Case Report
RVOT ventricular tachycardia ablation in a patient with atrial septal defect: a case report

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Abstract: PVCs originating in the RVOT are the most common form of idiopathic PVCs. However, numerous medical reports have identified RVOT changes using cardiac MRI in patients with idiopathic PVCs. Therefore cardiac MRI is an examination that has begun to be increasingly used for ablation procedures. We present the case of a patient with high number PVCs and VT, originating in a dilated portion of RVOT. A 49-year-old male patient was referred to our cardiology department for high-burden PVC. The ECG showed frequent PVCs with an LBBB morphology and precordial transition in lead V5. Holter ECG showed > 30.000 PVC/24 hours (> 37%) with repeated episodes of non-sustained and sustained VT. Cardiac MRI excluded an ARVC, but identified a type ostium seccundum ASD with mild dilation of the RV and RVOT. Electroanatomical mapping during PVC and VT showed earliest activation at the antero-lateral RVOT region. Catheter ablation was performed using radiofrequency current applied with a 4 mm Navistar Biosense catheter resulting in the complete disappearance of PVCs and VT episodes. At the 30 day follow-up, Holter ECG showed no PVC. At the 12 month follow-up the patient remained asymptomatic, with no PVC at Holter ECG. Our case report demonstrates that RVOT VT can associate with ASD. This association may be accidental, or due to the presence of dilated RVOT.

**Keywords:** RVOT, premature ventricular contractions, ventricular tachycardia, catheter ablation, MRI

Introduction

PVCs are the most common arrhythmias found in clinical practice. Those originating from the RVOT are the most common idiopathic PVCs. Catheter ablation is considered the most suitable approach to treat PVCs located in the RVOT, with a high success rate and a low rate of complications [1].

Atrial septal defect (ASD) is one of the most frequent forms of congenital heart disease. Among different types of ASD, the most frequent is ostium seccundum. When ASD leads to right heart chamber volume overload, the structure and function of RVOT can also be affected [2].

Several studies, using cardiac MRI in patients with idiopathic RVOT-VT have shown structural and functional changes of the RVOT in up to 70% of patients [3]. Markowitz et al. showed that up to 9% of patients with idiopathic VT had RV dilatation [4]. In patients with RV dilation, Corrado et al. found that 15/23 had abnormal voltage maps during electroanatomical mapping, and 8/23 had no abnormality during voltage mapping. They demonstrated that areas of low voltage correlate with abnormal findings such as: myocardial loss and fibrofatty replacement [5].

RVOT-VT may be associated with ASD by chance or due to structural changes of the RVOT. We present the case of a patient with atrial septal
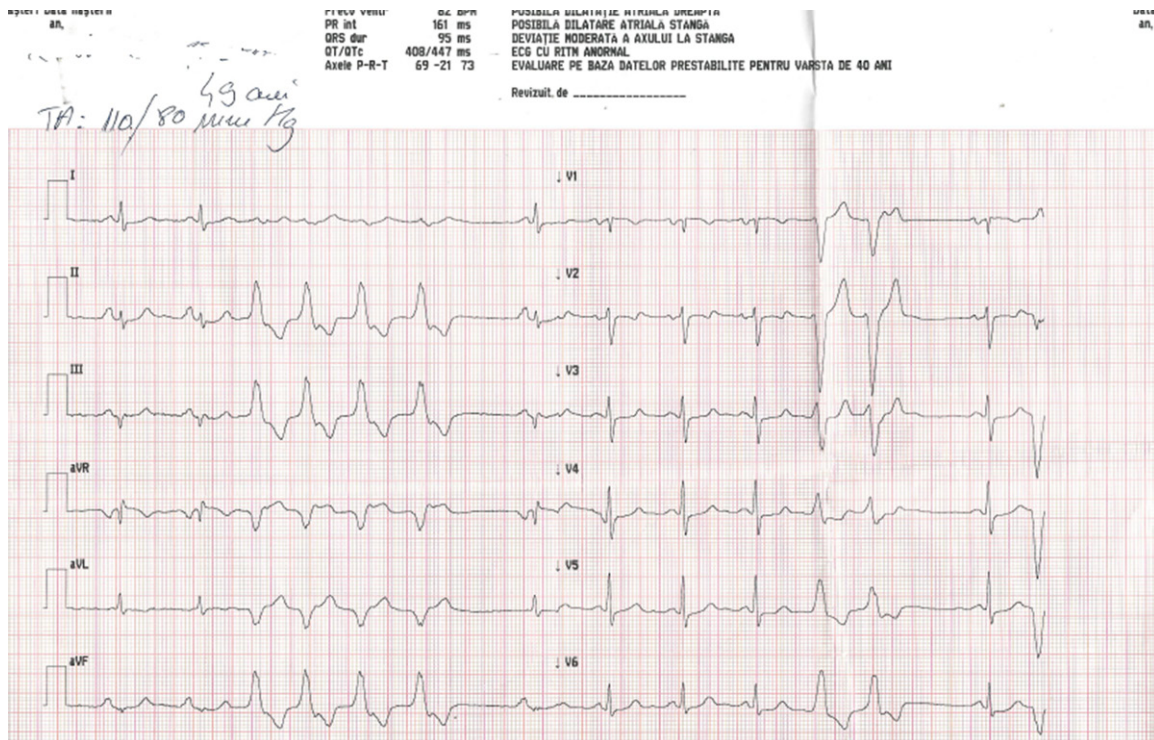
Figure 1. Non-sustained episode of ventricular tachycardia with the same morphology as the PVC: LBBB appearance with inferior axis and precordial transition at V5.

