Electrophysiological Manifestations of Rare Supra-ventricular Tachycardias with Concealed Nodo-ventricular Fibers

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Abstract

Background Nodo-ventricular(NV) fiber-related reentrant tachycardias are so rare that most of them were reported by case, while few reports have summarized their common and individual features. Objectives To clarify the electrophysiological mechanism of supra-ventricular tachycardias (SVT) related to concealed NV fibers. Methods and Results We studied the intra-cardiac electrograms during electrophysiological study of 3 cases of SVT concerning concealed NV fibers. Maneuvers including ventricular entrainments, His bundle refractory period ventricular stimuli, adenosine triphosphate injection and so on were done for differential diagnosis before ablation. Among these patients, one had AVNRT with a bystander NV fiber, the other 2 had NV fiber-mediated orthodromic reentrant tachycardias (NVRT). VA dissociation were observed during SVT in all with antegrade His bundle conduction sequence. His bundle refractory period ventricular stimuli reset tachycardias with resetting of the H-H interval advancing the V-V interval, suggesting the existence of an accessory pathway. The cycle length of an NVRT prolonged during the status of functional right bundle branch block. Multiple QRS fusion morphologies during ventricular entrainments on a fixed site could be observed. Conclusions Concealed NV fibers can mediate orthodromic SVT or be a bystander of AVNRT. V-A dissociation usually occur during such SVTs. An NV fiber not only expresses the characteristics of an AP, but also the characteristics of the AV node. Multiple QRS fusion morphologies during ventricular entrainments or His bundle refractory period ventricular stimuli on a fixed site can discriminate NV fibers from NF fibers.

Introduction

Nodo-ventricular (NV) fiber is a rare type of Mahaim's bundle which connects the atrio-ventricular (AV) node and the ventricular myocardium. In the light of low prevalence of Mahaim's bundle (<3%)[1], occurrence of NV fibers is even rare. Previous literatures about NV fibers were mainly case reports and not all of them were describe clearly. We met with 3 cases of NV fibers from 3 hospitals in the recent 3 years. We will clarify the electrophysiological mechanisms of these NV fibers by analyzing their similarities and individual characteristics.

Methods

Patient enrollment

In total 3 adult patients from 3 hospitals were diagnosed as SVT with an NV fiber by invasive electrophysiological study (EPS) from April 2016 to June 2019. All patients had signed informed consents for invasive EPS and ablation before the procedure.

Electrophysiological study

Two quadripolar mapping catheters including a His bundle catheter and a right ventricular apex (RVA) catheter were placed via the right femoral vein under fluoroscopy guidance. A decapolar (inter-electode spaces being 2-8-2 mm) coronary sinus (CS) vein catheter was placed via the left/right femoral vein. Intracardiac electrocardiograms during sinus rhythm and arrhythmias were analyzed. EPS techniques including burst pacing, programmed stimulation (S1, S1S2, S1S2S3), right ventricular apex (RVA) entrainment, and His bundle refractory period premature ventricular stimuli were used for differential diagnosis.

Ablation and procedure endpoints

Three-dimensional electro-anatomical mapping systems (Carto 3, Biosense Webster, Diamond Bar, CA, US and Ensite NAVX, Abbott, Minneapolis, MN, US) were used for these procedures. Temperature-controlled ablation catheters (Navistar, Biosense Webster, Diamond Bar, CA, US and Safire, Abbott, Minneapolis, MN, US) were applied (55-60°C, 40-50 W, 90-120 seconds). The ablation targets located on the AV nodal slow pathway area, within the lower one third of the triangle of Koch.

If the patients had passed EPS after initial ablation without any evidence of AP, they then underwent an adenosine triphosphate (ATP) test. Ventricular backup pacing by the RVA catheter was ready for protection against slow ventricular rhythm during the ATP test. An ATP bolus of 30 mg was then injected rapidly via peripheral vein to block the AV node. When AV nodal block was observed without antegrade A-V nodal conduction nor pre-excitation, ventricular pacing was further performed to exclude retrograde V-A conduction via a concealed AP. The procedure was concluded after confirmation by both EPS and the ATP test.

Follow-up

All three patients were followed up for at least 3 months after successful ablation. Surface ECGs were checked in a month after ablation to confirm that no clinically significant AV conduction block took place. If the patients complained of palpitation, they were informed to come back for further examination like tranesophageal atrial programmed pacing. Patients were also advised to go to the emergency room immediately whenever they suffered from symptoms related to SVT to record real-time surface ECGs. If the patient was confirmed to have recurrences, he/she was advised to undergo a repeat ablation procedure.

