Case Report

AV Nodal Reentrant Tachycardia in a Patient with Persistent Left Superior Vena Cava: Distinction between AV Nodal Versus Atrial Reentry

Terunobu Fukuda MD, Tetsuya Haruna MD, Hidehiro Ito MD, Kenichi Sasaki MD, Tomomi Abe MD, Eisaku Nakane MD, Shouichi Miyamoto MD, Kyoukun Uehara MD, Muneo Ooba MD, Kouji Ueyama MD, Moriai Inoko MD, Ryuji Nohara MD

Department of Cardiology, Kitano Hospital Medical Research Institute

A 75-year-old male presented with palpitation on exertion. He suffered from frequent tachycardia attacks. His 12-leads electrocardiogram showed irregular cycle lengths (400–550 ms) of tachycardia with occasional 2:1 atrioventricular conduction (thus AV reentry was excluded). He had a complex anatomy of persistent left superior vena cava (PLSVC)/enlarged coronary sinus (CS). The activation map in a 3-dimensional CARTO system (Biosense-Webster, USA) was merged with the multi-detector computed tomography image and revealed that the tachycardia spread centrifugally from the junction between the PLSVC and enlarged CS. However, delivery of radio frequency (RF) energy to the earliest atrial activation site did not affect the tachycardia. Finally, the tachycardia was diagnosed as a fast/slow type atrioventricular nodal reentrant tachycardia (AVNRT) because the tachycardia was cured only after the anterograde/retrograde AV conduction was disturbed by the application of RF energy to the posteroseptal perimital area, possibly due to the injury to the AV node. (J Arrhythmia 2010; 26: 134–139)

Key words: Persistent left superior vena cava, Atrial tachycardia, Enlarged coronary sinus, Radiofrequency catheter ablation

Introduction

During normal development, the left superior vena cava transforms into the Marshall vein/ligament and rarely remains intact as the persistent left superior vena cava (PLSVC), and connects to the coronary sinus (CS). Recently, the Marshall vein/ligaments have been reported to be associated with atrial arrhythmogenesis. On the other hand, it has been reported that the enlarged CS from the PLSVC could displace the compact atrioventricular (AV) node and the His-bundle. Therefore, much attention should be paid to avoid injury of the compact AV node and His-bundle during application of radio-frequency (RF) energy.

We experienced a type of fast/slow atrioventricular nodal reentrant tachycardia (AVNRT), which was difficult to differentiate from an atrial reentrant tachycardia in a patient with the PLSVC and enlarged CS. The AVNRT was distinguished from...
the atrial reentrant tachycardia as follows; the

tachycardia elimination was made possible only

after the (partial) damage to the anterograde/retro-

grade AV nodal pathways was obtained by the

application of RF energy to the AV junctional area.

A new 3-dimensional electroanatomical mapping

system (CARTO Merge, Boston, USA) was very

helpful in the RF catheter ablation procedure.

Case Report

A 75-year-old man complained of palpitation and
faintness on exertion. He underwent bypass graft
surgery for coarctation of the descending aorta and
right atrium (RA) isthmus catheter ablation for
common atrial flutter at the age of 65 and 68 years,
respectively. He had a 6-year-history of another
tachycardia, and his 12-lead electrocardiogram
(ECG) showed an irregular cycle length of the
supraventricular tachycardia with a 1:1 or infre-
quently a 2:1 AV ratio and right bundle branch
block. Axis in the P-wave morphology was biphasic
(+/−) on I, II, III, aVF, and V1 leads (Figure 1).

Chest X-ray showed an enlarged cardiac silhouette.
Contrast computed tomography of the thorax re-
vealed that the PLSVC was draining into the
enlarged CS and the RA.

Although the supraventricular tachycardia was
partially controlled with propafenone (300 mg/day),
pirmenol (100 mg/day), and carvedilol (5 mg/day),
he still suffered from palpitation and faintness on
exertion. In addition, the QRS duration increased
gradually. Echocardiography showed that the left
ventricle ejection fraction (LVEF) was reduced to
35% as compared to that in the previous study
(61%). The patient wished to discontinue all anti-
arrhythmic agents and undergo RF catheter ablation.