defect and dilated RVOT with high-burden idio-pathic PVCs. After we performed voltage map and demonstrated lack of myocardial scarring, we successfully treated PVCs and VT by catheter ablation.

Case report

A 49-year-old male patient was referred to our cardiology department for high-burden PVC. His only complaint was dyspnea that started 3 years earlier. Physical examination revealed an overweight, hypertensive, and dyslipidemic patient, under treatment with ACEI and statins. He tried several antiarrhythmic drugs, all of which proved to have an insignificant reduction effect on the PVC burden. These drugs included: Metoprolol, Bisoprolol, and Propafenone. The only effective antiarrhythmic drug was Amiodarone which made his symptoms disappear; however, he interrupted the treatment after 3 days after reading the possible side-effects of the drug. Upon his arrival in our cardiology department the ECG showed frequent PVCs with a LBBB morphology and precordial transition in lead V5. He also presented with repeated episodes of non-sustained ventricu-lar tachycardia with the same morphology as the PVCs. The Holter ECG showed > 30.000 PVC/24 hours (> 37%) with repeated episodes of NSVT or sustained VT (**Figures 1-3**). The echocardiogram showed a mild increase in RA and RV diameters, with grade 2 tricuspid re-gurgitation and ostium secundum ASD. The ASD size was 12 mm determined from the api-cal 4 chamber view and the subcostal view at end-systole. The tricuspid regurgitation flow was measured in order to estimate the systolic pressure in the pulmonary artery. The estima-ted PASP (pulmonary artery systolic pressure) was 43 mmHg. RVOT proximal diameter was increased to 41 mm from the parasternal sh-ort-axis view during end-diastole (**Figure 4**).

A cardiac MRI was performed in order to evalu-ate the RV and RVOT function, as well as tissue changes (**Figure 5**).

