Electrophysiological Characteristics and Radiofrequency Ablation of Focal Atrial Tachycardia Originating From the Superior Vena Cava

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The initiation of focal atrial tachycardia (AT) from the superior vena cava (SVC) remains unclear. In 3 patients (2 females, 1 male; aged 57, 66 and 50 years, respectively) with focal AT arising from different parts of the SVC, the AT occurred spontaneously, rather than being induced by electrical stimulation. The cycle length of the tachycardia was highly variable, ranging between 190 and 300 ms in patient 1, 180 and 320 ms in patient 2, and 200 and 300 ms in patient 3. The clinical or associated arrhythmias were atrial fibrillation (AF) (patients 1, 3) and atrial flutter (AFL) (patients 2, 3). A presumed SVC potential that was earlier than the activation of all the other mapping sites was recorded during AT at the lower anterior (15-mm above the atrio caval junction), the mid-anterior (25-mm above the atrio caval junction) and the lower posterior aspect of the SVC (17-mm above the atrio caval junction). Radiofrequency (RF) ablation targeting the SVC focus with the SVC potential promptly eliminated the focal AT in all 3 patients. The coexistent typical AFL was ablated, but the AF was not. The follow-up period was 6, 6, and 3 months, respectively, for each of the patients under no antiarrhythmic medication; there has not been a recurrence of symptomatic palpitation. In conclusion, focal electrical firing in the SVC can initiate AT and this type of focal AT is always associated with AFL or AF. RF ablation guided by the presumed SVC potential is safe and highly effective in eliminating the tachycardia. (Jpn Circ J 2001; 65: 1034–1040)

Key Words: Focal atrial tachycardia; Radiofrequency ablation; Superior vena cava

Methods

Patients

The study group were 3 patients with drug-refractory atrial tachyarrhythmias who were admitted for electrophysiological study and RF ablation therapy. Each patient had a focal AT originating from the SVC that had been diagnosed and confirmed by the electrophysiological study, SVC angiography and RF ablation. Two (cases 1 and 2) of the 3 patients did not have significant organic heart diseases detectable by physical examination, chest roentgenograms, echocardiography, and coronary angiography. The other patient (case 3) had one-vessel coronary artery disease. The definition of focal AT was based on previously established criteria.7–12

Electrophysiological Study and RF Ablation

The electrophysiological study was performed in a postabsorptive state after each patient gave written informed consent. All antiarrhythmic drugs except amiodarone were discontinued for at least 5 half-lives before the study. Two 6F quadripolar electrode catheters with a 5-mm interelectrode spacing were positioned at the high right atrium and the right ventricular apex, respectively, for pacing and recording. Another 6F quadripolar electrode catheter with a 10-mm interelectrode spacing was positioned across the tricuspid annulus to record the His bundle potential. A 6F decapolar electrode catheter with a 2-10-2-mm interelectrode spacing (Daig Corp) was positioned in the coronary sinus for recording and pacing. A 7F deflectable duodecapolar halo catheter (Cordis-Webster Co) was positioned along the tricuspid annulus for recording. A 7F...
deflectable catheter with a 4-mm tip and a 2.5-2-mm inter-electrode spacing (Mansfield Division, Boston Scientific Corp or Cordis-Webster Co) was used as a roving catheter for pacing, mapping and ablation. Surface ECG leads I, aVF and V1, as well as intracardiac electrograms, were simultaneously displayed on a multi-channel oscilloscopic recorder (Prucka Engineering Inc) and stored on a magnetic optical disc. The pacing stimuli were provided by a digital programmable stimulator (Bloom and Associates, DTU-215) at twice the diastolic threshold and 2 ms in duration. In all 3 patients, atrial stimulation was not performed for specifying the mechanism of the tachycardia during AT because of the risk of inducing AF and difficulty in interpreting the stimulation results. RF ablation was performed by a generator (Osyka 300 Smart) that delivered a continuous unmodulated current at 500 kHz with temperature feedback. The target temperature during each RF current application was set at 60°C and the duration was 30 s per pulse. If the patient developed chest pain, cough or severe bradycardia during ablation, the RF current application was terminated immediately and the target temperature was decreased to 50–55°C. At the presumed successful ablation site, further RF pulses lasting for a total of 60–120 s were needed to ensure complete elimination of the arrhythmic focus. Post-ablation clinical follow-up at intervals of 1 week, 2 weeks, 4 weeks and then 3 months consisted of history taking, physical examination, a 12-lead ECG at each visit and a 24-h Holter ECG at 2 weeks. When there were symptoms suggestive of arrhythmia, another Holter recording or a follow-up electrophysiological study was performed. Telephone interviews were carried out for those patients who were lost to regular clinical follow-up.

