Ventricular Tachycardia Originating From the Valsalva Sinus of Left Coronary Cusp

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Summary

A 21-year-old man presented with frequent episodes of palpitations. A 12-lead ECG revealed an incessant ventricular tachycardia (VT) originating from the outflow tract at a rate of 144 bpm. During electrophysiologic study, clinical sustained VT was repeatedly inducible with programmed ventricular stimulation. An S wave in lead I, a precordial R wave transition in lead V1 and the absence of S wave in leads V5 or V6 conducted the origin of VT as left ventricular outflow and supravalvular region. Pace- and activation-mapping and diastolic activity directed the ablation catheter to the Valsalva of the left coronary sinus. With the guidance of coronary angiography, a single radiofrequency application terminated the VT. No early or late complications or recurrence of VT was observed during follow-up. ***(Türk Kardiyol Dern Arş 2004; 32: 393-396)***

Key words: *Idiopathic ventricular tachycardia, left ventricular outflow tract, radiofrequency ablation*

Özet

Sol Koroner Küspis Valsalvasından Kaynaklanan Ventriküler Taşikardi

Yirmibir yaşında erkek hasta sık çarpıntı atakları ile müracaat etti. Alınan 12 derivasyonlu EKG'si çıkış yolundan kaynağını alan hızı dakikada 144 olan ventriküler taşikardi gösteriyordu. Elektrofizyolojik çalışmada, programlı ventriküler uyarı ile uzamış ventriküler taşikardi indüklendi. DI'de S, V1'de R dalgası bulunması ve V5-6'da S dalgası olmaması taşikardinin sol ventrikül çıkış yolunda ve supravalvüler yerleşimli olduğunu gösteriyordu. Pace ve aktivasyon haritalaması ile diyastolik aktivasyon ablasyon kateterini sol koroner sinüs Valsalva bölgesine yönlendirdi. Koroner anjiyogram kılavuzluğunda verilen tek bir radyofrekans uygulaması taşikardiyi sonlandırdı. Erken ve geç komplikasyon olmayan olguda nüks izlenmedi. ***(Türk Kardiyol Dern Arş 2004; 32: 393-396)***

Anahtar kelimeler: *İdiyopatik ventriküler taşikardi, radyofrekans ablasyonu, sol ventrikül çıkış yolu*

Ventricular tachycardia (VT) in patients without structural heart disease is generally benign and the site of origin delineates the clinical and therapeutic approaches. Right ventricular outflow tract (RVOT) and left ventricular fascicular VT are the representative of this group of patients. Beta blockers and verapamil are the first-line therapeutic options, respectively. Ventricular tachycardias originating from the left ventricular outflow tract (LVOT) are of special interest in their origin and rarity. We presented a case with LVOT-VT and its successful ablation.

CASE PRESENTATION

A 21 years-old man presented with frequent episodes of palpitations and presyncope. A 12-Lead

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ECG revealed a wide complex tachycardia at a rate of 144 bpm. The axis of the ventricular activation was inferior and rightward causing positive QRS in inferior leads and negative in aVL and I. A precordial R wave transition was also observed at V1. No S wave was detected in either V5 or V6 (Figure 1). The tachycardia was terminated spontaneously and revealed normal sinus rhythm without preexcitation with frequent premature ventricular beats (PVB) with the same morphology and axis as the presenting tachycardia. He denied any syncope but experienced frequent episodes of palpitation and presyncope. He was on beta blocker without any success. No structural abnormalities were found by physical and echocardiographic examination.

He underwent an electrophysiologic study. The clinical sustained VT was reproducibly inducible with programmed ventricular stimulation and diagnosis was confirmed by AV dissociation. He also presented frequent PVBs with the same morphology and axis as the ventricular tachycardia throughout the study. Radiofrequency ablation catheter was used for mapping the activation sequence and the timing of the ventricular activation. Local electrogram from the RVOT was far behind the earliest QRS during VT. The left ventricular outflow was mapped for the earliest ventricular activation during VT and PVB. The first ventricular activation was recorded within the Valsalva of the left coronary sinus. First, a couple of unsuccessful radiofrequency application was delivered just under the left cusp, since this location would be safer. Then with the guidance of coronary angiography, focal activation of the tachycardia could be identified by the earliest local activation within the aortic sinus of Valsalva during the tachycardia. This was also confirmed by pace-mapping. A diastolic potential was also recorded with an exit block concomitant with the termination of VT. Single radiofrequency application resulted with the termination of VT in 1.7 sec at this point where the local ventricular activity preceded the surface ECG by 40 ms (Figure 2). Proximity of the ablation catheter to the left main coronary artery emphasizes the importance of the guidance of coronary angiogram, since an acute occlusion of the main coronary artery would be catastrophic in such a patient (Figure 3).