The cardiac MRI revealed an overall expansion of the right ventricle and RVOT, without signs of aneurysm. The T1-weighted images and the delayed myocardial enhancment MRI, excluded the presence of myocardial fat, and scarred areas.
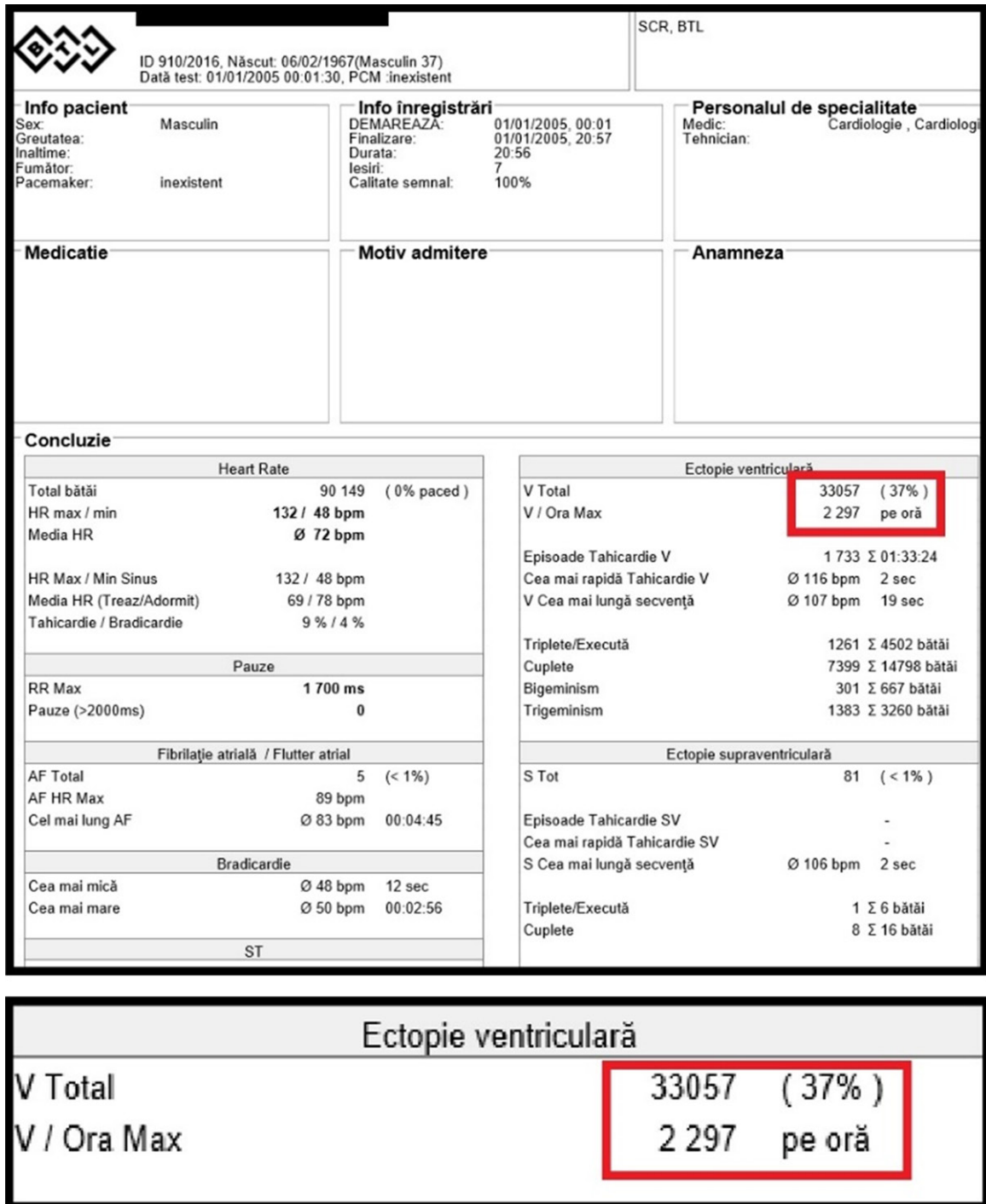
Figure 2. Holter ECG showed more than 33.000 PVCs/24 hours with a high-burden of > 37%/day.

Overall cardiac MRI excluded an ARVC, but identified a type ostium secundum ASD, with mild dilation of the right cavities. In coronography, the presence of coronary stenosis was excluded. Due to the high-burden PVCs, catheter ablation was proposed to the patient.

