

Case Report

Unusual Site of Origin of a Non-Automatic Focal Right Ventricular Tachycardia

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Focal right ventricular tachycardia is relatively uncommon. It usually arises from specific anatomic locations. A 59-year-old woman with a structurally normal heart and an automatic cardioverter-defibrillator implanted beforehand presented with drug-resistant incessant ventricular tachycardia for which 1786 anti-tachycardia pacing therapies and 119 shocks had been delivered. Electroanatomical mapping showed focal tachycardia originating from the acute margin of the right ventricle. Irrigated catheter ablation was performed successfully.

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Focal ventricular tachycardia (VT) is an uncommon arrhythmia. The underlying mechanism is triggered activity, micro-re-entry or abnormal automaticity.^{1,2} In patients with structurally normal hearts it usually originates from specific anatomical locations, such as ventricular outflow tracts, atrioventricular annuli, aortic/pulmonic cusps, or papillary muscles. Other locations of the arrhythmia focus seem to be exceedingly rare.^{3,4}

Case presentation

A 59-year-old female patient was admitted for ablation of recurrent drug-refractory, sustained monomorphic, left bundle branch block, left superior axis VT (Figure 1). The first time she had experienced the above-described syncopal tachycardia was 6 years earlier, when it degenerated into ventricular fibrillation terminated by external defibrillation. Reversible arrhythmia causes were excluded. Her family history was negative for sudden cardiac death. The 12-lead electrocardiogram (ECG), signal-averaged ECG, echocardiography, and coronary angiography were normal. During an electrophysiological study VT could

not be induced. Accessory pathways, dual atrioventricular nodal physiology, and atrial tachycardias were excluded. She was discharged on amiodarone as an implantable cardioverter-defibrillator (ICD) was an unavailable option at that time.

Five years later the patient was switched over to propafenone and metoprolol because of amiodarone-induced hyperthyroidism, after which she had recurrences of non-syncopal VT. A repeat electrophysiological study after interruption of antiarrhythmic medication was again negative. She was given sotalol, which she stopped of her own will. She soon had VT recurrences, for which a dual chamber ICD was implanted, and she was put back on propafenone and metoprolol. Fifteen months later the patient started to feel multiple ICD shocks. She was found to be in incessant VT, refractory to lidocaine, verapamil and magnesium sulphate, recurring shortly after successful anti-tachycardia pacing (ATP) or ICD shock. Device interrogation showed that it had detected 735 episodes of VT and 1 of ventricular fibrillation, and had delivered 1786 ATPs and 119 shocks. Thyroid stimulating hormone level, 12-lead ECG, echocardiogra-

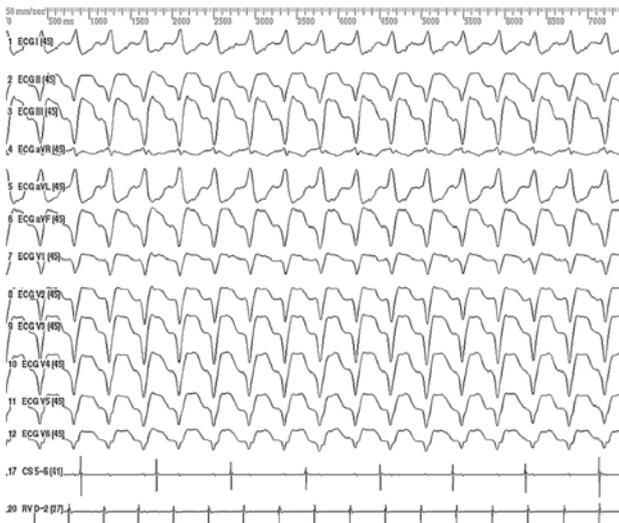


Figure 1. Twelve-lead ECG of the clinical ventricular tachycardia. Intracardiac electrograms from the coronary sinus (CS 5-6) and the right ventricle (RV D-2) are shown at the bottom. Ventricular-atrial dissociation is clearly visible.

phy, cardiac 64-slice multi-detector computed tomography (Figure 2A), and coronary angiography were normal. Right ventricular (RV) angiography showed a trabeculated septal apical portion (Figure 2B), with an otherwise normal RV (Figure 2C).

