Case Report

Atrioventricular Re-Entrant and Ventricular Tachycardias of Left Origin: A Rare Combination

Polychronis Dilaveris, Charalambos Vassilopoulos, Panagiotis Ioannidis, Konstantinos Gatzoulis, Christodoulos Stefanadis

First Department of Cardiology, University of Athens Medical School, Athens, Greece

Key words: Atrioventricular reentrant tachycardia, concealed pathway, ventricular tachycardia. The simultaneous occurrence of atrial and ventricular tachycardias, or junctional and ventricular tachycardias, has been reported previously. We report on a patient presenting with syncope and demonstrating, simultaneously, atrioventricular re-entrant tachycardia and ventricular tachycardia of left origin. A left lateral concealed pathway was responsible for the former arrhythmia, while the latter arrhythmia was located in the anterolateral wall of the left ventricle. This is a rare type of double tachycardia and stresses the need for a thorough investigation of patients who have concealed accessory pathways and a previous history of atrioventricular re-entrant tachycardia, when they present with syncope.

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Address:
Polychronis Dilaveris

22 Miltiadou St. 155 61 Holargos Athens, Greece e-mail: hrodil@yahoo.com

he simultaneous occurrence of atrial and ventricular tachycardias, or junctional and ventricular tachycardias, has been reported previously. 1,2 Exercise- or catecholamine-induced double tachycardia,^{3,4} as well as the coexistence of atrioventricular (AV) re-entrant tachycardia and ventricular tachycardia originating in the right ventricular (RV) outflow tract,⁵ have also been described. However, reports of the coexistence of AV re-entrant tachycardia and ventricular tachycardia of left origin are rather sparse in the literature.⁶ We describe a patient with a left lateral concealed pathway demonstrating both AV reentrant tachycardia and ventricular tachycardia of left origin.

Case presentation

A 65-year-old man with a history of a left lateral concealed pathway and sporadic episodes of AV re-entrant tachycardia was referred to our hospital because of syncope. He had experienced palpitations for several years, which in most cases were spontaneously terminated. This was his first synco-

pal episode. The 12-lead electrocardiogram (ECG) showed no obvious pre-excitation (Figure 1), while the signal-averaged ECG was marginally abnormal (filtered QRS duration=114 ms; root-mean-square voltage in terminal 40 ms= $21 \mu V$; duration of highfrequency-low-amplitude signals $<40 \,\mu\text{V}=$ 43 ms). Informed consent was obtained and the electrophysiology study was performed in the post-absorptive, non-sedated state. Quadripolar catheters were inserted via the femoral vein using the Seldinger technique and advanced to the high right atrium, RV apex, and His bundle position. A 16-polar deflectable-tip catheter was positioned in the coronary sinus (CS) via the left subclavian vein. With incremental right atrial pacing and extrastimulus testing, orthodromic AV re-entrant tachycardia was induced with a stable cycle length of 329 ms (Figure 2). The ventriculoatrial (VA) conduction time was 178 ms and the earliest atrial activation was recorded on the distal CS electrogram. With ventricular extrastimulus testing, retrograde atrial activation was earliest on the distal CS electrogram. Radiofrequency ablation was performed us-

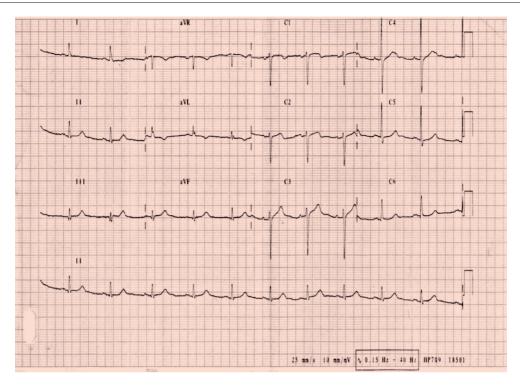


Figure 1. Surface 12-lead electrocardiogram of the patient during sinus rhythm.

ing a deflectable 4 mm tip ablation catheter (Cordis/Webster, Boston Scientific International, Boston MA, USA), which was inserted via the femoral vein and introduced through an open *foramen ovale* to the left

atrium.^{7,8} Detailed endocardial mapping along the mitral valve was performed during orthodromic AV reentrant tachycardia. The earliest VA conduction time was determined in the left lateral site of the mitral

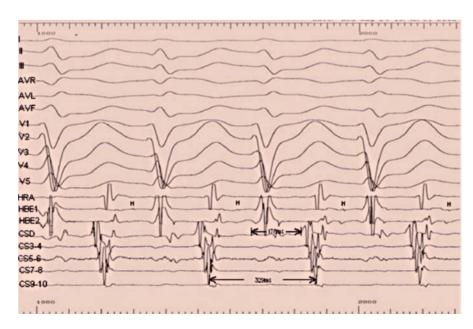


Figure 2. Inducible orthodromic atrioventricular re-entrant tachycardia with a stable cycle length of 329 ms. The ventriculoatrial conduction time is 178 ms and the earliest atrial activation is recorded on the distal coronary sinus (CS) electrogram. Recording speed 100 mm/s.

valve, and the accessory pathway was successfully ablated at this site by applying radiofrequency current (35 W) for 1 minute. Thus, AV re-entrant tachycardia was terminated and was not inducible by isoprenaline or programmed stimulation.

