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The Occurrence of New Arrhythmias after Catheter-Ablation of Accessory Pathway: Delayed Arrhythmic Side-Effect of Curative Radiofrequency Lesion?

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SUMMARY

Introduction New arrhythmias (NA) may appear late after accessory pathway (AP) ablation, but their relation to curative radiofrequency (RF) lesion is unknown.

Objective The aim of this study was to determine the prevalence and predictors for NA occurrence after AP ablation and to investigate pro-arrhythmic effect of RF.

Methods Total of 124 patients (88 males, mean age 43±14 years) with Wolff–Parkinson–White syndrome and single AP have been followed after successful RF ablation. Post-ablation finding of arrhythmia, not recorded before the procedure, was considered a NA. The origin of NA was assessed by analysis of P-wave and/or QRS-complex morphology, and, thereafter, it was compared with locations of previously ablated APs.

Results Over the follow-up of 4.3±3.9 years, NA was registered in 20 patients (16%). The prevalence of specific NAs was as follows: atrioventricular (AV) block 0.8%, atrial premature beats 1.6%, atrial fibrillation 5.4%, atrial flutter 0.8%, sinus tachycardia 4.8%, ventricular premature beats (VPBs) 7.3%. Multivariate Cox-regression analysis identified (1) pre-ablation history of pathway-mediated tachyarrhythmias >10 years (HR=3.54, p=0.016) and (2) septal AP location (HR=4.25, p=0.003), as the independent predictors for NA occurrence. In four NA cases (two cases of septal VPBs, one of typical AFL and one of AV-block) presumed NA origin was identified in the vicinity of previous ablation target.

Conclusion NAs were found in 16% of patients after AP elimination. In few of these cases, late on-site arrhythmic effect of initially curative RF lesion might be possible. While earlier intervention could prevent NA occurrence, closer follow-up is advised after ablation of septal AP.

Keywords: radiofrequency ablation; catheter-ablation; accessory pathway

INTRODUCTION

Radiofrequency (RF) catheter-ablation has become the therapy of choice for symptomatic patients with atrioventricular (AV) accessory pathway (AP) [1]. The ideal is to make the so-called point ablation, i.e. to destroy AP with a single or two RF current applications. However, sometimes it is necessary to apply several adjacent „burnings" to achieve a permanent pathway block.

Only few studies on very long-term outcome after AP ablation have been conducted. Although symptom recurrence [2] and extension of anti-arrhythmic drug (AAD) therapy [3] related to various new arrhythmias (NAs) after successful AP ablation have been reported, the true prevalence and distribution of NAs have not been completely elucidated.

Chronic RF lesion is permanent and unhomogeneous [4] and several studies raised concerns about delayed arrhythmic potential of RF lesion(s) after ablation of AV nodal reentrant tachycardia (AVNRT), ventricular tachycardia and atrial fibrillation (AF) [5, 6, 7]. Nevertheless, late proarrhythmic effects of RF after AP ablation have not been evaluated.

OBJECTIVE

The objectives were to determine: 1) prevalence of NAs after AP ablation; 2) potential clinical and procedural predictors for NA occurrence; and 3) to investigate possible arrhythmic side-effects of RF lesion as the source of NAs.

METHODS

Data on all consecutive patients with AP, undergoing RF catheter-ablation were examined at our institution between 1994 and 2008. The following inclusion criteria were defined: 1) ventricular preexcitation; 2) symptomatic pathway-mediated tachycardia/tachyarrhythmia before the procedure; 3) single AP on electrophysiological (EP) study; 4) successful primary outcome of catheter-ablation without AP conduction recurrence after the procedure; and 5) follow-up ≥1 year after ablation.

All patients had a physical examination and echocardiogram, while patients with history of chest pain underwent coronary angiography before ablation. Data on symptoms and arrhythmias prior to the ablation were obtained by anal-

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ysis of all available electrocardiograms (ECG), Holter-recordings and other medical documentation recorded before the procedure.

Ablation

All AADs were discontinued 2-3 days prior to the ablation. Left-sided pathways were accessed from the ventricular aspect, retrogradely from the femoral artery and aorta. Right-sided pathways were approached from the atrial side through the femoral vein.

Intracardiac mapping was conducted using a 4-mm tip ablation catheter (RF Marinr MC, Medtronic Inc., U.S.A.). A RF current generator (Atakr/Atakr II, Medtronic Inc., U.S.A.) with temperature-guided energy application was used. The preselected temperature was 50-60°C with power output limit 30-60 W. Preferably RF was applied during a sinus rhythm at the site with established intracardiac criteria [1]. Where the loss of preexcitation was not observed within 20 seconds, RF delivery was stopped; otherwise, RF application was continued up to 60-120 seconds. Programmed electrical stimulation from the right atrium and ventricle was performed 30 minutes after the ablation to confirm stable pathway block and to exclude other sustained arrhythmias.