The procedure was performed using the EnSite NavX electroanatomic mapping system (St. Jude Medical, Inc., St. Paul, MN, USA). RV activation and voltage maps were created during ongoing VT. Ventricular premature beats induced by catheter movements terminated VT several times. Fortunately, this time it was possible to induce it with ventricular burst

pacing. The voltage map did not show low voltage areas. A centrifugal activation pattern was clearly described on the activation map (Figure 3A). The area of earliest activation was located at the acute margin of the RV free wall, where the potentials were of normal amplitude and not fractionated. A good match between VT and paced QRS complexes was found during pacing in this area (Figure 3B). Radiofrequency irrigated catheter ablation was done. During the eighth RF application an audible pop phenomenon occurred, the patient reported moderately severe chest pain radiating to the right shoulder, and a small pericardial effusion was diagnosed by echocardiography. Thereafter, the VT could not be re-induced with burst and programmed ventricular pacing, even during catecholamine infusion. The pericardial effusion resolved within 3 days and the patient was discharged on metoprolol. Fifteen months later she was free of arrhythmia.

Discussion

Idiopathic focal RV tachycardia, although uncommon, is quite well described.^{1,2,5} It shows a characteristic anatomical predilection, with RV foci typically located in the outflow tract or around the tricuspid annulus. There are only scanty data concerning idiopathic focal RV tachycardia arising outside these sites.^{3,4}

We report here on a patient with focal VT originating at the acute RV margin in the absence of detectable structural heart disease. Satish et al³ described an origin in the sub-tricuspid septum, although this seemed to be the tricuspid annular position as far as

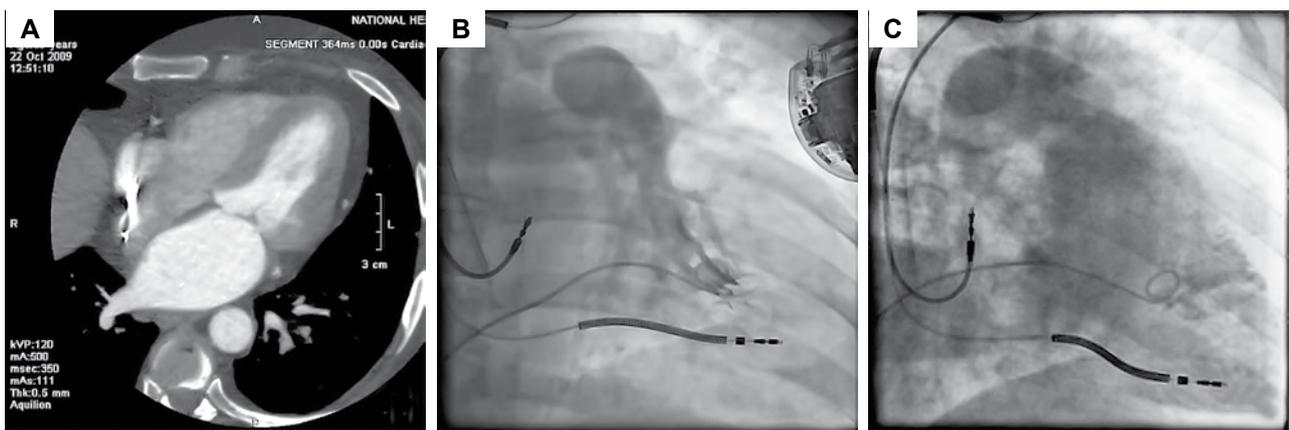


Figure 2. A. Contrast-enhanced multi-detector computed tomographic angiography of the left atrium and left ventricle – four-chamber view of the heart. B. Right ventricular angiography (systole) in anteroposterior projection showing heavy trabeculation anteroapically and anteroseptally, but no akinetic or dyskinetic areas. C. Right ventricular angiography (diastole) in 45° right anterior oblique projection – trabeculation is still visible.