During the endocardial mapping along the mitral valve, under AV re-entrant tachycardia and isoprenaline infusion (0.33 µg/min), sustained monomorphic ventricular tachycardia with a right bundle branch block (RBBB) morphology, a right QRS axis and a cycle length of 240 ms was twice provoked and terminated by overdrive pacing (Figure 3). The patient underwent a complete workup, which included transthoracic and transoesophageal echocardiography, left and right heart catheterisation, and cardiac magnetic resonance imaging that revealed only the presence of a small atrial septal defect. Although there was no evidence that the sustained ventricular tachycardia induced was clinically relevant and it could have been provoked by isoprenaline infusion, radiofrequency ablation of this arrhythmia was decided upon, with the assistance of an electroanatomical mapping system (CARTO XP EP Navigation System, Biosence Webster Inc., a Johnson & Johnson company, Diamond Bar CA, USA). Ablation was performed by introducing the deflectable 4 mm tip D-

type ablation catheter Navistar (Johnson & Johnson company, Diamond Bar CA, USA) using the retrograde transaortic approach across the aortic valve into the left ventricle. Left ventricular endocardial mapping was performed during sinus rhythm, and revealed the presence of abnormally low potentials in the anterolateral wall of the left ventricle. Activation mapping and pace mapping criteria were used to localise the potential ablation sites in the left ventricular anterolateral wall. Radiofrequency energy was delivered in sites where pace maps showed a QRS complex identical to that of ventricular tachycardia in all 12 ECG leads and a Purkinje potential that was recorded before ventricular activation during sinus rhythm. Radiofrequency ablation induced a slow ventricular rhythm identical to that of ventricular tachycardia, which was terminated. Although the target tachycardia was non-inducible thereafter, two other forms of ventricular tachycardia with a different axis from the first were induced by programmed stimulation and before the re-institution of isoprenaline infusion. Both were successfully mapped along the left lateral wall (Figure 4) and successfully ablated. Thereafter, ventricular extrastimulus testing did not induce sustained ventricular tachycardia.

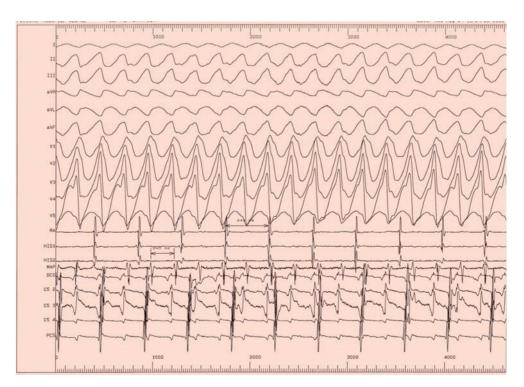


Figure 3. Inducible sustained monomorphic ventricular tachycardia with right bundle branch block morphology, a right QRS axis and a cycle length of 240 ms. Recording speed 50 mm/s.

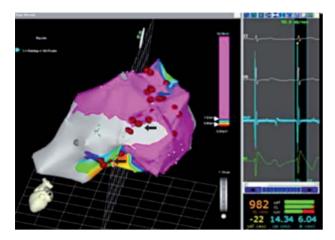


Figure 4. Electroanatomical mapping in our patient in a modified left lateral view, indicating the ablation sites (brown dots). The tip of the ablation catheter is indicated by the grey dot. The blue arrow indicates the site where radiofrequency ablation was delivered to eliminate the first form of ventricular tachycardia. The black arrows indicate the sites where radiofrequency ablation was delivered to eliminate the two other forms of ventricular tachycardia.

Discussion

Here we describe a case of simultaneous occurrence of AV re-entrant tachycardia and ventricular tachycardia of left origin. A left lateral concealed pathway was responsible for the former arrhythmia. This pathway was mapped and successfully ablated in the left lateral site of the mitral valve. The latter arrhythmia was located in the anterolateral wall of the left ventricle, was probably caused by re-entry and showed multiple "exit" sites with multiple QRS morphologies. The presence of re-entry as the potential substrate of this left-sided ventricular tachycardia is substantiated by the initiation and termination characteristics of the arrhythmia and the presence of a marginally abnormal signal-averaged ECG. Although no organic heart disease was detected in this patient by our detailed non-invasive and invasive workup, we did not perform endocardial biopsy to rule out the possible presence of diffused or patchy endocardial fibrosis. Commonly, idiopathic left ventricular tachycardia arises from the septal wall and has RBBB morphology with left axis deviation. 10,11 However, in our patient the left-sided ventricular tachycardia was located in the anterolateral wall and had RBBB morphology with right axis deviation. The presence of a re-entrant circuit with multiple "exit" sites in the left lateral wall could explain the appearance of multiple (three) forms of ventricular tachycardia that were induced by programmed stimulation and, subsequently, the need for the application of radiofrequency ablation at the respective sites of the left lateral wall. Although isoprenaline infusion might reasonably be expected to have contributed to the provocation of the first form of ventricular tachycardia, it cannot explain the induction of the two other forms of ventricular tachycardia after the successful elimination of the first form of the arrhythmia. Moreover, we cannot exclude the possibility that the lesion caused by the radiofrequency ablation of the AV re-entrant tachycardia could have contributed to the appearance of ventricular tachycardias.

The simultaneous occurrence of atrial and ventricular tachycardias, or junctional and ventricular tachycardias, has been reported previously. 1,2 This type of tachycardia has been described in digitalis intoxication, generally in patients with left ventricular dysfunction.^{1,2} Exercise- or catecholamine-induced atrial and ventricular tachycardias, classically defined as double tachycardia, have also been reported.^{3,4} In addition, the coexistence of AV re-entrant tachycardia and ventricular tachycardia originating in the RV outflow tract has been demonstrated.⁵ The coexistence of AV re-entrant tachycardia and ventricular tachycardia of left origin has been only sparsely reported previously. Our case presents this rare type of double tachycardia and stresses the need for a thorough investigation of patients with concealed accessory pathways and a previous history of AV re-entrant tachycardia, when they present with syncope.

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