A Case of Radiofrequency Catheter Ablation of Atrioventricular Nodal Reentrant Tachycardia Using Transseptal Approach, Guided by Electro-Anatomical Mapping System

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A 65 year-old female was admitted to our hospital because of syncope. She has been previously diagnosed with dilated cardiomyopathy. Twelve-lead electrocardiogram (ECG) presented narrow QRS tachycardia. Electrophysiological study and radiofrequency catheter ablation was performed. Under isoproterenol infusion, supraventricular tachycardia (SVT) was easily induced followed by critical AH interval. The mechanism of SVT was diagnosed with slow-fast atrioventricular nodal reentrant tachycardia (AVNRT). We tried slow-pathway ablation from venous approach targeted at the triangle of Koch using electrogram guide and anatomical approach. Slow-pathway conduction was not eliminated, however, and SVT still could be easily induced. Therefore we delivered radiofrequency (RF) from the left atrial (LA) septum by transseptal approach using an electro-anatomical mapping (EAM) system. After RF application at mid-septum from the LA, the slow-pathway conduction was modified and SVT could not be induced.

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Introduction

Radiofrequency catheter ablation of slow-fast atrioventricular nodal reentrant tachycardia (AVNRT) has been reported earlier, and is generally accepted as a standard treatment. RF application targeting the right atrial (RA) septum, including the triangle of Koch, can ablate the slow pathway with an electrogram-guided or anatomical approach.1–3) Although most cases can be treated by this approach, there are a few reports on slow pathway ablation from the left atrium (LA) using a transseptal approach to treat slow-fast AVNRT which could not be treated by conventional ablation methods.4) We used an electro-anatomical mapping (EAM) system as a navigation tool for slow-pathway ablation from the LA. This is the first report of a successful slow pathway ablation using the transseptal approach guided by an EAM system.
Case Report

A 65-year-old female who had been diagnosed with dilated cardiomyopathy was admitted to our hospital complaining of syncope. Her 12-lead surface ECG presented narrow QRS tachycardia and heart rate of 180 ppm, which coincided with the QRS morphology of her baseline ECG (Figure 1). Trans-thoracic echocardiography showed poor left ventricular function, slightly dilated left ventricle dimension, left ventricular ejection fraction (LVEF) of 29%, and left ventricular end-diastolic diameter (LVDd) of 52 mm which decreased to 45 mm during sinus rhythm. Other physical examinations and chest X-rays were within normal range. SVT spontaneously converted to sinus rhythm, but the SVT and episodes of syncope continued. After written informed consent was obtained, an electrophysiological study was performed. Multipolar electrode catheters were introduced percutaneously, and positioned at high right atrium (HRA), His bundle potential at AV junction (His), right ventricle apex (RVA), and coronary sinus (CS). Surface and intracardiac electrocardiograms from catheters were amplified, filtered (30–500 Hz), monitored, recorded, and analyzed by using EP-WorkMate (St. Jude Medical Inc.). Electrical stimulation was performed using EP3, a digital stimulator (St. Jude Medical Inc.). Dual atrioventricular (AV) nodal physiology, i.e. the jump up phenomena, was not present when applying the pacing maneuver at baseline. But, the SVT was easily induced from critical AH interval by burst pacing from HRA under isoproterenol infusion (Figure 2). AH interval was prolonged from 57 to 266 msec at the initiation of SVT. These short and long AH intervals at the initiation indicate fast- and slow-pathway conduction and dual AV nodal physiology. Ventricular stimulation delivered during His refractoriness could not reset the SVT. We diagnosed this SVT as slow-fast AVNRT, and tried slow-pathway ablation at RA septum, including the triangle of Koch. Many RF applications were attempted even inside the CS, within 10 mm from its ostium, and near the AV node where a small His potential was recorded, targeting at 55°C at a maximum setting of 30 W (Ablaze, Japan Lifeline

Figure 1 12-lead electrocardiogram. 1-A represents baseline ECG and 1-B represents narrow QRS tachycardia. QRS morphology coincides with baseline ECG.
Co., Ltd.). RF applications were delivered many times; however the SVT was still easily induced and even an accelerated junctional rhythm (AJR) never emerged. We then aborted ablation from the RA and switched to the LA. The ablation catheter was introduced to the LA by transseptal puncture and positioned at a mid-to-posterior septal position of the mitral valve annulus. However, the ablation catheter was unstable and it was difficult to recognize the exact catheter position at this area. Therefore we selected the EAM system (CARTO, Biosense Webster Inc.) as a navigation tool. We were able to map the triangle of Koch from RA (Figure 3) which we used as anatomical landmark and to create a map from the LA. Finally, we were able to record the slow potential at the mid-septum from the LA (Figure 4). This slow potential was low in amplitude, dull and originating just after the atrial potential during sinus rhythm. RF application was delivered, targeted at 50°C within 20 W (Navistar, Biosense Webster Inc.). AJR emerged soon after the RF application. RF application was delivered for 60 seconds. After that, the SVT could not be induced. Still, dual AV nodal physiology was demonstrated by jump up phenomenon under isoproterenol infusion, but no echo remained. Therefore we delivered an additional RF application at the same site, but ventriculo-atrial interval prolonged during AJR and RF application stopped immediately. After that, electrophysiological study did not show any finding of permanent AV node injury. After a 1 year follow up, sinus rhythm has been maintained. LV function has not improved in spite of sinus rhythm maintenance.

Discussion

Usually, slow-fast AVNRT can be successfully treated by radiofrequency catheter ablation from the RA septum near the triangle of Koch. On the other hand, it has been reported that slow pathways cannot be ablated from the RA in less than 1% of slow-fast AVNRT. In these cases, catheter ablation was performed from the LA by transseptal approach or retrograde approach.4,5)
Shin Inoue et al. speculated on the relationship between electrophysiological slow pathways and anatomical posterior nodal extensions (PNE). Cases that cannot be treated by conventional ablation from the RA might be due to an anatomical variation in which only leftward PNE is present. In this case, we targeted the slow potential, and RF application targeted at this slow potential was able to modify the slow pathway conduction and suppress slow-fast AVNRT. However, we could not prove the relationship between PNE anatomy and the electrophysiological slow potential, and it is considered that slow
potential does not represent slow pathway conduction activity.

We believe that use of the EAM system was advantageous in this ablation case. The ablation catheter positioned in LA septum was unstable. In addition, ablation targeting an unusual site, the LA septal mitral valve annulus (MVA), made the attempt difficult. Therefore, we elected to use the EAM system as a navigation tool to place the ablation catheter at the target site. In previous reports, ablation sites in the LA were described only in the mid or posterior septum of the MVA. We were able to determine the anatomical position in association with the triangle of Koch, AV node of the RA, and the site where RF was delivered in the LA using the EAM system. The His potential could not be recorded from the LA. However, the distance between the successful RF site and the His potential recorded site in the RA was only 16 mm apart, and the successful site was located on the superior and posterior side of the site where the His potential was recorded. There is no previous report of a successful slow pathway ablation using the transseptal approach guided by EAM system, but we found this method effective and helpful.

This case was diagnosed as a dilated cardiomyopathy, and tachycardia induced cardiomyopathy is ruled out from clinical course after RFCA. Although the association of this heart disease with a left-side slow pathway has never been reported, this case might have some implications regarding the association of anatomical variation and electrophysiological variation.

References