The patient underwent an electrophysiological study using 3 catheters: a bipolar lead introduced at the level of the RV apex, a quadripolar diagnostic catheter introduced inside the coronary sinus and a 4 mm Bionsense Webster Navistar catheter introduced at the level of
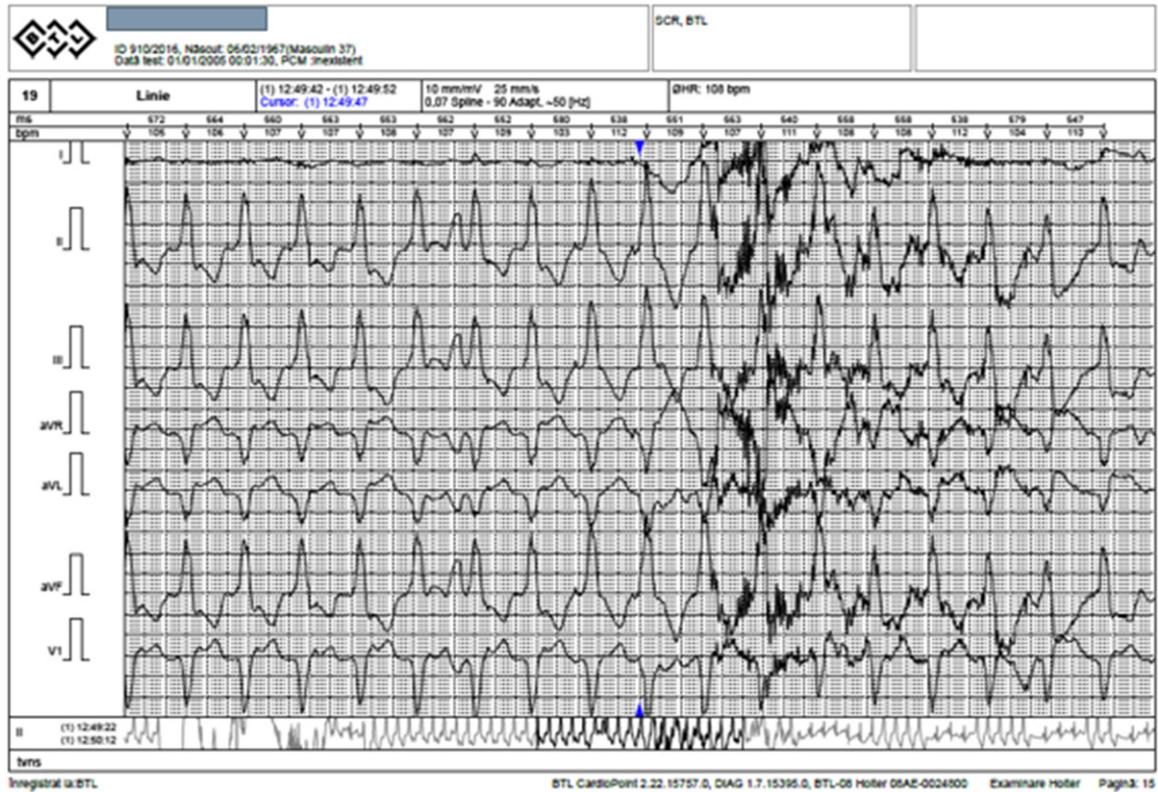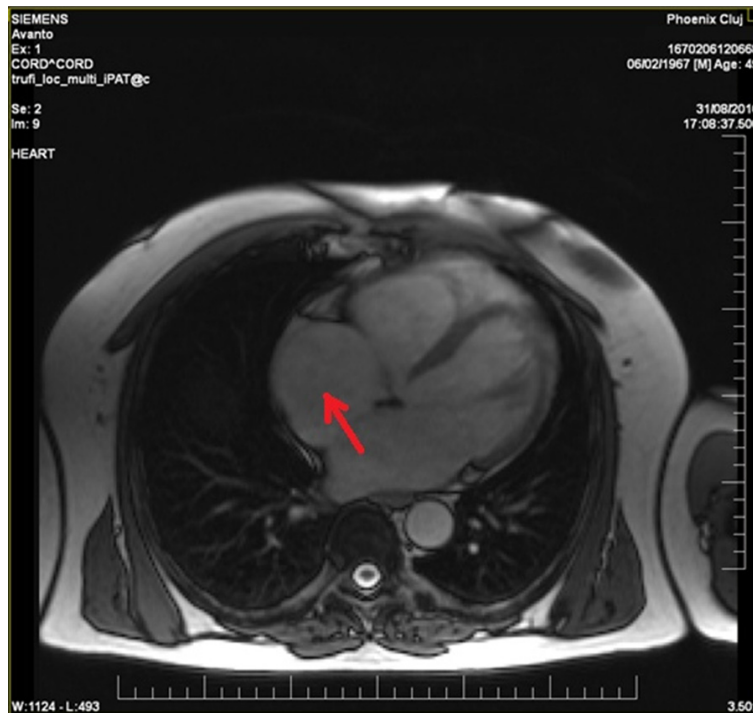