The pathway location was determined based on the catheter-tip position at successful RF application, using suggested nomenclature [8]. For the purposes of the study, all ablation data from repeated procedures were added and analyzed as cumulative values per patient.

Table 1. Baseline characteristics of patients

Parameter	No new arrhythmias (n=104)	New arrhythmias (n=20)	p
Males	76 (73%)	12 (60%)	0.238
Age (years)	42±14	48±10	0.074
Age ≥35 years	65 (63%)	18 (90%)	0.019
Symptoms (years)	12±10	18±12	0.033
Symptoms >10 years	40 (38%)	15 (75%)	0.007
Palpitation attacks (per month)	8±16	5±8	0.770
Duration of palpitation attacks (minutes)	94±125	108±99	0.310
Ortodromic AVRT	84 (81%)	18 (90%)	0.523
Antidromic AVRT	11 (11%)	4 (205)	0.263
Atrial fibrillation	45 (43%)	5 (255)	0.127
Atrial flutter	5 (5%)	1 (5%)	>0.999
Ventricular fibrillation	4 (4%)	2 (105)	0.196
Ventricular premature beats	6 (6%)	0	0.588
Structural heart disease	15 (14%)	4 (205)	0.508
Co-morbidities	32 (31%)	8 (40%)	0.419
Number of drugs	1.9±1.2	2.4±1.4	0.077
Left free-wall AP	66 (63%)	8 (40%)	0.050
Septal AP	31 (30%)	12 (60%)	0.009
Right free-wall AP	7 (7%)	0	0.597

Values are expressed as X±SD or n (%).
AVRT – atrioventricular reentrant tachycardia; AP – accessory pathway

Follow-up

Initially, no AADs were prescribed after successful procedure. The follow-up began on the first day after final ablation and ended on the day of the last visit. The follow-up was based principally on periodic ECG recordings and symptoms; all patients were regularly seen in our outpatient clinic after 1, 3, 6 months and, thereafter, yearly after the procedure. The reappearance of delta-wave or reciprocating tachycardia during the follow-up was considered a recovery of AP conduction. In symptomatic patients, attempts were made at identifying causal relationship between complaints and underlying arrhythmia by repeated examinations, review of all recorded ECGs with ongoing symptoms and by Holter-recordings or hospital observation, if needed. When arrhythmia had been documented by 12-lead ECG, its origin was assessed by analysis of P-wave and/or QRS-complex morphology, using the ECG-guided algorithms [9-12]. Post-ablation finding of arrhythmia, already registered before the procedure, was considered an arrhythmia recurrence, while post-ablation finding of arrhythmia, not recorded before the procedure, was considered a NA.

Statistical analysis

Descriptive statistics are presented as mean ±SD for continuous variables and counts with percentages for categorical variables. In case of normally distributed variables, the unpaired t-test, otherwise, the Mann–Whitney U-test was used. Differences in categorical variables were evaluated by chi-square or Fisher's test, as appropriate. A two-tailed p-value <0.05 was considered statistically significant.

ROC curves were used to define the cut-off point in continuous variable before its dichotomization. Event-free survival probability was estimated using the Kaplan-Meier curves and differences between survival curves were assessed by log-rank test. Cox proportional hazards regression analysis was used to identify predictors of NA. In patients with >1 NA, the time period from ablation to the first recorded NA was considered. Those variables that showed a potentially predictive value in the univariate analyses ($p \leq 0.1$) were included in the multivariate forward model and independent predictors were defined ($p<0.05$). All statistical analyses were performed using SPSS/PC 10.0 software.

RESULTS

The study population consisted of 124 patients (88 men) who met the recruitment criteria. The mean age at ablation was 43±14 years, ranging from 16 to 77. The baseline patients' characteristics are presented in Table 1.

Before the ablation, 82% of the patients complained of orthodromic tachycardia, 12% of antidromic tachycardia, 40% of AF, 5% of atrial flutter (AFL), 5% of ventricular premature beats (VPBs) and 5% was successfully resuscitated of ventricular fibrillation. Associated structural heart

disease and co-morbidities were found in 15% and 32% of the patients, respectively.

The procedure was repeated in 20 patients (16%). There were no differences in cumulative ablation parameters in patients with and without NAs (Table 2).

Over the follow-up of 4.3±3.4 years (ranging from 1.0 to 12.7 years), six patients died; the mean time from procedure to death was 5.2±2.9 years (1.9–9.1 years). One male, known for coronary disease and new post-ablation AF, suddenly died at the age of 70 years. Other patients died of non-cardiac causes.