Results

These patients presented some common and some individual electrophysiological characteristics. Their electrophysiological phenomena were displayed in the following figures.

Patient 1

Patient 1 has been introduced detailly in a former publishment written by some of our authors [2]. He was a 25-year-old man who was otherwise healthy. His tachycardia could be induced by ventricular burst pacing, with a complete right bundle branch block (CRBBB) pattern, cycle length (CL) variating from 220 to 300 ms. But S1S2 and S1S2S3 stimulation of the atrium and the ventricle did not induce any abnormal phenomena. There were 3 types of VA relationships during tachycardia. One was V-A 1:1 relationship with long RP intervals, another was VA dissociation with V faster than A rate, the 3rd was VA dissociation with atrial fibrillation (Figure 1A). Thus atrial tachycardia and atrio-ventricular reentrant tachycardia (AVRT) could be excluded. HV intervals during sinus rhythm and tachycardia were equal (50ms) which excluded ventricular tachycardia. His bundle refractory period ventricular stimuli could reset the tachycardia with H-H interval reseted earlier than the V-V interval and terminate the tachycardia repeatedly which suggested

the existence of an AP. His bundle refractory period ventricular stimuli during tachycardia complicated with AF suggested that the AP was not connected to the atrium, but to the AV node (Figure 1B). Multiple QRS fusion morphologies during His bundle refractory period ventricular stimuli on a fixed RVA site could be observed, suggesting the AP was a nodo-ventricular (NV) fiber which provides a broader period for His bundle refractory period ventricular stimuli to enter the circuit more easily (Figure 1B) [9]. S1S1 pacing of RVA led to dissociation of H and V, which excluded the ventricle from the reentrant circuit and further suggested that it were an AVNRT (Figure 1C). Both evidences confirmed that it was an AVNRT with a concealed NV fiber as a bystander. Sketch maps of the tachycardia are shown in our previous work (Figure 1D) [2].

After thermo-controlled ablation (55°C, 40 watt, 90 seconds) of the slow pathway of AV node at the lower 1/3 of the triangle of Koch, the tachycardia could not be induced anymore.

Patient 2

This patient was a 40 years old woman. She suffered from palpitation for 2 years. She was otherwise healthy. Atrial S1S2S3 decremental stimulation discovered "jump-up" phenomenon of AH interval by 100ms (at S1S2S3=400/280/250-240ms) which demonstrated the existence of dual atrioventricular (AV) nodal pathways. Burst stimulation of the RVA induced a narrow QRS tachycardia repeatedly with CL variations from 325 to 368 ms. The tachycardia showed VA dissociation which excluded atrial tachycardia and AVRT. H and V were in 1:1 relationship and H-V interval during tachycardia equaled to that during sinus rhythm (48 ms), suggesting it were a supra-ventricular tachycardia (Figure 2A). His bundle refractory period ventricular stimuli could not only reset H-H interval and then V-V interval, but also terminate the tachycardia. All of the above phenomena suggested that the patient had an AP which did not connect the atrium. ATP (20 mg bolus) injection terminated the tachycardia, further suggested that the AV node were probably within the reentrant circuit (Figure 2C). RVA entrainment at 300 ms generated a more fully captured QRS morphology than at 310 ms (Figure 2B). This phenomenon suggested that the AP were an NV fiber, but not an NF fiber. Thus this tachycardia was an orthodromic nodo-ventricular reentrant tachycardia (NVRT). Sketch maps of the tachycardia are shown in Figure 2E.

After thermo-controlled ablation (55°C, 35 watt, 120 seconds) of the slow pathway of AV node at the lower 1/3 of the triangle of Koch (Figure 2D), the tachycardia was not induced, and repeated EPS did not cause "jump-up" phenomenon of A-H interval anymore.