Figure 3. Holter ECG: Episode of sustained ventricular tachycardia with the same morphology as the PVC. The duration of the episode was longer than 30 seconds and the patient felt dizziness.
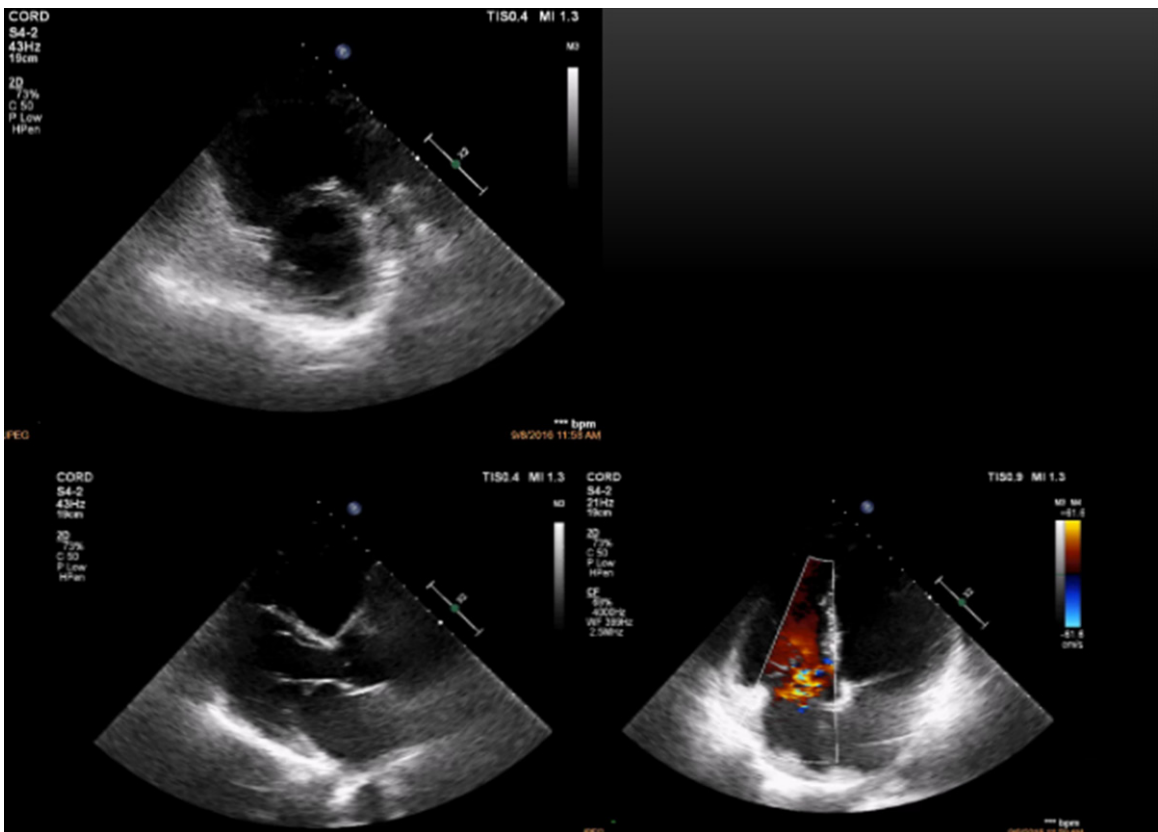
Figure 4. Transthoracic echocardiography: the left cavities are normal, non-dilated, but the right cavities are dilated with moderate tricuspid regurgitation.