New arrhythmias after ablation

Overall, during the follow-up, arrhythmias were revealed in 30 of 124 patients (24.2%). The arrhythmia recurrence was registered in 11 patients (8.9%), including seven with AF, one with typical AFL and three with VPBs of the same QRS-morphology as before the ablation. However, 20 patients (16.1%) experienced the NAs following ablation; in three of these patients two different forms of NAs were recorded and, therefore, a total of 23 NA cases were identified. In a single patient both recurrent arrhythmia and NA were detected.

The mean time from procedure to NA appearance was 20±29 months (ranging from two days to eight years). In 16 of 20 patients (80%) NA was recorded within the first post-ablation year. Detailed characteristics of NA patients are presented in Table 3. On the symptomatic basis, in 90% of these patients AAD therapy was reinstituted or additional interventional treatment was undertaken.

In a 52-year-old male (0.8%), third degree AV-block coupled with syncope was registered 6 years after ablation of right midseptal AP (patient 2 in Table 3).

New ventricular arrhythmia was diagnosed in 9 patients (7.3%); only one of these patients was previously known for structural heart disease. Sustained ventricular tachyarrhythmias were not recorded after the procedure. However, non-sustained monomorphic ventricular tachycardia of slow rate (100 bpm) was noted in a single patient, while isolated VPBs were found in the remaining eight patients.

New atrial premature contractions (APC) were recorded in two patients (1.6%), free of structural heart disease. Eventually, in one of these patients, new APCs culminated in AF. In total, new AF was documented in four of 74 patients without AF at baseline (5.4%); the mean age at the new AF onset was 65±5 years and two of these were known for structural heart disease. In another, otherwise healthy patient (0.8%), new typical AFL evolved following ablation. In additional six patients (4.8%) daily activities were limited by inappropriate sinus tachycardia not registered before the procedure; previously, one of these patients was known for essential hypertension.

Table 2. Ablation parameters

Parameter	No new arrhythmias (n=104)	New arrhythmias (n=20)	p
Repeat procedure	17 (16%)	3 (15%)	>0.999
RF energy (W)	11711±11805	9678±9034	0.453
RF pulses	9.3±8.0	10.4±10.4	>0.999
Catheter-tip temperature (°C)	54.4±7.7	56.7±8.7	0.378
RF application time (s)	329±280	281±217	0.599
Fluoroscopy (min)	24.8±28.3	34.0±42.9	0.709

Values are expressed as X±SD or n (%).

Table 3. Characteristics of patients with new arrhythmias (NA)

No.	Age (years)	Gender	Heart disease and co-morbidities	AP location	NA	Origin of NA	Time of NA onset	Follow-up (years)	Treatment
1	52	M	-	RAS	APC	?	2 y	7.2	Ppf
2	50	M	HT	RMS	nsVT; AV-b	MA-AL; AVN	2 d; 6 y	7.1	Pace
3	36	M	-	RMS	VPB	RVOT	1 m	1.0	-
4	41	M	-	RPS	Typical AFL	CTI	1 m	1.3	Ami+BB
5	56	M	DCM	RMS	VPB	TA-MS	1 m	12.7	Ami
6	28	F	-	RPS	ST	SN	3 m	3.1	Ppf
7	54	F	HT	RPS	VPB	?	3 m	5.2	Ppf
8	32	F	HT	RPS	ST	SN	6 m	2.0	-
9	50	F	-	RPS	VPB	?	1 y	6.8	BB
10	61	M	CD, LD	RPS	AF	?	8 y	9.0	Ami+Ver
11	51	F	HT	LPS	VPB	RVOT	2 m	1.0	BB
12	48	M	-	LPS	VPB	MA-PS	1.5 y	1.8	Ver
13	57	M	-	LP	VPB	RVOT	1 y	10.0	Ami
14	57	F	HT, LD	LP	AF	?	6 y	7.3	Ami
15	42	M	CD	LPL	ST	SN	1 y	8.2	BB
16	52	F	HL	LPL	APC; AF	?; ?	4 m; 6.3 y	7.0	Ppf+BB
17	48	F	-	LL	ST	SN	6 m	11.8	BB
18	37	M	-	LL	ST; VPB	SN; ?	1 m; 1 y	10.7	BB
19	37	M	-	LL	ST	SN	1 m	9.8	Ver+BB
20	67	M	AS, HT	LL	AF	?	8 m	2.0	Sot