Results

Patient 1

This patient, a 57-year old female, had experienced episodic palpitations for 10 years. A 12-lead ECG during palpitation showed an irregular narrow QRS complex tachycardia with an average ventricular rate of 166 beats/min, consistent with AF. A direct current shock of 50 J was required to restore sinus rhythm because of the drug-refractory tachycardia. The resting ECG displayed left axis deviation and non-specific ST–T changes. During the baseline electrophysiological study, spontaneous initiation of sustained AF occurred before starting the programmed electrical stimulation. An attempt to restore sinus rhythm by intravenous administration of 300 mg of amiodarone...
was ineffective, so an external direct current shock of 200 J
was applied and the AF was terminated. However, after 2
sinus beats, a persistent AT was initiated by an ectopic beat
originating from the high right atrial region. The cycle
length of the tachycardia varied from approximately 190 to
300 ms and during tachycardia, the P wave deflection was
upright over leads II, III, aVF, downward over lead aVR, and
biphasic over lead V1, a P wave morphology that was very
similar to that of the sinus beat (Fig 1). At the anterior
aspect of the SVC approximately 15 mm above the atrio-
caval junction, as evaluated from a left lateral SVC
angiogram, the roving catheter recorded a low-amplitude
(0.2–0.4 mV) spiky potential, which was followed by an
atrial electrogram during AT. Spontaneous Wenckebach-
type exit block was observed between the spiky potential
and the atrial electrogram (Fig 2). The low-amplitude spiky
potential preceded the onset of the surface P wave by 40
ms, which was measured from a beat with the shortest
conduction time from the spike to the atrial electrogram.
Delivery of RF current at the SVC focus with the spiky
potential terminated the AT in 4.6 s (Fig 3). After ablation,
neither AT nor AF occurred spontaneously. During isopro-
ter enol infusion, programmed stimulation could not induce
either atrial tachyarrhythmias and the patient has been free
of symptomatic palpitations and syncope for 6 months
under no antiarrhythmic medications.

Patient 2

Patient 2, a 66-year-old woman, had suffered from
paroxysmal palpitation for 2 years. A 12-lead ECG during
palpitation displayed atrial flutter (AFL) with 2:1 atrioven-
tricular (AV) conduction and a ventricular rate of 150
beats/min. The resting ECG was normal. During the base-
line electrophysiological study, rapid atrial pacing from the
right atrium at a cycle length of 250 ms induced the
clinical AFL (flutter cycle length = 250 ms) with 2:1 AV
conduction. Pacing from the coronary sinus ostium at cycle
lengths shorter than the flutter cycle length revealed
concealed entrainment. The post-pacing interval measured from the coronary sinus ostium was approximately that of the flutter cycle length. Linear RF ablation targeting the septal isthmus terminated the AFL. However, spontaneous repetitive bursts of AT with a varying tachycardia cycle lengths ranging between 180 and 320 ms ensued. During tachycardia, the P wave was upright over leads II, III, aVF, downward over lead aVR, and biphasic over lead V1, a P wave morphology resembling that of the sinus beat (Fig 1). Intracardiac recordings disclosed local atrial activation at the high right atrium that was earlier than that of all the other recording sites. Detail mapping of the high right atrial region was accomplished by using the roving catheter and at the anterior aspect of the SVC, it recorded a high-amplitude (0.8–1.6 mV) spiky potential, which appeared immediately after an atrial electrogram during sinus beats. However, during bursts of AT, the activation of the spiky potential preceded that of the atrial electrogram (Fig 4). At a more upward location of the anterior SVC, approximately 25 mm above the atrio caval junction, as evaluated from a left lateral SVC angiogram, the roving catheter recorded the alternate occurrence of a low-amplitude (0.2–0.4 mV) and a high-amplitude (0.6–1.2 mV) spiky potentials. The low-amplitude spiky potential, which was very similar in morphology to the spiky potential recorded in case 1, displayed exit block to the atrial activation. The activation of the high-amplitude spiky potential preceded the onset of the surface P wave by 75 ms. Delivery of RF current at this SVC focus terminated the tachycardia immediately. Double asterisk, sinus beat after ablation of AT. Abbreviations as in Fig 2.
**Patient 3**