Figure 5. Cardiac MRI shows an ostium seccundum atrial septal defect with mild dilation of the right atrium and right ventricle.

RVOT. The EP study was carried out using the Carto 3 electroanatomical three-dimensional mapping system (Biosense Webster). Voltage map detected healthy RV and RVOT without areas of scarring. Subsequently, the activation map "during PVCs" revealed the most early "zone" at the antero-lateral RVOT level, with the local potential preceding the onset of QRS by 30 ms (**Figures 6**, **7**). The pacemap in the area of the earliest signal showed a 97% concordance with the patient's clinical PVC. The ablation was performed using radiofrequency current, applied with the 4 mm Navistar Biosense catheter, resulting in the complete disappearance of the extrasystoles and VT episodes. At the end of EP study the patient had no PVCs. During 24 hours of monitoring in the ICU, the patient did not have PVCs. At the thirty days follow-up, the patient was monitored for 24 hours, using a Holter ECG, which showed no PVC (**Figure 8**). At the 12 month follow-up, the patient remained asymptomatic, with no PVC at Holter ECG. Repeated ECG and Stress Tests found no PVC during rest or during effort.

Discussion

The association between RVOT-VT and ASD can be accidental or it may be a causal relationship; namely the dilation of the right ventricle and RVOT induces RVOT-VT. At the level of RVOT, there are circumferential muscle fibers that produce a radial contraction during ventricular systole. RVOT is less susceptible to regional abnormalities than other RV regions; such as the apex, inflow tract, or lateral wall [6]. However, right heart volume overload in patients with ostium secundum ASD is associated with structural and functional changes of the RVOT. Koestenberger, et al. [7] demonstrated an increase in RVOT diameter in children with ostium secundum ASD, compared to healthy children. RVOT regions affected by fibrosis can be identified either by cardiac MRI or by measuring the local voltage during electroanatomical mapping. In the series of Carlson et al. [8] cardiac MRI revealed structural and functional changes of the right ventricle in 22 of 24 patients with RVOT-VT (95%); especially focal areas of decreased systolic thickening of the RVOT walls. Our patient had RVOT dialation, but did not have areas of fibrosis neither on MRI nor during electroanatomical mapping.