We obtained written informed consent from the
patient. Electrophysiological study and catheter
ablation were performed 3 days after discontinuation
of all anti-arrhythmic agents. First, the anatomy of
the enlarged CS and the RA was visualized by both
RA angiography and contrast infusion into the
PLSVC (Figures 2A and 2B). The junction between
the PLSVC and the enlarged CS, however, could not
be clearly identified. A decapolar 7-Fr electrode
catheter (Nihon Koden, Japan) was advanced into
the CS via the right internal jugular vein for
recording and pacing at both the RA and the CS. A
7-Fr Halo catheter (SJM, Irvin, USA) was introduced
into the RA via the right femoral vein and positioned
around the Tricuspid Annulus (TA). A 4-mm tip of
a Navistar catheter ( Biosense-Webster, USA) was
introduced into the RA and the enlarged CS through
the right femoral vein for 3-dimensional nonfluoro-
scopic, electroanatomical mapping (CARTO, Bio-
sense-Webstar). The His bundle electrogram could
not be identified around the usual area probably
because the enlarged CS might have displaced the
compact AV node and the proximal portion of the
His bundle. The tachycardia was easily induced by
either mechanical catheter contact or provocative

Figure 1 A 12-lead electrocardiogram of the ta-
chycardia at 25 mm/s paper speed.
Arrows indicate the P-wave of the tachycardia.
atrial pacing. Once it was induced, it incessantly terminated and reinitiated. The cycle length of the tachycardia varied from 420 ms to 500 ms with 1:1 or occasionally 2:1 AV conduction. Therefore, the conduction property of the anterograde/retrograde AV nodal pathways and the relation between the tachycardia origin and the location of the AV node could not be fully clarified. Intra-cardiac ECGs during the tachycardia showed an early atrial activation sequence in the mid CS despite changes in the cycle length. Within the distal to mid CS, multi-component electrograms were recorded. The earliest endocardial activation site was located at the top of the probable junction between the PLSVC and the CS, which was about 4.0 cm distal to the CS ostium (Figure 2C). The local electrograms preceded the onset of the P-wave by 28 ms and had a very sharp deflection as initial component (Figure 2E). The CARTO mapping in both the PLSVC/enlarged CS area and the RA demonstrated that the tachycardia propagated from the probable junction between the PLSVC and the CS into the RA in a centrifugal pattern (Figure 2D). The tachycardia was therefore diagnosed as an atrial tachycardia originating from the PLSVC/CS area due to local triggered activity or micro re-entry. Although RF energy (up to 20 W) was applied 3 times to the earliest site in a temperature controlled mode (maximum temperature: 50°C), there were few remarkable changes in the atrial activation sequence of the tachycardia. We speculated that the tachycardia might originate from the epicardial site of the left atrium (LA) and that the RF energy might not be high enough to affect the tachycardia foci. However, we were concerned that the application of more frequent or higher RF energy delivery could cause vascular damage. Therefore, the RF energy was applied from the endocardial site of the LA, which allowed us to increase the frequency and quantity of energy delivered to the tachycardia foci. The CARTO map of the LA was
merged with a multi-detector CT image before the ablation procedure. The merged image demonstrated that the tachycardia spread from the posteroseptal perimitral area, which was opposite to the earliest site in the CS over the LA wall (Figure 3A). The atrial electrogram at the earliest site in the LA preceded the onset of the P-wave by 20 ms (Figures 3B and 3C). The RF energy was applied to the earliest site in the LA at a maximum power of 35 W to achieve 55°C. Figure 4 demonstrates intracardiac electrograms during termination of the tachycardia. The final application of RF energy gradually slowed and eventually terminated the tachycardia. After the termination, the AV interval remarkably prolonged to 380 ms in the sinus rhythm. A Wenckebach pattern of AV block appeared at a pacing cycle length of 500 ms. During RV apical pacing, VA conduction was very poor and observed only in the presence of isoproterenol. Tachycardia was no longer inducible even during intravenous infusion of isoproterenol. These findings strongly suggested that the application of RF energy to the posteroseptal perimitral area injured the antegrade/retrograde AV nodal pathways, thereby eliminating the substrate for the tachycardia circuit. On the basis of these data, we concluded that the tachycardia mechanism was not atrial reentry but fast/slow type AV nodal reentry.