M – male; F – female; HT – hypertension; DCM – dilated cardiomyopathy; CD – coronary disease; LD – lung disease; HL – hyperlipidemia; AS – aortic stenosis; RAS – right anteroseptal; RMS – right midseptal; RPS – right posteroseptal; LPS – left posteroseptal; LP – left posterior; LPL – left posterolateral; LL – left lateral; APC – atrial premature contraction; nsVT – nonsustained ventricular tachycardia; AV-b – atrioventricular block; VPB – ventricular premature beat; AFL – atrial flutter; ST – sinus tachycardia; AF – atrial fibrillation; MA-AL – mitral annulus-anterolateral; AVN – atrioventricular node; RVOT – right ventricular outflow tract; CTI – cavotricuspid isthmus; TA-MS – tricuspid annulus-midseptal; SN – sinus node; MA-PS – mitral annulus-posteroseptal; y – years; d – day; m – month; Ppf – propafenone; Pace – pacemaker; Ami – amiodarone; BB – beta-blocker; Ver – verapamil; Sot – sotalol

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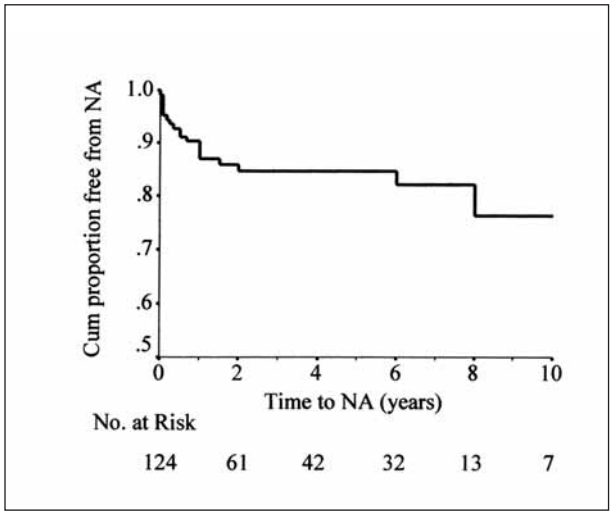
Predictors for new arrhythmia occurrence

Univariate analysis showed that age at ablation ≥35 years (p=0.038), pre-ablation history of tachyarrhythmias >10 years (p=0.021) and septal AP location (p=0.012) were significantly related to NA occurrence, while left free-wall pathway location (p=0.042) was a favourable marker. However, multivariate Cox regression analysis revealed only two independent predictors for NA occurrence: 1) septal AP location (HR=4.25; 95%CI: 1.66–10.88; p=0.003), and 2) pre-ablation duration of symptoms >10 years (HR=3.54; 95%CI: 1.27-9.88; p=0.016).

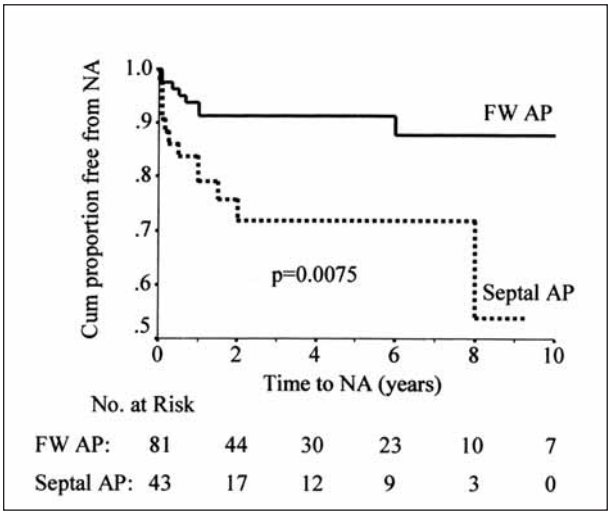
The global actuarial curve for freedom from NA is shown in Graph 1. After a mean one-year follow-up event-free survival was 87.1% (81.2-93.0%), at two years 84.6% (77.9-91.3%) and at six years 82.2% (74.2-90.2%). In addition, the patients with shorter pre-ablation history of tachyarrhythmias (≤10 years) showed better event-free survival, compared to the patients with a longer history of tachyarrhythmias before the procedure (p=0.014), Graph 2. Graph 3 shows better event-free survival after ablation of free wall APs, then after septal APs ablation (p=0.0075). Finally, the event-free survival was significantly worse in patients of age ≥35 years at procedure than in patients who were treated at a younger age (p=0.021), Graph 4.