It is postulated that idiopathic arrhythmias from RVOT [9], occur in patients without structural cardiac involvement. Among these arrhythmias are: RVOT-PVCs, monomorphic VT, and repetitive monomorphic tachycardia. These all have similar ECG features that suggest the origin under the pulmonic valve. The mechanism of RVOT-VT is catecholamine-mediated delayed after-depolarizations [10]. This triggered activity depends on the stimulation of cAMP, which leads to increased intracellular calcium. The triggered activity mechanism is supported by the difficult induction of RVOT-VT with programmed ventricular stimulation, by the facili-
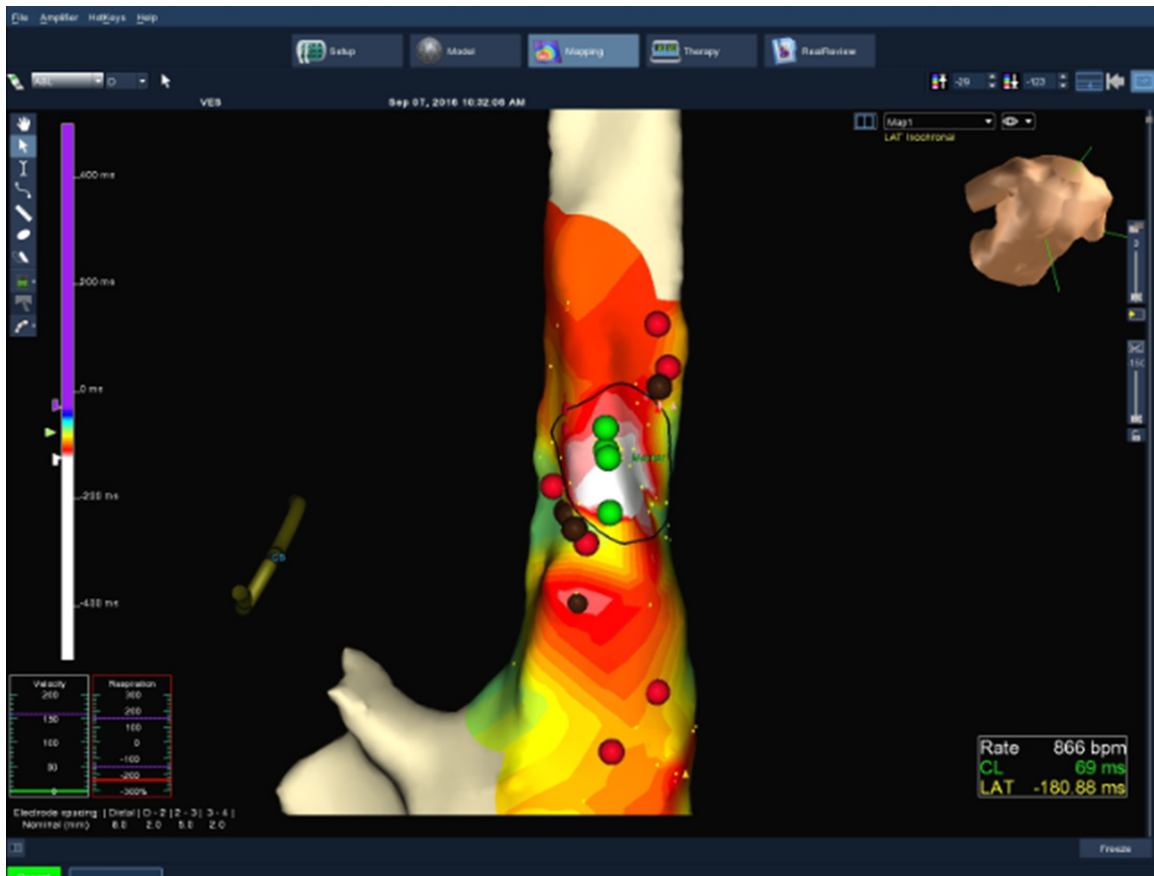
Figure 6. Electronatomical mapping during RVOT PVC shows earliest activation at the antero-lateral region of the RVOT. The earliest region is white, ant the latest is green. Green dots were spots where RF application was effective in stopping ventricular tachycardia and PVCs.