DISCUSSION

Left ventricular outflow tract VT represents a small portion of all tachycardias arising from the ventricular outflow. A recent study reviewed 68 articles and a total of 748 patients with idiopathic VT. Only 8% patients had LVOT tachycardias [1]. Among LVOT VT, only a small portion arises from the Valsalva of the left coronary sinus as in our case. The basic electrocardiographic landmarks of LVOT-VT were as follows: [1] an S wave in lead I; [2] a precordial R wave transition at V1 or V2. Coro-

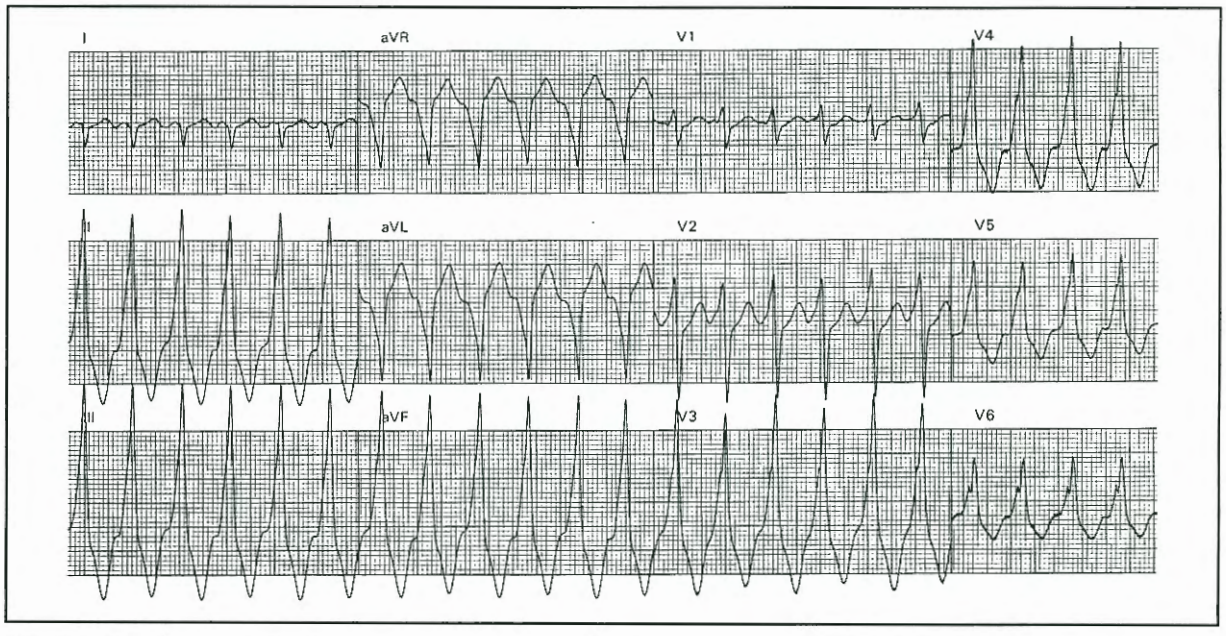

Figure 1. The clinical ventricular tachycardia at a rate of 144 bpm.