Origin of new arrhythmias

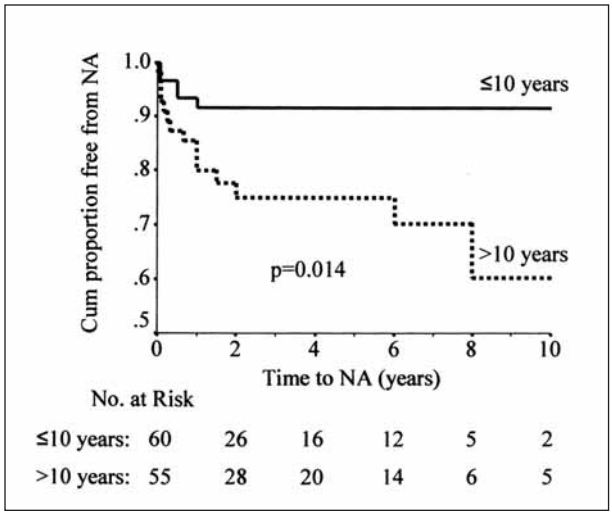
Twelve-lead ECG and Holter-recording during arrhythmia were available for analysis in 18 (78.2%) and five (21.8%) of 23 NA cases, respectively. In four of these cases (patients 2, 4, 5 and 12 in Table 3), the presumed origin sites of NA were in the vicinity of previous ablation targets: two cases of VPBs of right midseptal and left posteroseptal morphology after right midseptal and left posteroseptal AP ablation, respectively (Figure 1), one case of typical AFL after right posteroseptal AP ablation and single case of AV-block after right midseptal AP ablation. However, the location of NA focus was far away from the index RF lesion in 10 cases, while in the remaining nine it was impossible to analyze the arrhythmia origin site (four AF cases and five NAs obtained only from 2-channel Holter-recordings).



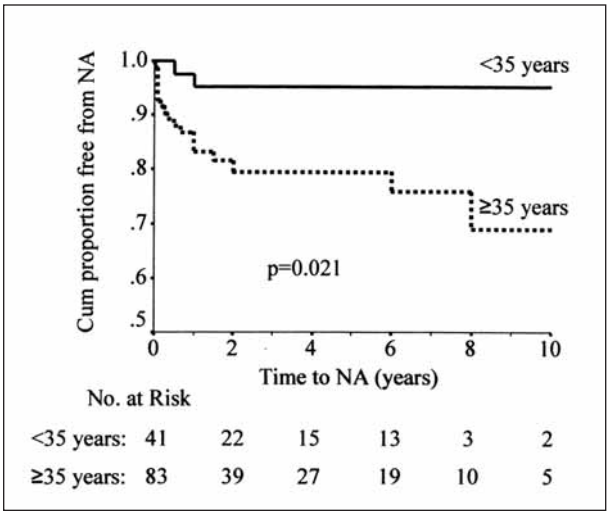
Graph 1. Cumulative proportion post-ablation freedom from new arrhythmia (NA) events



Graph 3. Comparison between patients with septal and free-wall pathways (AP – accessory pathway; FW – free-wall)



Graph 2. Comparison between patients with pre-ablation history of symptoms ≤10 and >10 years