Patient 3

This patient was a 50 year old man. He suffered from palpitation for 12 years. He was otherwise healthy. Either atrial or ventricular EPS could induce the tachycardia with CL variation from 260 to 275 ms. This tachycardia had three patterns including a narrow QRS pattern, a complete left bundle branch block pattern (CLBBB) with superior axis and a CRBBB pattern with superior axis (Figure 3A). All 3 patterns could switch to each other incessantly. Cycle length of the narrow QRS pattern equaled to that of the CLBBB pattern (265 ms), while CL of the CRBBB pattern was longer (275ms). V-A dissociation was observed during all 3 patterns of tachycardia which excluded atrial tachycardia and AVRT. H-V intervals of all 3 patterns of tachycardia and sinus rhythm were equal (46 ms, Figure 3B). Activation sequence of His on both sides during tachycardia was from the proximal to the distal which suggested that the tachycardia was a supra-ventricular one. His bundle refractory period ventricular stimuli could reset H-H interval, indicating the existence of an AP that did not connect to the atrium (Figure 3C). RVA pacing during sinus rhythm produced V to H sequential conduction without V to A conduction, and the proximal H was earlier than the distal H (Figure 3C). This phenomenon further suggested that there was an AP which was not connected to the atrium. Since CL of the tachycardia was longer during CRBBB than during CLBBB (Figure 3A), it could be deduced that the AP located on the right side [4]. RVA entrainment at 240 ms generated a more fully captured QRS morphologies than that at 250 ms (Figure 3D). This phenomenon suggested that the AP was an NV fiber, but not an nodo-fascicular (NF) fiber. This tachycardia was an orthodromic reentrant tachycardia mediated by a concealed NV fiber which located on the right side. Sketch maps of the tachycardia are shown in Figure

3E.

After thermo-controlled ablation (55°C, 40 watt, 100 seconds) of the slow pathway of AV node at the lower 1/3 of the triangle of Koch, the tachycardia could not be induced, and repeated EPS did not cause "jump-up" phenomenon of A-H interval any more.

All three patients survived up till now without any symptoms of recurrences.

Discussion

It was in 1941 that Mahaim and Winston described the histology of anomalous connections that arise from the AV node and insert into the right ventricle (RV) [5]. This was the first description of NV or so-called Mahaim's APs. Mahaim's bundle with decremental conduction properties that connected the atrium to the right bundle branch were subsequently mapped mainly at the lateral wall of the tricuspid annulus, thus the term atriofascicular bundle appeared[6-7]. Decrementally conducting connections can exist between the right atrium or the AV node and the RV in or close to the right bundle branch [6-8]. There are five types of Mahaim's bundle as presently defined, including decremental antegradely conducting atrio-ventricular fiber, atrio-fascicular fiber, NV fiber, NF fiber and fasciculo-ventricular fiber[9,10]. Since the last one does not cause reentry, it rarely needs ablation [10].

The distal part of Mahaim's bundle usually connects to the RV in the majority cases [11]. Thus QRS morphologies related to Maihaim's bundle are usually left bundle branch block pattern. But there are some exceptions. Nodoventricular fiber is a rare type of AP that connects the AV node and the right ventricular myocardium. Since atrium is out of the reentrant circuit of NVRT, NVRT can show VA dissociation which will not appear in other AP-mediated reentrant tachycardias except for nodo-fascicular reentrant tachycardia (NFRT). A tachycardia with VA dissociation could be misdiagnosed as a ventricular tachycardia (VT), especially when it shows a broad QRS pattern. Methods to differentiate between VT and SVT include comparing H-V intervals during tachycardia and sinus rhythm and recording conduction sequence of H potential during tachycardia. For orthodromic SVT, H-V interval during tachycardia should equal to that during sinus rhythm. Conduction sequence of H potential during orthodromic SVT should be from the proximal to the distal part. Otherwise, the tachycardia should be diagnosed as VT.

By excluding VT, a tachycardia with V-A dissociation should mainly focus on differentiation among AVNRT, NVRT and NFRT. Since all these tachycardias depend on the existence of dual AV nodal pathways, an A-H interval "jump-up" phenomenon is not enough for definite diagnosis. Further more, once the patient had an NV/NF fiber, he/she actually had 3 pathways that concern the AV node. Permutation of 3 pathways obtains 2 possible patterns of tachycardia, one is AVNRT complicated with a bystander NV/NF fiber as Patient 1, the other is NV/NF-RT as Patient 2 & 3. So, one should not rush to diagnosis of AVNRT with VA dissociation after VT is excluded. Further differential maneuvers are necessary. His bundle refractory period ventricular stimuli resetting the tachycardia points to the existence of a retrogradely conducting AP. For concealed NV fibers, His bundle refractory period ventricular stimuli reset the H-H interval earlier than the V-V interval because the conduction sequence of an orthodromic SVT is from His bundle to the ventricle. But para-Hisian pacing at different current intensity during sinus rhythm which is used to differentiate between ordinary septal AP and retrograde V-A conduction via AV node could be less helpful for differentiation between an NV/NF fiber and retrograde V-A conduction via AV node, because the retrograde VA conduction in NV/NF fiber could not bypass AV node and form a much shortened VA interval as ordinary AP.