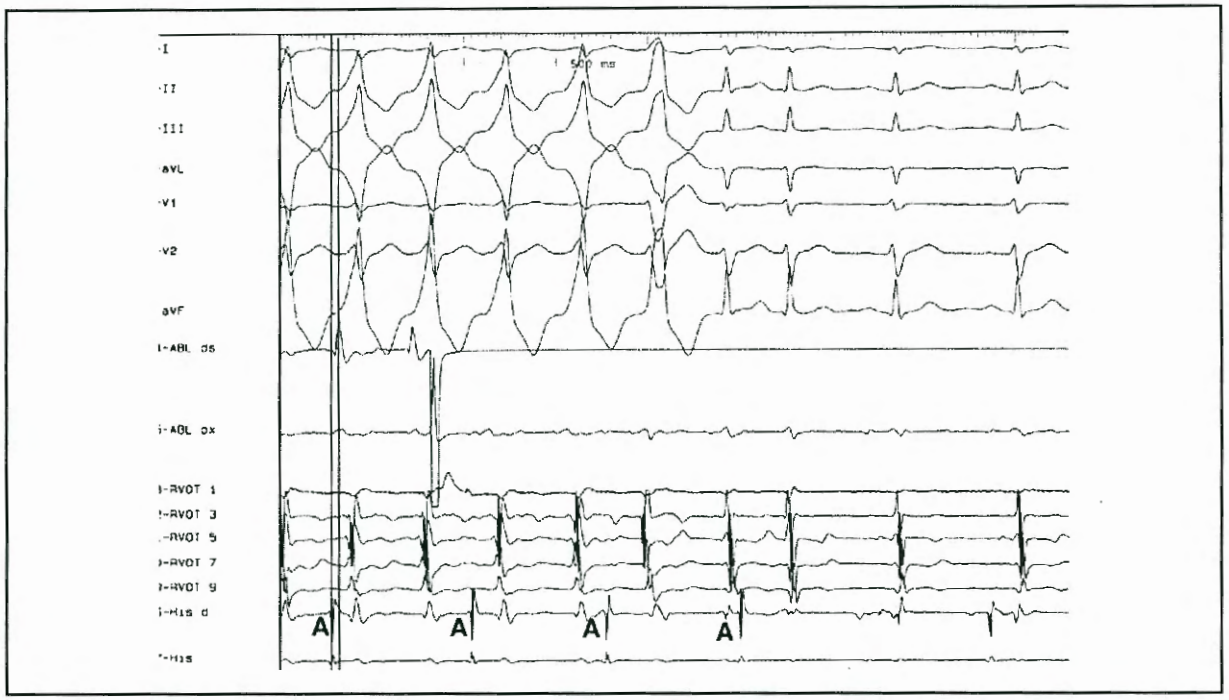

Figure 2. AV dissociation during the wide complex tachycardia confirms the diagnosis of VT. The termination of the VT soon after radiofrequency energy delivered (arrow) via the ablation catheter. The local electrogram from the ablation catheter at the successful site preceded the surface QRS by 40 ms (the time interval between vertical lines). A = atrial activity. Abl ds/prox = electrograms recoded from distal and proximal pair of electrodes from the ablation catheter, respectively. His d/His = electrograms recorded from distal and proximal pair of electrodes from the His catheter, respectively. RVOT 9, RVOT 7, RVOT 5, RVOT 3 and RVOT 1 = bipolar leads from most proximal (9) to most distal decapolar catheter located at the outflow of right ventricle.

nary cusp localization of the LVOT-VT was predicted only by the absence of s wave in either V5 or V6 in one study [2].

In a recent study, Ito and al [3] developed an algorithm correlating 12-lead ECG findings with the catheter ablation site in 80 patients with outflow tract VT and tested prospectively in 88 patients. Ventricular tachycardia or PVBs originating from the left coronary sinus of Valsalva was predicted by 1) absence of a S wave ≥0.1 mV in lead V6, 2) precordial transition zone not ≥ V4, 3) R/S ratio not < 0.3 in leads V1 and V2, and 4) the ratio of Q wave amplitude in aVL/aVR not > 1.4 and the S wave amplitude in V1 not >1.2 mV. When we apply these criteria to our case's ECG findings during VT: 1) no S wave in lead V6, 2) precordial transition zone < V3, 3) R/S ratio of 3/5 and 10/14 in leads V1 and V2, respectively, 4) Q:aVL/aVR ratio of 16/13, and 5) S amplitude of 5 mV in lead V1 (the only criteria which did not fulfill the above criteria), the origin of the first ventricular activation was localized as the left ventricular epicardium around the transitional area from the cardiac vein to the anterior interventricular vein according to Ito et al [3]. The sensitivity of this algorithm in correctly detecting the origin of VT as left coronary sinus of Valsalva was found only 80%. The reason may be secondary to the inherent definition of the origin of left ventricular epicardial VT, which was defined as unsuccessfully ablated from the left sinus of Valsalva.

