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Coexisting Sustained Tachyarrthymia in Patients With Atrial Fibrillation Undergoing Catheter Ablation

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ABSTRACT

Background and Objectives: During the index procedure of catheter ablation (CA) for atrial fibrillation (AF), it is important to assess whether other atrial or ventricular tachyarrhythmia coexist. Their symptoms are often attributed to residual tachycardia after successful elimination of AF by CA. This tachycardia could also be non-pulmonary vein (PV) foci initiated AF. This study examined the coexistence of other sustained tachyarrhythmia of patients who underwent radiofrequency CA (RFCA) for AF. **Subjects and Methods**: Four hundred fifty-nine consecutive patients (375 males, aged 53.4±11.4 years) who underwent RFCA for AF were investigated. Atrial and ventricular programmed stimulation (PS) with or without isoproterenol infusion were performed, and spontaneously developed tachycardias were analyzed. **Results**: Fifteen patients (3.3% of total) were diagnosed to have other sustained arrhythmias that included slow-fast type atrioventricular nodal reentrant tachycardia (AVNRT, n=6), atrioventricular reentrant tachycardia (AVRT, n=5) that utilized left posteroseptal (n=4) and parahisian bypass tract (n=1), atrial tachycardia (AT, n=2) originating from the foramen ovale (n=1) and the ostium of coronary sinus (n=1), sustained ventricular tachycardia (VT, n=2) involving one from the apical posterolateral wall of left ventricule in a normal heart and one from an anterolateral wall in an underlying myocardial infarction (MI). These sustained tachycardias were neither clinically documented nor had structural heart diseases, with the exception of one patient with MI associated VT. Two patients had the triple tachycardia; one involved AVNRT, AVRT, and AF, and the other involved VT, AT, and AF. All associated tachycardias were successfully eliminated by RFCA. **Conclusion**: Fifteen (3.3%) patients with AF had coexisting sustained tachycardia. RFCA was successful in these patients. Identification of tachycardia by PS before RFCA for AF should be done to maximize the efficacy of the first ablation session. **(Korean Circ J 2010;40:235-238)**

KEY WORDS: Atrial fibrillation; Tachycardia supraventricular; Catheter ablation.

Introduction

Patients with supraventricular tachycardia (SVT) occasionally experience atrial fibrillation (AF).[1-3] Some forms of AF could be caused by a rapid atrial tachycardia (AT) and radiofrequency catheter ablation (RFCA) of this focus is curative.[3)4)] AF can be associated with other forms of SVT.[5)6)] AF may initiate from atrial flutter,[4)] atrioventricular nodal reentrant tachycardia (AVNRT),[7)] or atrioventricular reentrant tachycardia (AVRT) due to an accessory pathway.[8)] Sometimes, palpitation or other symptoms in patients with AF may be attributable to these tachyarrhymias.

Therefore, these coexistent arrhythmias could cause residual symptoms after catheter ablation (CA) for AF. Identification of patients with coexistent tachyarrhythmia-induced AF could affect the ablation strategy and improve the efficacy of the CA procedure. The aim of this study was to investigate the prevalence of coexistence of other sustained tachyarrhythmia of patients who underwent RFCA for AF and the effect of their ablation on the procedural outcome.

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Subjects and Methods

Patient records

We retrospectively collected patient data from medical records. Medical records in our institution were searched from 1998-2007 and all patients who were referred for CA of AF were included in this study. Electrophysiology study and ablation reports, as well as complete medical records of patients with symptomatic, paroxysmal AF (PAF) that was defined as AF with self-termination within 7 days or persistent AF that was defined without spontaneous termination in 7 days and which required chemical or electrical cardioversion were reviewed. Holter recordings, events recordings, and resting electrocardiograms were reviewed. All patients were subjected to a full diagnostic electrophysiological assessment with rapid atrial pacing with isoproterenol as described below to reveal arrhythmogenic foci. Only patients referred for an initial AF ablation were included. The study was approved by our Institutional Review Boards and all patients had provided their written informed consent.