The third patient, a 50-year-old male, presented with recurrent palpitations for 6 months after a previous ablation procedure at another institution 18 months ago. The 12-lead ECGs during palpitation episodes recorded 2 types of atrial tachyarrhythmia: one displaying an irregular narrow QRS complex tachycardia with an average ventricular rate of 190 beats/min consistent with AF, the other showing a repetitive form of non-sustained AT. The resting ECG was normal. During isoproterenol infusion at a rate of 1 μg/min, rapid atrial pacing from the high right atrium at a cycle length of 170 ms reproducibly induced AF. The AF subsequently changed to AFL (flap cycle length = 200 ms) with 2:1 AV conduction. Pacing from the coronary sinus ostium at cycle lengths of 180 and 170 ms demonstrated concealed entrainment. The post-pacing interval measured from the coronary sinus ostium was approximately that of the flutter cycle length. However, this subeustachian isthmus-dependent typical AFL subsequently degenerated into AF and then terminated spontaneously. Following termination, an incessant, repetitive form of AT with almost beat-to-beat variations in tachycardia cycle length (ranging between 200 and 300 ms) occurred spontaneously. During tachycardia, the P wave was upright over leads II, III and aVF, downward over lead aVR, and biphasic over lead V1, a P wave morphology that very similar to that of the sinus beats (Fig 1). We decided to ablate the AT first. As the local atrial activation at the high right atrium was earlier than that of all the other recording sites, we used the roving catheter to perform detail mapping of the high right atrial region. At the posterior aspect of the SVC, approximately 17 mm above the atroio caval junction, as evaluated from an anterior oblique projection of a left lateral SVC angiogram, the roving catheter recorded a high-amplitude (2.4–3.2 mV) spiky potential during AT. This potential, followed by a relatively low-amplitude atrial electrogram, preceded the onset of the surface P wave by 61 ms during tachycardia. The AT terminated suddenly during mapping, when the roving catheter was positioned at the SVC focus with the spiky potential. During sinus rhythm, the spiky potential was activated after the low-amplitude atrial electrogram, which was in contrast to the recordings that occurred during AT. Applying RF current to this SVC focus resulted in complete elimination of the AT. Following ablation of the AT, linear ablation of the subeustachian isthmus to eradicate the typical AFL was then performed during sinus rhythm. Post-ablation rapid atrial pacing at the same cycle length of 170 ms could not induce AF with or without isoproterenol provocation. The patient has been free of symptomatic palpitation for 3 months without antiarrhythmic medications.

**Discussion**

The principle finding of the current study is that a distinct type of focal AT can originate from various parts of the SVC. The clinical and electrophysiological characteristics of this focal AT include (1) P wave morphology during AT that is very similar to that of the sinus beat; (2) tachycardia that is initiated spontaneously; (3) an association with AFL or AF; (4) a presumed SVC potential with highly variable cycle lengths that can be recorded during AT; (5) the SVC potential possibly displaying 1:1 conduction or second-degree exit block to the atrial electrograms during AT; and (6) safe and highly effective elimination of the focal AT by application of RF current to the SVC focus with the SVC potential.

Markowitz et al defined focal AT by its electrophysiological characteristics of a centrifugal atrial activation pattern, almost dissociation of both atria from tachycardia with atrial extrastimuli, an identifiable early local atrial activation relative to the surface P wave, and inability to demonstrate manifest entrainment. In the present study, delivery of atrial extrastimuli and using overdrive pacing to specify the tachycardia mechanism was not performed in the 3 patients because of the risk of inducing AF and difficulty in interpreting the stimulation results because of marked beat to beat variations in the tachycardia cycle length. However, the AT in each patient all displayed an eccentric atrial activation sequence and had a presumed SVC potential preceding the onset of the surface P wave during tachycardia. RF ablation targeting the SVC focus with the SVC potential promptly abolished the AT. These findings suggest a focal origin of the AT.