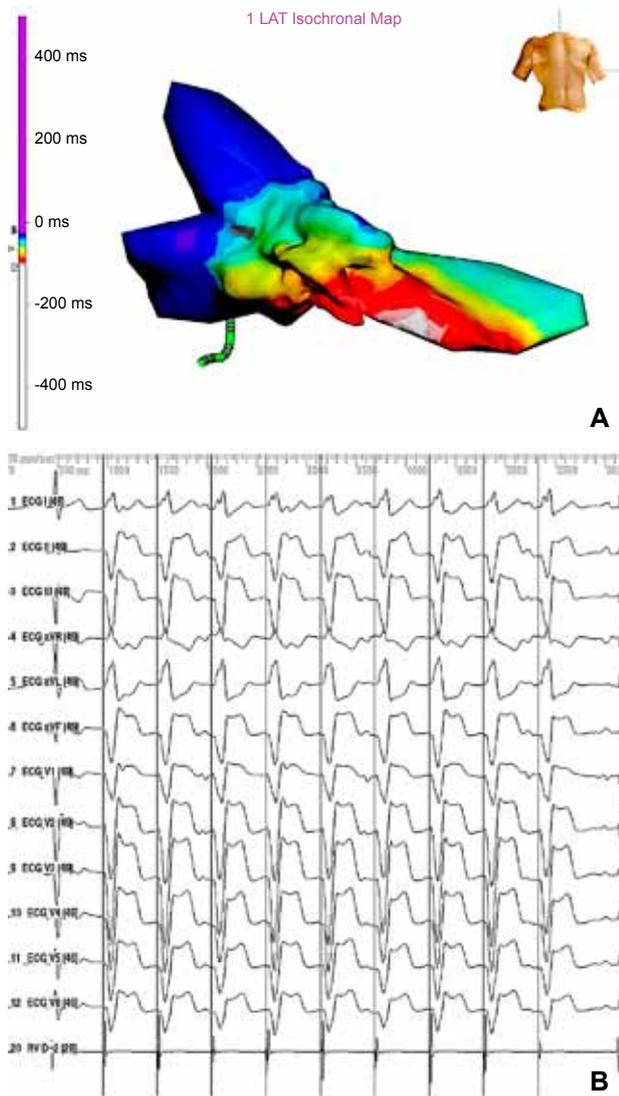


Figure 3. A. Local activation map of the ventricular tachycardia. B. Twelve-lead ECG during pacing at the origin of the tachycardia.

one can judge from the fluoroscopic image and the intracardiac electrogram. Navarrete described a true apical exit site.⁴

The ECG morphology of the VT in this patient differed significantly from the common ECG patterns of idiopathic VTs.⁶ It led us to expect an exit site in or near the RV apex. A possible explanation for the discrepancy could be the lack of precision of the 12-lead ECG or a clockwise rotation of the heart in the horizontal plane. Interestingly, in our case the QRS in aVR was positive/negative, while in the paper by Navarrete⁴ it was negative, which is not expected when the excitation front has an apical origin.

The mechanism of the described VT is non-automatic. Induction by stimulation and termination by ATP, ICD shocks, and mechanically induced ventricular premature beats reliably excludes automaticity.^{1,7} The focal pattern of activation is compatible with micro-re-entry or epicardial or mid-myocardial macro-re-entry with an endocardial exit site.^{1,5,6,8} Macro-re-entry is mostly a theoretical possibility in this case, because this patient had a structurally normal heart and single VT morphology. Besides, ablation at the exit site can hardly be expected to render a macro-reentrant tachycardia uninducible. The response to adenosine in relation to triggered activity could not be tested because the drug was unavailable. The induction by pacing during only one of three electrophysiological studies is in favour of a triggered mechanism.

Although it was not possible to detect any significant structural abnormalities, we cannot completely exclude subclinical structural heart disease. However, this is unlikely, as thorough echocardiographic and angiographic studies done 6 years apart were virtually identical. Besides, the voltage map showed normal myocardial voltage in the RV.

In conclusion, idiopathic focal RV tachycardia may present an unusual site of origin. It may, nevertheless, be successfully ablated, although the creation of a transmural lesion might be necessary.

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