Electrophysiological study and radiofrequency catheter ablation

Electrophysiological studies were carried out according to standard protocol in patients sedated with midazolam and phentanyl sodium. Each patient was in the post-absorptive state of the antiarrhyhmic agent and the antiarrhythmic agent had been discontinued for the number of days corresponding to at least 4-5 times the agent's half-life. A duodecapolar catheter was positioned in the coronary sinus and the lateral wall of the right atrium (RA), a decapolar catheter in the RA, and a quadripolar catheter in the superior vena cava (SVC). Pacing with RA and right ventricular refractory periods at one or more cycle lengths were performed as well as decremental pacing. Arrhythmia induction was facilitated by isoproterenol infusion at rates up to 10 mg/min. Bipolar electrograms were filtered at 30-500 Hz and displayed and acquired, together with surface electrocardiograms.

Ablation of accessory pathways and slow pathway for AVNRT was performed according to standard techniques. RFCA for AF was accomplished via an ostial or circumferential approach, and induction of sustained supraventricular arrhythmias other than AF was performed after completion of RFCA for AF.

Follow up

Patients were followed-up by clinical assessments and ambulatory electrocardiographic monitoring. Initial post-ablation follow-up took place at 1 week and 1 month, and every 3-6 months thereafter. Beyond this interval, 6-month outpatient clinic visits with Holter monitoring were scheduled. All patients were instructed to maintain personal records with descriptions of every episode of symptomatic palpitations. A successful outcome over the follow-up period was defined as the lack of electrocardiographically recorded SVT and AF, and no other sustained arrhythmias.

Results

In total, 459 patients (374 males, aged 53.4±11.4 years) were included in the study. Analysis of medical records, ablation reports, and electrophysiology revealed 15 (3.3%, aged 45.4±14.1 years) patients had accompanying sustained arrhythmias, which comprised AVNRT (n=6), AVRT (n=5), AT (n=2), and ventricular tachycardia (VT) (n=2). All patients with AVNRT were slow-fast type; cases were revealed by programmed stimulation (PS) (n=4) and during catheter manipulation (n=1) before RFCA for AF, and one AVNRT was clinically documented 2 months after AF ablation. All five patients with AVRT were identified to have a concealed accessory pathway, utilizing the left posteroseptal (n=4) and parahisian bypass tracts (n=1). Two ATs originated from the foramen ovale (n= 1) and the ostium of CS (n=1), which were not related to initiation and maintenance of AF; these were documented by PS before RFCA for AF. Patients with the other tachyarrhythmias were younger (45.4±14.1 years) than those without them (53.4±11.4 years, p=0.01). Sustained VTs were identified in two patients, one from the apical posterolateral wall of left ventricule in a normal heart and one from anterolateral wall in an underlying myocardial infarction (MI). Fourteen of the 15 patients with tachycardia were not clinically documented. None had a structural heart disease, except one patient with VT in MI. Two patients had triple tachycardia; one involved AVNRT, AVRT, and AF, and the other involved AF, AT, and idiopathic VT. All associated tachycardias were successfully eliminated by RFCA. All 13 patients with SVT were free from AF during follow-up (27.9±19.6 months) without use of an anti-arrhythmic agent except one patient. One other patient with persistent AF underwent the second RFCA for recurrent AF.

Discussion

In this study, we report a 3.3% incidence of other sustained tachyarrhythmias in patients who presented exclusively with drug refractory AF at the time of CA. Although this represents a small proportion of the total population of patients who present with AF, it is not insignificant. It has been recently described that some forms of AF might be due to the presence of a rapid AT and that radiofrequency ablation of the foci of AT resulted in cure of the AF.[4] It is also well-known that patients with an atrioventricular (AV) accessory pathway have a higher incidence of AF than the general populat-

ion, and that ablation of the accessory pathway is associated with a decrease in the subsequent incidence of AF.[9]

