The coexistence of outflow tract ventricular tachycardia originating from the left aortic sinus cusp and atrioventricular nodal reentrant tachycardia: catheter ablation in the same session

Sol aortik sinus kuspsiinden kaynaklanan ventriküler çıkış yolu taşkırdisi ve aтриoventriküler nodal re-entrant taşkırdanın aynı seansta kateter ablasyonu

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Repetitive monomorphic outflow tract ventricular tachycardia originating from the left aortic sinus cusp and typical atrioventricular nodal reentrant tachycardia in a 23-year-old male patient were ablated in the same session. No complication was observed during and after ablation. To our knowledge, this is the first case of both tachycardias in the same patient.

Key words: Catheter ablation; electrocardiography; tachycardia, atrioventricular nodal reentry; tachycardia, ventricular.

Outflow tract ventricular tachycardia (VT) originating from the left aortic sinus cusp (LASC) is relatively rare among VTs.⁴ The coexistence of outflow tract VT from the LASC and typical atrioventricular nodal reentrant tachycardia (AVNRT) has not previously been reported. Radiofrequency catheter ablation is the crucial treatment method for both tachycardias, but the ablation strategy has not been well-established in dual tachycardias. In this report, we presented a patient who underwent successful radiofrequency catheter ablation for outflow tract VT from the LASC and AVNRT in the same session.

CASE REPORT

A 23-year-old man with palpitations for 10 years had been receiving multiple antiarrhythmic drugs without adequate control of tachycardia attacks. A 12-lead electrocardiogram (ECG) showed a repetitive monomorphic VT of left bundle branch block (LBBB)-like morphology, right axis deviation, negative QRS in lead aVL, no S wave in V6, R/S<1.0 in lead I and RS transition zone in lead V3 (Fig. 1). Echocardiography revealed normal structure and function. After informed consent was obtained, all antiarrhythmic drugs were discontinued for elimination of at least five half-lives of all drugs until the electrophysiologic study.

Catheters were inserted through the femoral vein to the high right atrium, His bundle, coronary sinus (CS), and right ventricular outflow tract (RVOT). At baseline, nonsustained episodes of VT were present. Basal cycles, atrio-His (AH), and His-ventricle (HV) intervals were calculated as 780 ms, 92 ms, and 42 ms, respectively. A cycle length of 380 ms sustained narrow QRS tachycardia with a short ventriculoatrial (VA) interval and jumping at AH interval was induced with programmed stimulation from the proximal CS (Fig. 2). The ventriculoatrial interval during
tachycardia suggests the diagnosis of AVNRT. In addition, the presence of dual physiology in the beginning of tachycardia and occurrence of retrograde decremental conduction after stimulation of the right ventricle may exclude the possibility of concealed conduction. Atrial activation could not be preceded by ventricular extra stimulus technique when His was refractory during tachycardia. These results were highly suggestive for AVNRT. Ventricular tachycardia was suppressed spontaneously after the induction of AVNRT. Then, AVNRT was stopped by pacing with a cycle length of 330 ms from the proximal CS. Since he had permanent VT without programmed electrical stimulation, VT was not stimulated again. A 7-French quadripolar ablation catheter with a 4-mm tip electrode (Marinr, Medtronic, San Jose, California, USA) was introduced into the right ventricle retrogradely to map the VT through the

Figure 1. An 12-lead ECG showing left bundle branch block-like morphology, right axis deviation, negative QRS in lead aVL, R/S<1.0 in lead I, and RS transition zone in lead V3.

Figure 2. Typical atroventricular nodal reentrant tachycardia with jumping at atrio-His and short ventriculoatrial interval. Surface ECG leads I, II, aVF and V1, and intracardiac electrograms from quadripolar catheters positioned in the coronary sinus (CSd: Distal coronary sinus; CS2: Coronary sinus 2; CS3: Coronary sinus 3; CS4: Coronary sinus 4; CSp: Proximal coronary sinus) and anteroseptal tricuspid valve annulus (HISd: Distal His; HISp: Proximal His).
femoral vein. Pace mapping in the endocardial region of the RVOT failed to reach the early transition zone and the local early activation time was never less than -15 ms. Then we decided to map the VT in the left ventricular outflow tract (LVOT). The earliest site of ventricular activation was observed just above the base of the LASC. The ventricular ECG at the LASC preceded the QRS of VT by 42 ms (Fig. 3) and pace mapping at this side produced the same QRS during VT (Fig. 4). The surface ECG (Fig. 1) and the ECG during ablation (Fig. 4) differed in that the former exhibited an RS complex in V1, while the latter

Figure 3. The ventricular electrocardiogram at the left aortic cusp preceded the QRS of the spontaneous ventricular tachycardia by 42 ms.