Graph 4. Comparison between patients <35 and >35 years of age at procedure

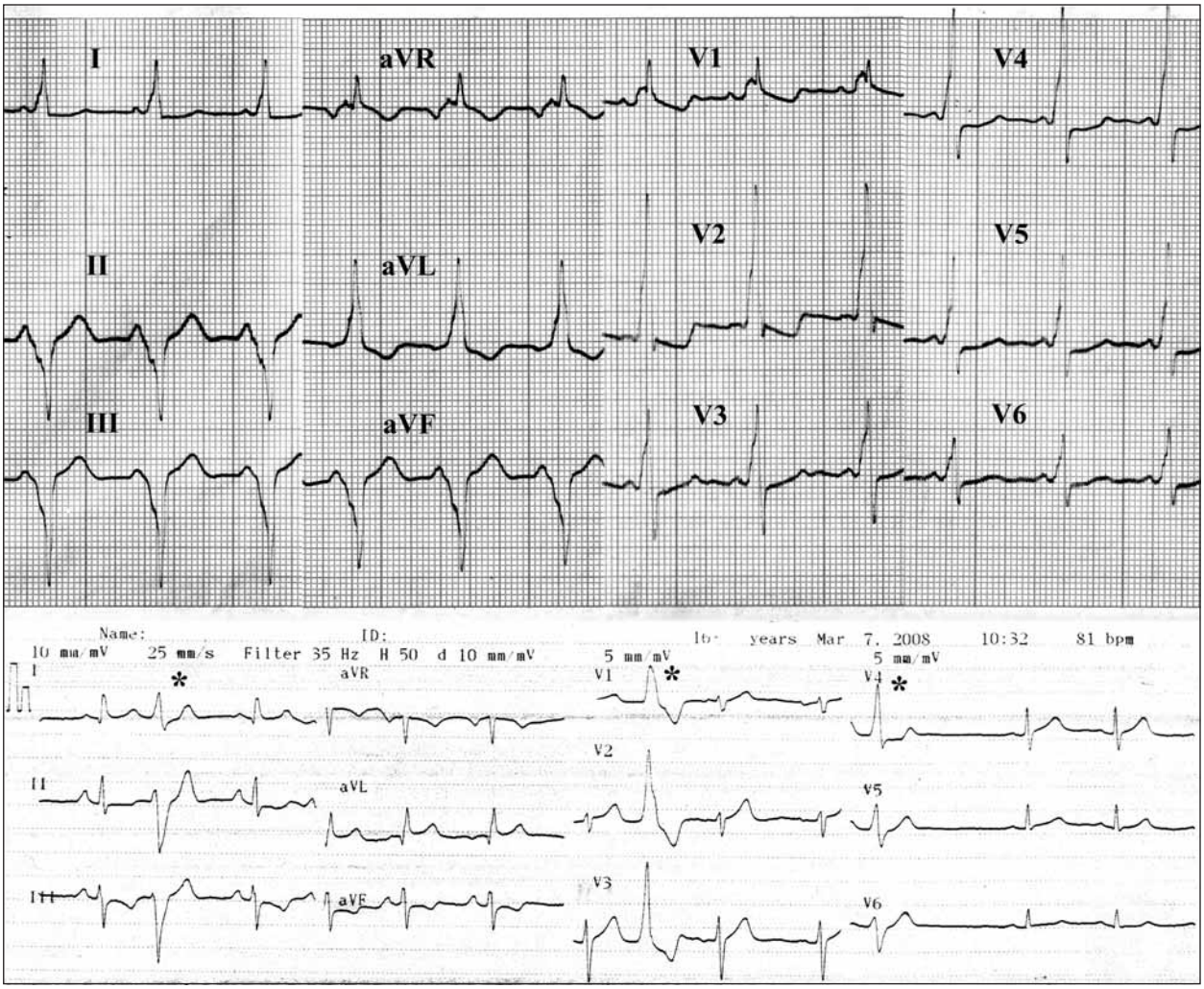


Figure 1. Upper panel represents an ECG obtained before ablation of left posteroseptal AP in patient 12 from Table 3. Eighteen months following procedure, 24-hour Holter-monitoring revealed more than 40,000 symptomatic ventricular premature beats (VPBs), not recorded before the procedure and ECG showed VPBs (bottom panel, asterisks) of left posteroseptal morphology [13]. Note that QRS-morphology of VPBs is very similar to delta-wave morphology before ablation. Thus, the VPBs focus could be in the close proximity to previous ablation target (i.e. AP) and delayed on-site arrhythmic side-effect of an old RF lesion seems possible.

DISCUSSION

Previous long-term studies after successful AP ablation have focused mainly on the recurrence of pathway conduction [2, 3] and relapse of AF [13, 14], while development of NAs was not evaluated extensively. It was reported that late after permanent AP elimination, 13% of patients complained of short bouts of palpitations different from the pre-ablation period [2]. The noninvasive investigations showed isolated or short runs of atrial or ventricular premature beats or normal sinus rhythm related to symptom recurrence in 48% of these patients. However, in 52% of symptomatic patients symptom-related arrhythmias remained undetermined.

In the present study NAs were found in 16% of the patients during the mean follow-up of 4.3 years after successful AP ablation. The NAs occurred between two days and eight years after the procedure, but in the majority of patients (80%) these arrhythmias occurred within the first post-ablation year, which was the minimal follow-up time required for all patients in this study. Therefore, longer follow-up would probably identify a small number of new patients with NAs. Although benign arrhythmias (i.e. sinus tachycardia, isolated VPBs and paroxysmal AF) were encountered most frequently and sustained ventricular tachyarrhythmias were not recorded in this study, there was one case of late progression of AV-block, accompanied by syncope. Noteworthy, in 90% of NA patients, additional pharmacological or interventional treatment was required after AP elimination, mainly on the symptomatic basis. It was recognized that patients with a clinical history of paroxysmal supraventricular tachycardias are very susceptible to irregular heart beat after successful ablation and are more easily treated with drug therapy for any arrhythmia after the procedure [5, 15]. These patients may develop an increased level of discomfort during normally innocent and asymptomatic arrhythmias that they have learned to recognize as triggers of tachycardia before ablation.

The present study identified two independent predictors for NA occurrence after RF ablation of AP: 1) pre-ablation history of tachyarrhythmia-related symptoms >10 years; and 2) septal pathway location.

