that the stenosis of CA, which developed immediately after radiofrequency damage, does not always require immediate stenting. In some cases, it is only necessary to monitor the patient. The use of IVUS may assist in deciding on the undesirability of early stenting. In deciding on the possibility of discharge the patient from the hospital, stress testing can help. Also, since there are literature cases describing late development of stenosis in the CA despite of ‘normal’ state immediately after the RFA, observation is required for at least three months with repeated coronary angiography in the delayed period. All the above allows one to safely avoid unnecessary stenting of the CA in some cases. In this example, a comparison of clinical data with the results of CA and IVUS made it possible to confine ourselves for conservative treatment of 60% stenosis of the left coronary artery trunk in conditions of targeted dynamic monitoring of the patient.

**REFERENCES**

1. Roberts-Thomson KC, Steven D, Seiler J, et al. Coronary artery injury due to catheter ablation in adults: presentations and outcomes. *Circulation*. 2009;120: 1465-1473.
2. Scheinman MM, Huang S. The 1998 NASPE prospective catheter ablation registry. *Pacing Clin Electrophysiol*. 2000;23: 1020-1028.
3. Katie A. Walsh, MB, MR.Gerard J. Fahy, MD. Anatomy of the left main coronary artery of particular relevance to ablation of left atrial and outflow tract arrhythmias. *Heart Rhythm*. 2014;11: 2231-2238.
4. Chatelain P, Zimmermann M, Weber R, Campanini C, Adamec R. Acute coronary occlusion secondary to radiof-
rency catheter ablation of a left lateral accessory pathway. *Eur Heart J.* 1995;16: 859-861.
5. Piotr Waciński et al. Acute left main coronary artery occlusion following inadvertent delivery of radiofrequency energy during ventricular tachycardia ablation successfully treated by rescue angioplasty with stenting: A two-year follow-up. *Cardiol J.* 2013;20(1): 100-102.
6. Li Yue-Chun, Lin Jia-Feng, Guan Xue-Qiang and Chen Peng. Chronic Left Coronary Artery Stenosis After Radiofrequency Ablation of Idiopathic Premature Ventricular Contraction Originating From Left Coronary Sinus Cusp. *Circ Arrhythm Electrophysiol.* 2016;9: e004353. DOI:10.1161/CIRCEP.116.004353.
7. Hasdemir C, Yavuzgil O, Payzin S, Aydin M, Ulucan C, Kayikcioglu M, Can LH, Turkoglu C, Kultursay H. Angiographic analysis of the anatomic relation of coronary arteries to mitral and tricuspid annulus and implications for radiofrequency ablation. *Am J Cardiol.* 2007;100: 666-671.
8. Schneider HE, Kriebel T, Gravenhorst VD, Paul T. Incidence of coronary artery injury immediately after catheter ablation for supraventricular tachycardias in infants and children. *Heart Rhythm.* 2009;6: 461-467.
9. Castao A, Crawford T, Yamazaki M, Avula UMR, Kalifa J. Coronary arteropathophysiology after radiofrequency catheter ablation: Review and perspectives. *Heart Rhythm.* 2011;8(12): 1975-1980.
10. Haines DE, Watson DD. Tissue heating during radiofrequency catheter ablation: a thermodynamic model and observations in isolated perfused and superfused canine right ventricular free wall. *Pacing Clin Electrophysiol.* 1989;12: 962-976.
11. Huang SK, Bharati S, Lev M, Marcus FI. Electrophysiologic and histologic observations of chronic atrioventricular block induced by closed-chest catheter desiccation with radiofrequency energy. *Pacing Clin Electrophysiol.* 1987;10: 805-816.