After 6 months of follow-up, the patient was free of tachycardias. There was no advent of complete AV block. The echocardiography revealed improvement in the LVEF (51%).

**Discussion**

In the embryonic heart, pacemaker cells are available near both sinus horn and common cardinal vein sites. During the development of the heart, the left site transforms to a series of developmental remnants, the Marshall vein/ligaments and the CS. These developmental remnants are reported to be associated with atrial arrhythmogenesis since
they retain their pacemaker function and ectopic automaticity. Especially in cases with PLSVC, more blood from the PLSVC could persistently stretch the CS wall and hence might cause increased arrhythmogenesis in the CS musculature.

Because of the irregularity in its cycle length and the occurrence of an incessant pattern, we first considered the tachycardia to be an atrial tachycardia that originated from the enlarged CS. Badhwar et al. elegantly described the electrophysiological features of the atrial tachycardia in the boundary area between the CS and the LA and the outcome after RF ablation. In most cases, the atrial tachycardia was successfully terminated by applying RF energy to the CS under guidance of a CS potential, which was characteristic of a discrete, sharp deflection and preceded the onset of the P-wave of the tachycardia. Application of RF energy to the endocardial site of the LA was not required. Likewise in our case, a very sharp potential was recorded in multi-component electrograms at the earliest site of the CS; however, application of RF energy at this site hardly affected the tachycardia. Eventually, the tachycardia was terminated by application of RF energy to the earliest site of the LA opposite to the earliest site of the CS. Recordings during the final RF application (Figure 4) revealed that the tachycardia was terminated (disappearance of the P-wave) after the 5th beat. This finding implies that application of RF energy might either eliminate the tachycardia foci or block the retrograde pathways within the reentry circuit. Immediately after termination, neither ventricular premature beat nor the first sinus beat was conducted through the AV node. The subsequent sinus beats passed through the AV node with prolonged AV interval. Although VA conduction was observed only in the presence of isoproterenol, tachycardia was no longer inducible. These findings strongly suggest that the anterograde/retrograde AV nodal pathways were likely to form the tachycardia circuit. Since the tachycardia sustained even in the presence of an AV block before application of RF energy, the tachycardia was finally diagnosed as a fast/slow type AVNRT with lower common pathway.

Okishige et al. reported AVNRT cases in patients with PLSVC, the enlarged CS due to the PLSVC might displace the compact AV node. They explained that during application of RF energy around the CS in patients with PLSVC much attention should be paid to avoid the occurrence of AV node injury and perforation of the CS. It has been suggested that application of RF energy to the left anterosetal or midseptal accessory pathways has a moderate risk to damage the compact AV node. In our case, there was also a possible risk that the application of relatively high RF energy to the posteroseptal perimitral area could injure the AV node.
node, because the pathways and/or the compact AV
node itself might be displaced; however, application
to the CS did not affect the pathways. Before the
final application of RF energy, it was difficult for us
to differentiate a fast/slow AVNRT from an atrial
tachycardia due to lack of information on the
relationship between the tachycardia origin and the
AV node. Recently it has been suggested that
the CS musculature is not only a source of atrial anrrhythmogenesis, but also may work as pathways
for the interatrial conduction system in sinus rhythm
or arrhythmias. Especially in AVNRT cases the CS
musculatures are potentially involved in the reen-
trant circuit. Therefore, we should have consid-
ered an uncommon type of AVNRT as the mecha-
nism of the tachycardia, and have made a greater
effort to identify the precise position of the AV node
before RF energy application to confirm the mech-
anism of the tachycardia, and to avoid injury of the
AV node.

Finally, in regard to the application of RF energy,
the new 3-dimensional mapping system CARTO
aided us in our case of a complex PLSVC/CS
anatomy.

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