Figure 7. Local electrogram at the site of earliest activation. Abl d = bipolar signal of the distal bipole of the ablation catheter, Abl uni-unipolar signal of the ablation catheter. Local electrical activity is 30 ms earlier than QR onset and the unipolar signal shows QS pattern.
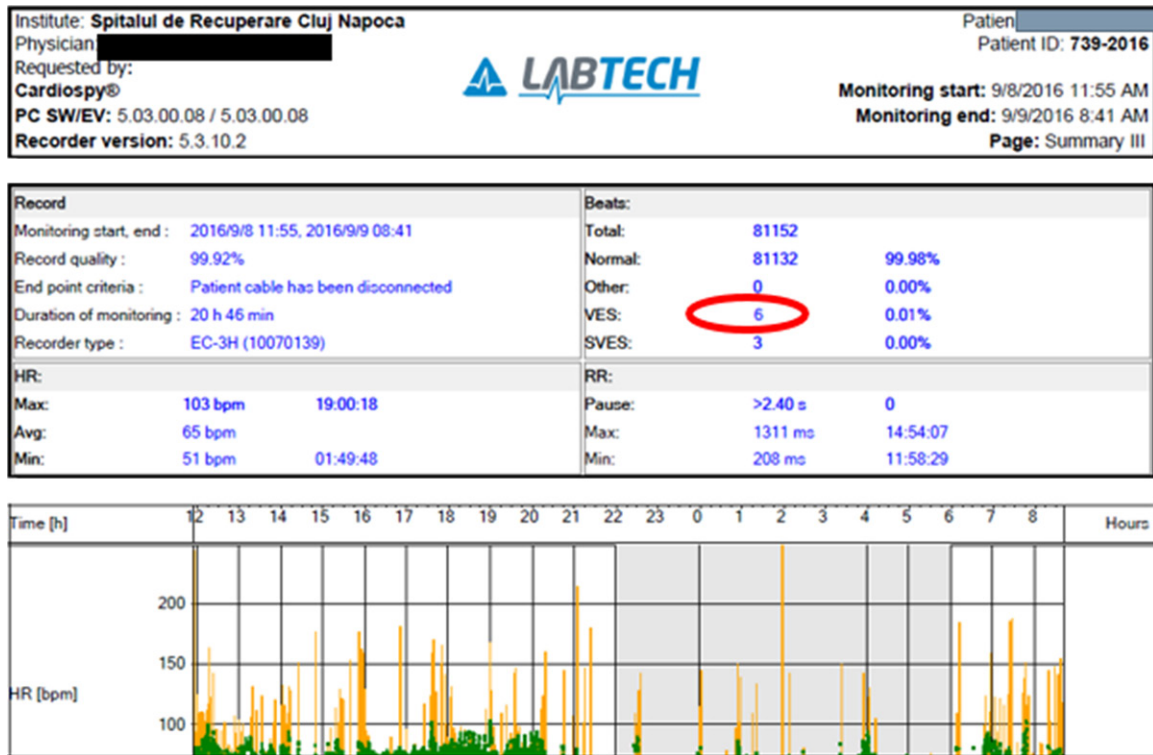
Figure 8. Holter ECG before the discharge of the patient shows 6 PVCs, none of which present the same morphology as the clinical PVC which was ablated.

tating effect of isoproterenol on VT induction, and by the cycle-length dependence of VT [11]. In contrast with other types of ventricular tachycardia, RVOT-VT is not treated by ICD implantation, but with drugs, or by catheter ablation- in the case that the drugs prove inefficent. These arrhythmias have focal origin and can be ablated by focal applications [12-14].

Over the past 20 years, numerous medical reports have identified RVOT changes using cardiac MRI in patients with idiopathic TV. Cardiac MRI is an examination that has begun to be increasingly used for ablation procedures, not only for complex, but also for the simplest ones [15, 16]. It is used in patients with RVOT-PVC to differentiate patients with a "normal heart" from patients with structural impairment of the RV and RVOT, such as: arrhythmogenic RV cardiomyopathy, sarcoidosis, or other disorders of the RV structure and function [17]. Cardiac MRI with gadolinium injection can pro-

vide a global picture of RVOT anatomical and functional changes; such as RVOT dilation, fatty infiltration, subtle changes in parietal kinetics, or limited scar.

ASD is the most common congenital heart disease with a left-to-right shunt. The clinical symptoms and the age at which these symptoms occur are highly variable. The most common symptoms are dyspnea on exertion and fatigue. There are three major types of ASD: ostium primum, ostium secundum, and type sinus venosus. The most common type is ASD secundum, which is located in the middle of the atrial septum at the level of fossa ovalis [18]. Cardiac MRI helps to define the cardiac anatomy, to calculate the ventricular diameter and volumes, and to estimate the shunt volume. If this ratio exceeds 1.5:1, surgical or interventional treatment of ASD is indicated. The most common arrhythmia found in patients with type ostium seccundum ASD is macroreentry in the right

atrium. For non-operated ASD, the most frequent reentrant arrhythmia remains the cavotricuspid isthmus dependent atrial flutter [19, 20]. A study of 218 patients with ASD showed that the most common arrhythmias found in this category of patients are: atrial flutter, atrial fibrillation, or combination of the two [21]. However, other arrhythmias such as PVCs may be present accidentally or due to structural changes of RV or RVOT.

In conclusion, our report demonstrates that RVOT VT can associate with ASD. This association may be due to the presence of dilated RVOT, without myocardial scarring in voltage mapping.

Disclosure of conflict of interest

None.

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