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It is well known that the prevalence of cardiac arrhythmias in overall population increases with aging [16, 17]. Although older age at ablation (≥35 years) was found by univariate analysis to implicate higher risk for NA occurrence in the present study, this was not maintained in the multivariate model. Therefore, longer pre-ablation history of symptoms in NA patients may not be simply the function of their older age at the procedure. We speculate that prolonged history of pathway-mediated tachyarrhyhmias could promote myocardial changes, making the heart susceptible to NAs after AP ablation. Analogously, it was reported that patients with relapses of AF after AP ablation had more frequent palpitation attacks per month before the procedure [13]. These findings concordantly suggest that early intervention in symptomatic patients may prevent later development of arrhythmias after AP elimination.

It has been reported that ablation of septal targets can be followed by the appearance of inappropriate sinus tachycardia in 8-21% of the patients [18, 19], as well as by increase in ventricular ectopic activity [20]. In addition, over the 10-year follow-up after AVNRT ablation using RF, late AV-block and new supraventricular arrhythmias were registered in 1.7% and 24% of the patients, respectively [5]. It is suggested that interatrial septum in humans is rich in autonomic innervation and that autonomic nerves become sparser as one moves along the AV-grooves toward the free walls [18]. Inadvertent destruction of septal autonomic fibres by RF may lead to transient parasympathetic dennervation of the heart, sometimes lasting more than one year [18-21]. In accordance with the aforementioned reports, the present study identified the septal AP location as the strongest independent predictor for NA occurrence after ablation (HR=4.25). Therefore, closer follow-up of patients after septal pathways ablation seems reasonable.

A RF lesion is characterized by ragged edges, less clearly demarcated from the underlying normal myocardium [4]. This can cause non-uniform anisotropy, which may be responsible for slow conduction that allows (micro) reentry tachycardia to occur [5]. In four of 23 NA cases, presumed location of arrhythmic focus was found in proximity to previous RF lesion. Namely, in two cases, VPBs of right midseptal and left posteroseptal QRS-morphology were recognized after ablation of right midseptal and left posteroseptal pathway, respectively. In the third patient, typical AFL evolved after right posteroseptal AP ablation, at the septal level of cavotricuspid isthmus, while in the forth patient, late AV-block was registered following right midseptal AP ablation. Obviously, at least in some of these cases, late on-site arrhythmic side effect of RF lesion seems possible.

Study limitations

This study has the following limitations: 1) the absence of post-ablation EP study to evaluate origin and mechanism of NAs; 2) as the follow-up was based principally on periodic ECG recordings and symptoms, underestimation of (asymptomatic) NAs was possible; 3) Holter-monitoring was not conducted systematically before and after the procedure in all patients.

CONCLUSION

This long-term follow-up study showed that symptomatic NA occurrence may influence clinical outcome in 16% of patients after definite AP ablation by RF current. Although benign arrhythmias were identified in the majority of these patients, a very late progression of AV-block was recorded. The baseline clinical and electrophysiological characteristics can help identify a subgroup of patients who are at a higher risk of developing NA after the procedure. Early intervention in symptomatic WPW-patients might reduce NA occurrence and closer follow-up with screening for NA occurrence at least in the first post-ablation year is advised after ablation of septal pathways. At least in some of NA cases, late pro-arrhythmic effect of initially curative RF lesion seems possible. These findings can help physicians inform patients properly before the procedure about possible NA occurrence, as well as on affect of the decision to ablate in asymptomatic patients with ventricular preexcitation.