Focal AT has been reported to arise from diverse anatomical structures in the right or the left atrium. More recently, Ino et al described a case with a 2:1 exit block of repetitive focal activity in a SVC focus manifesting as a high right AT. Furthermore, we found that AT can occur focally from different parts of the SVC, which was confirmed by the SVC angiogram showing the location of...
the successful ablation sites at various distances above the atrio caval junction and different positions within the SVC (Fig 7). These SVC foci were all located within 30 mm above from the atrio caval junction, which is in accordance with the histological and electrical connections of the musculature between the SVC and the right atrium.1-3

Although it remains unclear why the atrial muscle extending into the SVC becomes arrhythmogenic, Ito et al have shown phase 4 depolarization-initiated automatic activity14 and Yanaga demonstrated aconitine-induced abnormal automaticity and fibrillation in the musculature of the SVC.15 It is likely that if the musculature in SVC contains pacemaker activity derived from its early embryological development, then under certain circumstances, it could become arrhythmogenic. We propose abnormal automaticity or triggered activity as the possible underlying mechanism of the tachycardia in the 3 patients because the tachycardia occurred spontaneously rather than being induced by programmed electrical stimulation.

The difference in the P wave configuration in the 12-lead ECG during AT, when compared with one during sinus rhythm, is an important criterion to establish the diagnosis of AT. The P wave polarity during AT in the 3 patients was identical to that of the sinus beat, the only difference being a slightly larger positive deflection of the P wave over the inferior leads during AT in case 3. This indicates that the spread of intraatrial activation might be similar between the SVC-initiated focal AT and the sinus rhythm. Furthermore, the P wave configurations observed in the current study fulfilled the classification of cristal AT as suggested by Tada et al, therefore, one should be aware that focal AT originating from the SVC could be erroneously classified as a cristal AT if simply based on the ECG criteria for defining the site of origin of AT.

Recently, Tsai et al demonstrated and characterized a spiky SVC potential in focal AF, which appeared as a large spiky deflection and preceded a low-amplitude far-field atrial electrogram during SVC ectopy or SVC ectopy-initiated AF.19 In contrast to the recordings during AF, the SVC potential was activated after the far-field atrial electrogram during sinus rhythm. Ablation of the SVC ectopy was highly effective in terminating and/or preventing re-induction of AF. However, in the current study, we recorded a low-amplitude spiky SVC potential preceding an atrial electrogram during AT in case 1. The spiky potential exhibited an intermittent second-degree Wenckebach block to the atrial electrogram and there was an inverse relationship between the conduction time from the spiky potential to the atrial electrogram and the spike’s cycle length. Both the spiky potential and the AT were eliminated within a few seconds of RF current application. These findings indicate that the AT was initiated and maintained by the focal SVC firing, which had an AV node-like decremental conduction property to the atria. From cases 2 and 3, we recorded a high-amplitude spiky SVC potential preceding an atrial electrogram during AT, as opposed to the recording that occurred during sinus beat. The reversed activation sequence between the atrial electrogram and the spike during AT was similar to that described by Tsai et al and Haissaguerre et al in focal AF. In case 3, the high-amplitude spike and the atrial electrogram exhibited a 1:1 relationship, whereas, at the successful ablation site of case 2, the recordings showed an alternate occurrence of a high-amplitude and a low-amplitude spiky potential. The low-amplitude spiky potential, which was very similar in morphology to the spiky potential recorded in case 1, displayed exit block to the atrial activation. Application of RF current at the SVC foci with the spiky potentials promptly eliminated both the spikes and the AT in cases 2 and 3, indicating that the SVC foci also played a critical role in generating and maintaining the tachycardia in these patients.

Finally, an association with other paroxysmal atrial tachyarrhythmias was present in all 3 patients; that is, AF in cases 1 and 3, and typical AFL in cases 2 and 3. The coexistent AFL was ablated, but the AF was not. However, during the follow-up period, we found that none of these atrial tachyarrhythmias recurred.

Conclusions

Focal electrical firing in the SVC can initiate AT in addition to AF. This type of focal AT is always associated with paroxysmal AFL or AF. RF ablation guided by the presumed SVC potential is safe and highly effective in eliminating the tachycardia.

References


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