Successful Radiofrequency Current Catheter Ablation of Accessory Atrioventricular Pathway After Tricuspid Replacement in Ebstein’s Anomaly

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A 15-year-old female with Ebstein’s anomaly was referred to hospital for radiofrequency (RF) current catheter ablation of her refractory paroxysmal supraventricular tachycardia (PSVT) after tricuspid valve replacement. A surface ECG showed ventricular preexcitation of type B Wolff-Parkinson-White (WPW) syndrome. In a baseline electrophysiological study, two types of PSVT with left and right bundle branch block (LBBB and RBBB) configurations were induced. The LBBB type was antidromic and the RBBB type was orthodromic atrioventricular reciprocating tachycardia (AVRT) with a right posterolateral accessory pathway. RF current was successfully delivered at the posterolateral site above the prosthetic valve (V-delta interval = ~30 msec). The patient has been free from arrhythmias during a follow-up period of 9 months. RF current ablation seems to be useful for AVRT patients with corrected Ebstein’s anomaly. (Jpn Circ J 1998; 62: 791–793)

Key Words: Ebstein’s anomaly; Wolff-Parkinson-White syndrome; Radiofrequency catheter ablation

In Ebstein’s anomaly, atrioventricular reciprocating tachycardia (AVRT) due to atrioventricular accessory pathway (AP) shows a high incidence of 20–30%.1,2 APs are commonly found to be multiple and on the tricuspid side.2,3 Although radiofrequency (RF) current catheter ablation is now widely adopted to interrupt APs, there have been few reports describing RF ablation of APs in Ebstein’s anomaly patients after surgical correction. We report a case of successful RF current AP ablation in a patient after tricuspid valve replacement and provide anatomical and electrophysiological insights into atrioventricular connection in this anomaly.

Case Report

A 15-year-old female with recurrent palpitation was referred to hospital for treatment of paroxysmal supraventricular tachycardia (PSVT). She had been diagnosed at birth as having Ebstein’s anomaly. At the age of 9 years, because of severe tricuspid regurgitation, she underwent bio-prosthetic tricuspid valve replacement (#31 Carpenter-Edwards) with suture closure of the atrial septal defect. A 12-lead ECG showed no pre-excitation before the operation. One year after the surgery, she developed palpitations and syncope attacks, and PSVT due to Wolff-Parkinson-White (WPW) syndrome (type B) was diagnosed.

After written informed consent was obtained, an electrophysiological study was performed in the unsedated and postabsorptive state using the standard technique. All the drugs were discontinued for the period of at least their 5 half-lives. Programmed electrical stimulation was performed at high right atrium (HRA) and right ventricular apex (RVA). Atrioventricular (AV) conduction did not show any decrement. In contrast, ventriculoatrial (VA) conduction was decremental, although the rate-dependent prolongation of VA conduction was relatively small (~40 msec). Two types of wide QRS tachycardia with QRS configurations showing left bundle branch block (LBBB) and right bundle branch block (RBBB) types were induced by programmed stimulation (single premature stimulus from HRA; Fig 1). Cycle length in the LBBB-type tachycardia was 340 msec and that in the RBBB-type tachycardia was 360 msec.

Activation mapping was performed around the anatomical tricuspid ring, where the valve was replaced, during sinus rhythm and revealed that the earliest ventricular activation relative to the onset of the delta wave at the posterolateral site (~20 msec). During LBBB tachycardia, the earliest ventricular activation was found at the same site and that in sinus rhythm, and the earliest retrograde activation site was His bundle (HBE) (Fig 2). During RBBB tachycardia, activation was conducted through the His bundle, and the earliest retrograde activation was found at the posterolateral site of the right AV groove (Fig 2). Thus, the LBBB-type tachycardia was antidromic AVRT, and the RBBB-type was orthodromic AVRT with both types involving the right posterolateral AP.

Four applications of RF currents (40 W, Radionics INC, model RFG-3B generator system) were delivered to the posterolateral wall close to the prosthetic valve (V-delta interval = ~20 msec), but were unsuccessful. Further mapping using a long guiding sheath (Swartz SR2, Daig Co, USA), revealed another target site in the area about 8 mm away from the prosthetic valve, where the V-delta

Fig 1. Surface ECGs during PSVT. Left: LBBB-type configuration. Right: RBBB-type configuration.

Fig 2. Surface ECG (leads II and V1) and intracardiac electrogram during tachycardia. Left: LBBB-type configuration. The ablating catheter (ABL) was located at the posterolateral site of anatomical tricuspid annulus (unsuccessful site). Right: RBBB-type configuration. The ablation catheter (ABL) was located at the atrial site of the electrical tricuspid annulus (close to the successful ablation site). HRA, high right atrium; HBE, His bundle.

Fig 3. Fluoroscopic images on right and left anterior oblique (RAO and LAO) showed the successful ablation site. The shadow of the prosthetic valve is seen in the centre of the panel. ABL, ablating catheter; RV, right ventricle.
interval was –30 msec, and Kent potential was clearly recorded (Fig 2). After 5 additional applications of RF currents (40 W), the AP was successfully ablated, and AV conduction showed smooth and decremental property. There was no AP conduction with isoproterenol infusion.

Discussion

In Ebstein’s anomaly, the association of pre-excitation syndrome and AVRT is fatal. RF ablation has been considered as the primary therapeutic option for AVRT in this anomaly, but to our knowledge, there is only one report of this procedure being applied after tricuspid valve replacement.

In the patient reported here, detailed AP localization was impaired because local electrograms recorded at the anatomical annulus were abnormal and demonstrated continuously fragmented activities. These findings have been also noted in uncorrected Ebstein’s anomaly. In contrast, at the optimal ablation site, approximately 8 mm above the level of the prosthetic valve, the amplitude of the ventricular electrogram was twice of the atrial electrogram (Fig 3). The electrical AV junction was therefore distinct from the anatomical junction, and the prosthetic valve was sutured between the anatomical and electrical AV grooves. The atrialized right ventricle made it difficult to localize the ablation site precisely. The Swartz guiding sheath was found to be useful in obtaining stable positioning of the ablation catheter.

The retrograde conduction was decremental in our patient although this property has been reported infrequently for APs. The site generating the conduction delay, however, remains unknown. It may occur in the atrialized ventricular myocardium between the AP and recording electrode.

In conclusion, RF ablation would be the first therapeutic choice for AVRT patients with corrected Ebstein’s anomaly. In this subset of patients, more systematic mapping is needed because of a complex geometry and abnormal morphology of endocardial activation potentials.

References