As we know, CL of orthodromic AVRT can prolong if accompanied with ipsilateral bundle branch block, because it takes more time to go along the contralateral bundle branch and then go through the interventricular septum than just go along the ipsilateral bundle branch[12]. In Patient 3, we noticed that CLs were equal during normal QRS pattern and CLBBB pattern, while it became longer during CRBBB pattern. This suggested that the NV fiber located on the right side. We wonder that might all NV fibers locate on the right side because the AV node is mainly a right heart structure.

Since all the above phenomena were shared by concealed NV fiber and concealed NF fiber, we need further

differentiations between them. It has been reported that the presence of QRS fusion (defined as any QRS complex morphology other than that of a fully paced morphology) during entrainment of SVT by ventricular pacing proves that the ventricular myocardium is participating in the circuit [13]. That means if there were QRS fusion, it should be an NV fiber, otherwise it should be an NF fiber. Our understanding of this point of view is as the following: An NFRT is a closed loop reentry within the conduction system, while an NVRT is an open loop reentry that concern the ventricular myocardium. To entrain an NFRT, RV stimulation has to enter the distal part of right bundle branch retrogradely before supraventricular wavefront activates it antegradely and then goes into ventricular myocardium to form fused QRS, because antegrade conduction within the right bundle branch would cause refractory period itself that hinders entrainment capture. So, NFRT entrainment causes a fully paced morphology at the pacing site. For NVRT, RV stimuli can drive into the tachycardia via RV myocardium at any time, forming progressive QRS fusion. The shorter the entrainment CL, the closer the morphology to the fully paced one, as was seen in Patient 2 and 3. In our opinion, His bundle refractory period ventricular stimulus is like a one-beat entrainment of SVT. Thus, if an SVT could be reset by His bundle refractory period ventricular stimuli with only one QRS morphology at a fixed position which should be the full paced one, ventricular myocardium is ruled out from the reentrant circuit, and vise versa. That's why we deduced Patient 1 also had an NV fiber, but not an NF fiber.

Conclusions

Concealed NV fibers can mediate orthodromic SVT or be a bystander of AVNRT. V-A dissociation usually occur during such SVTs. An NV fiber not only expresses the characteristics of an AP, but also the characteristics of the AV node. Multiple QRS fusion morphologies during ventricular entrainments or His bundle refractory period ventricular stimuli at a fixed position can discriminate NV fibers from NF fibers.

Limitations

The main limitation of this study is that we did not meet with any cases of manifest NV fibers. Thus we cannot show full electrophysiological manifestations of all the NV fibers. Whether NV fibers could be manifest needs further demonstration in future.

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Conflicts of interests

None to declare.

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Figure legends

Figure 1 Phenomena observed during diagnostic process of patient 1

1A: 4 rhythms and the corresponding A-H-V relationships. 1B: His bundle refractory period ventricular stimuli during tachycardia either reset or terminated tachycardia. 1C: RVA pacing dissociated V and H and fluoroscopy showed the successful ablation target at the triangle of Koch. 1D: Sketch map of the mechanism in different situations. RVA=right ventricular apex.

Figure 2 Phenomena observed during diagnostic process of patient 2

2A: His bundle refractory period ventricular stimuli during tachycardia either reset or terminated tachycardia. 2B: RVA entrainment at different cycle lengths generated different fusion QRS morphologies, indicating ventricular myocardium took part in the reentry. 2C: EGM at successful ablation target and fluoroscopy showed the successful ablation target at the triangle of Koch. 2D: Sketch map of the mechanism in different situations. EGM=electrogram

Figure 3 Phenomena observed during diagnostic process of patient 3

3A: Tachycardia switched between LBBB, RBBB and normal QRS morphology, accompanied by cycle length change. 3B: Antegrade conduction of His bundle during tachycardia. 3C: A His bundle refractory period ventricular stimulus reset the tachycardia and antegrade conduction of His bundle during ventricular pacing revealed the existence of accessory pathway. 3D: Progressive QRS fusion and perfect PPI during RVA entrainment. LBBB=left bundle branch block; RBBB=right bundle branch block; PPI=post pacing interval.