Figure 4. Twelve-lead ECG recording showing local ventricular activation (A) during spontaneous ventricular tachycardia and identical pace mapping (B) from the successful site above the left aortic cusp.
a QS wave in V1. The reason for this was that we placed the precordial electrodes in the upper position on the chest to see the fluoroscopic views clearly during the procedure. Pace mapping revealed a focus about 11 mm beneath the origin of the left coronary artery. Radiofrequency catheter ablation was performed under the guidance of left coronary angiography to avoid damage to the coronary artery (Fig. 5). Radiofrequency catheter ablation with a power of 50 watt at 52 °C at this site immediately abolished VT. A subsequent ablation with a power of 50 watt at 55-56 °C at slow pathway of the AV node was performed, since AVNRT with jumping at AH interval was reproducibly induced by control atrial pacing. No complication was observed during and after ablation.

**DISCUSSION**

Although the coexistence of LVOT VT and AVNRT has been reported only in one patient, the coexistence of LASC VT and typical AVNRT has not previously been reported. Kautzner et al. reported coincidence of AVNRT and RVOT VT in seven cases. In both AVNRT and RVOT VT, arrhythmia originates from the endocardium, whereas the origin of aortic cusp VT is the epicardium. The AVNRT and outflow tract VT have different electrophysio-

or epicardial origin. The coexistence of these two tachycardias may be causal. Hasdemir et al. showed that muscles of both ventricles extend beyond the semilunar valves into the aorta and pulmonary artery. The ventricular muscle extensions may be a potential substrate for outflow tract VT.

Outflow tract VT with an LBBB-like morphology and inferior axis usually originates from the RVOT, and less frequently from the LVOT or aortic cusp. The differentiation of aortic cusp origin VT and RVOT VT is often difficult due to very close proximity of both arrhythmia localizations. Recently, three studies have described several 12-lead electrocardiographic features that can be used to identify the arrhythmia origin from the RVOT and the aortic cusp. Ouyang et al. reported that the ratio of R/QRS wave widths and the ratio of R/S wave amplitudes in V1 and V2 were significantly lower in RVOT VT than those in left or right aortic cusp origin VT. Kanagaratnam et al. suggested that patients with LBBB, inferior axis, and early precordial transition with Rs or R in V2 or V3 could be ablated from either the left or the non-coronary aortic sinus of Valsalva. They also described that patients with VT from the left sinus had an Rs pattern in lead I, while those having VT from the non-coronary sinus had a notched R wave in lead I. Tada et al. reported that patients with VT originating from the left ventricle epicardium had a prominent, tall R-wave in the inferior leads, an R wave in V1 and an S wave in V2, precordial R-wave transition in V2-4, a deep QS wave in aVL, and no S-wave in V6. Krebs et al. stated that VT with transition in lead V2 or V3 could not be ablated successfully from the right side. It has also been shown that some of these epicardial origin VTs can be ablated from the left sinus of Valsalva. In our patient, ECG findings were compatible with those described by Tada et al.

Ablation of dual tachycardias in the same session still remains questionable. There is also no consensus as to which tachycardia would be ablated first. The presence of AVNRT may cause VT induction and the ablation may control VT attacks. In our case, after stopping AVNRT by atrial overdrive stimulation, VT was still present. We ablated both tachycardias in the same session to prevent VT attacks. We ablated the VT first to obtain an adequate intracardiac electrogram of atrium/ventricle and to avoid inadequate catheter stabilization at slow pathway during VT. The potential target for ablation of VT originating from the coronary cusps was very close to the coronary arteries. Although there are some poten-
tial risks for aortic valve and coronary artery damage, LVOT VT can be successfully ablated from the coronary cusps.\cite{4,10} Coronary artery cannulation is recommended to prevent these injuries.\cite{10} We ablated the arrhythmia focus in the LASC by a guiding catheter in the left coronary artery.

In conclusion, AVNRT and LASC VT may coexist despite diverse anatomical and electrophysiological mechanisms. The differential diagnosis of aortic cusp origin VT and RVOT VT is important because of different ablation techniques. Both tachycardias should be ablated in the same session, with ablation of clinical arrhythmia in the first place.

REFERENCES