Ventricular Tachycardia With an Outflow Tract Septal Origin After Repair of Double Outlet Right Ventricle

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A 12-year-old boy born with double outlet right ventricle (RV) developed sustained ventricular tachycardia (VT) 6 years after the corrective surgery and underwent electrophysiologic testing and catheter ablation. Electroanatomic mapping of the right and left ventricles during the VT revealed a centrifugal activation from the outflow tract septum. Though an excellent pace map was obtained in the RV, successful ablation was achieved on the left side. These findings suggested that the VT origin might have been located in the intramural region of the ventricular outflow tract septum with a preferential breakout site in the RV outflow tract. (Circ J 2008; 72: 496–499)

Key Words: Double outlet right ventricle; Intramural origin; Radiofrequency catheter ablation; Ventricular outflow tract; Ventricular tachycardia

Case Report

A 12-year-old boy was born with DORV with a subpulmonic ventricular septal defect (VSD) (Taussig-Bing type) and coarctation of the aorta. He underwent complete surgical repair, including side-by-side transposition of the great arteries, Kawashima operation (tunneling from the left ventricle (LV) to the aorta and from the right ventricle (RV) to the pulmonary artery), subaortic fibrous resection, and transannular patch for subpulmonary stenosis by the age of 6. He developed sustained VT with presyncope, which required emergency cardioversion 6 years after the surgery, and he was admitted for catheter ablation of the VT.

After written informed consent was obtained, an electrophysiologic study was performed. Right ventriculography revealed a dilated RV and left ventriculography revealed a slightly reduced LV systolic function (ejection fraction = 48%) (Fig 1). At baseline, the heart rhythm was sinus rhythm and the 12-lead electrocardiogram showed an interventricular conduction delay (QRS duration = 150 ms) and left-axis deviation (Fig 2). For mapping and pacing, multipolar electrode catheters were positioned in the coronary sinus and His Bundle region and a 7.5-Fr 3.5-mm tip irrigated ablation catheter (NAVI-STAR™ THERMOCOOL™, Medtronic, Inc) was positioned in the left ventricle. A direct current RF catheter ablation was performed guided by electroanatomic mapping (Fig 3).

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Outflow Septal VT After Repair of DORV

Biosense Webster, Diamond Bar, CA, USA) was introduced into the RV via the right femoral vein. The clinical VT (left bundle branch block with a right inferior axis QRS morphology, cycle length = 250 ms) was induced by programmed ventricular stimulation from the RV with an isoproterenol infusion (Fig 2). Because the VT did not become sustained at first, pace mapping in the RV was performed. Pacing from the RV outflow tract septum reproduced an excellent pace map (Fig 2). Aggressive induction of the VT was attempted again and sustained clinical VT was successfully induced. Electroanatomic contact mapping of the RV was then performed during the VT, which revealed a centrifugal activation from the outflow tract septum where the voltage of the local ventricular electrogram was greater than 1.5 mV (Fig 3). The earliest activation site during the VT was identical to the site where the excellent pace map was obtained. However, a few RF applications with a target temperature of less than 40°C and power output titrated up to 40 W were delivered at the earliest activation site, with no interruption of the VT, but prolongation of the VT cycle length and a slight change in the QRS morphology of the VT occurred (Fig 4). Next, electroanatomic mapping of the LV was performed via a retrograde transaortic approach during the VT. The activation map in the LV also showed centrifugal activation from the outflow tract septum (Fig 3). RF was delivered at that site in the same manner as in the

Fig 2. Twelve-lead electrocardiogram. PM RV, pace mapping in the right ventricle; SR, sinus rhythm; VT, ventricular tachycardia.

Fig 3. Activation (Left) and voltage (Right) maps of the ventricles during ventricular tachycardia (VT). The activation map of the left ventricle (LV) was obtained after radiofrequency ablation in the right ventricle (RV), with the same reference of the QRS as before the ablation. Though the earliest ventricular activation in the LV seemed later than that in the RV, it was probably because of the slight change in the QRS morphology of the VT after the ablation in the RV. The purple indicates the area with a voltage on the local bipolar electrogram >1.5 mV, and the gray shows the great arteries in the voltage map. Ao, aorta; PA, pulmonary artery.
RV, with an interruption of the VT (Fig 4). An attenuated local ventricular electrogram with the same coupling interval was still observed at the successful ablation site immediately after termination of the VT (Fig 4). Thereafter, no VT could be induced, despite programmed electrical stimulation as well as isoproterenol infusion. During more than 3 months of follow-up, the patient has been free of any ventricular arrhythmic episodes without any antiarrhythmic drugs.

**Discussion**

In this case, the VT had a focal mechanism that originated from the outflow tract septum after complete surgical repair of Taussig-Bing type DORV. Though it might actually be impossible to have determined whether the successful ablation site could have been in an anatomical RV site functionally divided by a VSD patch or an anatomical LV site, successful ablation was achieved by a left-sided approach in the low voltage area in the outflow tract septum, which was presumably created by an operative suture along the VSD patch (Fig 5). As the pace mapping in the RV demonstrated, the presumed preferential breakout site was located in the normal voltage area in the RV outflow tract. These findings could explain the preferential conduction from the left-side origin to the right-side breakout site.

The detailed mechanism of the VT could not be investigated because the VT was difficult to induce. Actually, from the results of electrophysiologic study it was difficult to determine whether the VT mechanism was automaticity, triggered activity or micro-reentry. In consideration of the successful ablation site, the VT mechanism was most likely to have been micro-reentry that involved a slow conduction zone created by an operative suture along the VSD patch.

Activity of the VT origin was still observed at the successful ablation site immediately after termination of the VT, which suggests that the termination of the VT might have been achieved by isolation of the VT origin. Therefore, the VT origin might have been located in the intramural region of the ventricular outflow tract septum. Niwano et al speculated that VT occurring after complete surgical repair of DORV originated from the intramural region of the ventricu-
lar outflow tract septum because in their case a transseptal direct countershock applied using 2 catheters successfully eliminated the VT. Therefore, the intramural region of the ventricular outflow tract septum may be a major source of focal VT origin after complete surgical repair of DORV.

There are some other similar findings between the cases in the report by Niwano et al\(^2\) and ours. The VT developed 8 years after corrective surgery in their case and 6 years later in ours. The VT origin was located within the interventricular septum near the patch suture around the VSD in both cases, which suggests that structural remodeling after the surgery may have been responsible for the development of the VT.

References