Previous reports have described AVNRT-initiated AF.[7)10)11)] AVNRT as an isolated trigger of AF was first described by Brugada et al.[10)] Sauer et al.[7)] reported a 4.3% incidence of inducible AVNRT among patients referred for AF ablation. In their patients, slow pathway ablation abolished AF recurrences. It seems that in a considerable proportion of such patients, particularly in the young, elimination of AVNRT results in abolition of the AF episodes as well. Delise et al.[12)] reported an AF cure rate of 70% of patients presenting with both AVNRT and AF with slow-pathway modification alone. In addition, AF triggered by pulmonary vein (PV) ectopy during AVNRT has also been described and likely explains why some patients with AVNRT present with recurrent AF after successful slow-pathway modification.[6)] In contrast to earlier studies, we did not find any patient with AVNRT presenting as non-PV foci initiated AF. In one study, underlying arrhythmia was revealed in 7.6% of patients referred for AF ablation using electrophysiology, with study ablation proving successful results in abolishing PAF. Atrial flutter appeared to be the most common arrhythmia in this respect, followed by AVNRT, AVRT, and AT.[13)] Approximately 30% of patients undergoing successful ablation of atrial flutter may develop AF at follow-up,[14)] whereas a similar proportion of patients subjected to PV ablation for AF may experience isthmusdependent atrial flutter following the procedure.[15)] Typical atrial flutter and AF appear to represent separate clinical entities, with recent electrophysiologic data indicating important interrelationships between these arrhythmias and PAF in 12-46% of patients with unifocal or multifocal ectopic ATs.[16)] But, we did not include atrial flutter following PV isolation for AF, and atrial flutter did not develop while taking antiarrhythmic agents for AF. It may be that atrial flutter is not independent of AF, but rather lies at the same root. Furthermore, a detailed electrophysiology study may reveal the interactive mechanism of AF and other SVT or VT. However, we were unable to detect any SVT or VT spontaneously converting to AF, or visa versa.

AVRT can convert to AF and ablation of an accessory pathway may abolish AF recurrences, particularly in the young. In the series of Dagres et al.,[17)] 12% of patients with AF in the context of an accessory pathway and <50-years-of-age experienced AF recurrence following ablation of the pathway, as opposed to 55% of those >60-years-of-age. Ablation therapy of the underlying arrhythmia may not necessarily eliminate future AF recurrences.[8)18)]

Radiofrequency ablation either applied focally or circumferentially to isolate the PVs from the LA has been demonstrated to cure AF in a subgroup of patients.[19)20)] In one study, enlargement of the LA and septal origin of the atrial focus were independent predictors of coexisting AF and patients with a septal origin of the focal AT were older and had a higher rate of structural heart disease than patients with a non-septal foci.[21)] In the present study, AT originating from the foramen ovale did not initiate AF. Two patients underwent the CA for SVT after RFCA for AF. Their symptoms were attributed to residual tachycardias after successful elimination of AF by CA. One of these patients had AVRT with left posteroseptal bypass tract and the other had a slow-fast type AVNRT. In two patients with AF, sustained VT occurred. One VT developed in a patient who had previously experienced a MI. These VTs might have been missed if we did not perform PS including ventricular pacing. Although this finding is from just one patient, it is noteworthy in suggesting ventricular PS before CA has significant diagnostic value in patients with AF, especially in patients with structural heart diseases.

The success rate for curing AF along with slow-pathway modification is superior only to those with targeted AF ablation without inducible AVNRT.[7)] In our study, even we did not find the AVNRT initiated AF, successful elimination of AVNRT may have contributed to the elimination of potential triggers of AF.

Study limitations

The present study had three unavoidable limitations. It was retrospective. Secondly, patients with associated tachyarrhythmia did not have evidence of a transition from tachycardia to AF. Thirdly, the electrophysiological study did not follow the same protocol in all patients.

Conclusion

In our study, 3.3% of patients with AF had coexisting sustained tachycardia, for which CA was performed. Identification of these tachycardias by PS prior to CA for AF should be done to increase the efficacy of the first ablation session. Patient symptoms were mainly attributed to residual tachycardia after successful elimination of AF by RFCA.

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