Kanagaratnam et al. [4] mapped normal heart, left bundle branch block, inferior axis VT both endo- and epicardially in patients with previously failed ablation. Earliest ventricular activation was noted in the epicardium and the aortic cusps. All patients were successfully ablated from the aortic sinuses of Valsalva. The electro-

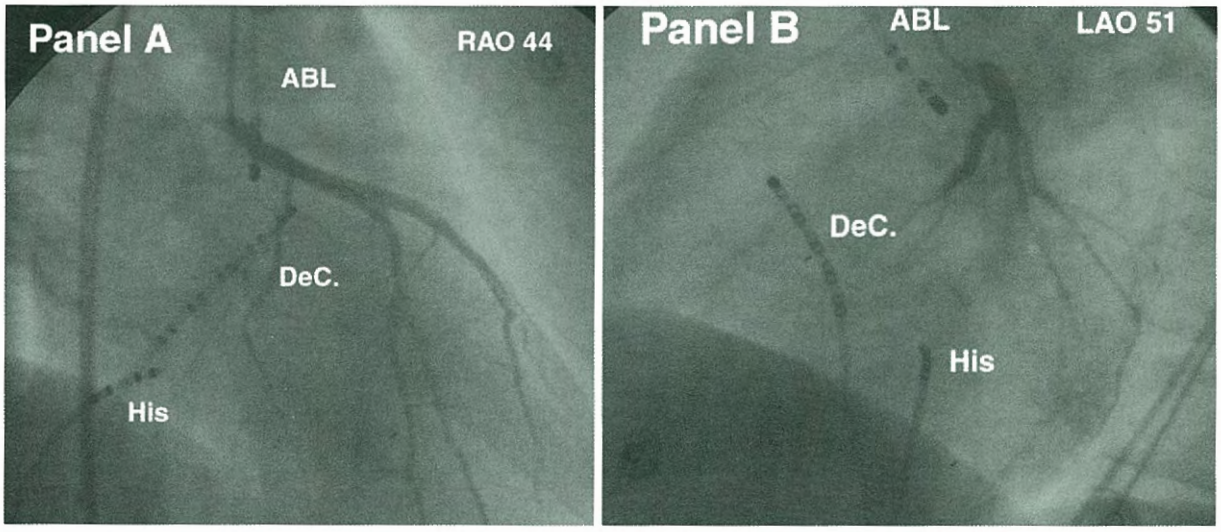

Figure 3. Radiographic localization of the ablation catheter at successful site in the Valsalva of the left coronary cusp and its relationship with the left main coronary artery in RAO 44° (Panel A) and in LAO 51° (Panel B) positions. ABL = ablation catheter, DeC. = decapolar catheter, His = His catheter.

cardiographic pattern associated with this VT was left bundle branch block, inferior axis and early precordial transition with Rs or R in V2 or V3. Ventricular tachycardia from the left sinus had rS pattern in lead I as in our case, and VT from the noncoronary sinus had a notched R wave in lead I.

Ouyang et al [5] revealed that R-wave duration and R/S-wave amplitude in leads V1 and V2 were significantly higher in VTs originating from coronary sinus cusps when compared to those originating from RVOT. Cut-off values for an R-wave duration index ≥50% and for an R/S-wave amplitude index ≥30% identifed 85% of patients with VT originating from the aortic sinus cusp. R-wave duration index in our case was 71% and R/S-wave amplitude index was 83% in lead V2, supporting the origin of VT from left sided coronary cusps.

Catheter ablation of LVOT-VT is rarely described because a safe ablation technique at this site has been challenging, and serious complications may occur. This case report emphasizes the important guidance of coronary angiogram in the ablation of VT arising from Valsalva of the coronary cusps.

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