REFERENCES

1. Jackman WM, Wang X, Friday KJ, Roman CA, Moulton KP, Beckman KJ, et al. Catheter ablation of accessory atrioventricular pathways (Wolff–Parkinson–White syndrome) by radiofrequency current. N Engl J Med. 1991; 324:1605-11.
2. Schläpfer J, Fromer M. Late clinical outcome after successful radiofrequency catheter-ablation of accessory pathways. Eur Heart J. 2001; 22:605-9.
3. Dagres N, Clague JR, Kottkamp H, Hindricks G, Breithardt G, Borggrefe M. Radiofrequency catheter ablation of accessory pathways. Outcome and use of antiarrhythmic drugs during follow-up. Eur Heart J. 1999; 20:1826-32.
4. Khairy P, Chauvet P, Lehmann J, Lambert J, Macle L, Tanguay JF, et al. Lower incidence of thrombus formation with cryoenergy versus radiofrequency catheter-ablation. Circulation. 2003; 107:2045-50.
5. Kimman GP, Bogaard MD, van Hemel NM, van Dessel PF, Jessurun ER, Boersma LV, et al. Ten year follow-up after radiofrequency catheter-ablation for atrioventricular nodal reentrant tachycardia in the early days forever cured, or a source for new arrhythmias? Pacing Clin Electrophysiol. 2005; 28:1302-9.
6. Aryana A, D'Avila A, Ruskin JN, Reddy VY. The proarrhythmic effect of incomplete pulmotricuspid isthmus ablation in a patient with sarcoid-related ventricular tachycardia? J Cardiovasc Electrophysiol. 2008; 19:869-72.
7. Daoud EG, Weiss R, Augostini R, Hummel JD, Kalbfleisch SJ, Van Deren JM, et al. Proarrhythmia of circumferential left atrial lesions for management of atrial fibrillation. J Cardiovasc Electrophysiol. 2006; 17:157-65.
8. Arruda MS, McClelland JH, Wang X, Beckman KJ, Widman LE, Gonzalez MD, et al. Development and validation of an ECG algorithm for identifying accessory pathway ablation site in Wolff-Parkinson-White syndrome. J Cardiovasc Electrophysiol. 1998; 9:2-12.
9. Kistler PM, Roberts-Thomson KC, Haqqani HM, Fynn SP, Singarayar S, Vohra JK, et al. P-wave morphology in focal atrial tachycardia: development of an algorithm to predict the anatomic site of origin. J Am Coll Cardiol. 2006; 48:1010-7.

10. Ito S, Tada H, Naito S, Kurosaki K, Ueda M, Hoshizaki H, et al. Development and validation of an ECG algorithm for identifying the optimal ablation site for idiopathic ventricular outflow tract tachycardia. J Cardiovasc Electrophysiol. 2003; 14:1280-6.
11. Tada H, Ito S, Naito S, Kurosaki K, Kubota S, Sugiyasu A, et al. Idiopathic ventricular arrhythmia arising from the mitral annulus: a distinct subgroup of idiopathic ventricular arrhythmias. J Am Coll Cardiol. 2005; 45:877-86.
12. Tada H, Tadokoro K, Ito S, Naito S, Hashimoto T, Kaseno K, et al. Idiopathic ventricular arrhythmias originating from the tricuspid annulus: prevalence, electrocardiographic characteristics, and results of radiofrequency catheter-ablation. Heart Rhythm. 2007; 4:7-16.
13. Oddsson H, Edvardsson N, Walfridsson H. Episodes of atrial fibrillation and atrial vulnerability after successful radiofrequency catheter-ablation in patients with Wolff–Parkinson–White syndrome. Europace. 2002; 4:201-6.
14. Dagres N, Clague JR, Kottkamp H, Hindricks G, Breithardt G, Borggrefe M. Impact of radiofrequency catheter-ablation of accessory pathways on the frequency of atrial fibrillation during long-term follow-up: high recurrence rate of atrial fibrillation in patients older than 50 years of age. Eur Heart J. 2001; 22:423-7.
15. Oddsson H, Walfridsson H, Edvardsson N. Perception and documentation of arrhythmias after successful radiofrequency catheter-ablation of accessory pathways. Ann Noninvasive Electrocardiol. 2001; 6:216-21.
16. Kostis JB, McCrone K, Moreyra AE, Gotzoyannis S, Aglitz NM, Natarajan N, et al. Premature ventricular complexes in the absence of identifiable heart disease. Circulation. 1981; 63:1351-6.
17. Heeringa J, van der Kuip DAM, Hofman A, Kors JA, van Herpen G, Stricker BH, et al. Prevalence, incidence and lifetime risk of atrial fibrillation: the Rotterdam Study. Eur Heart J. 2006; 27:949-53.
18. Kočović DZ, Harada T, Shea JB, Soroff D, Friedman PL. Alterations of heart rate and of heart rate variability after radiofrequency catheter-ablation of supraventricular tachycardia. Delineation of parasympathetic pathways in the human heart. Circulation. 1993; 88:1671-81.
19. Pappone C, Stabile G, Oreto G, De Simone A, Rillo M, Mazzone P, et al. Inappropriate sinus tachycardia after radiofrequency ablation of para-hisian accessory pathways. J Cardiovasc Electrophysiol. 1997; 8:1357-65.
20. Jinbo Y, Kobayashi Y, Miyata A, Chiyoda K, Nakagawa H, Tanno K, et al. Decreasing parasympathetic tone activity and proarrhythmic effect after radiofrequency catheter-ablation – differences in ablation site. Jpn Circ J. 1998; 62:733-40.
21. Guo H, Wang P, Xing X, Peng F, Jiang J, Yang B, et al. Delayed injury of autonomic nerve induced by radiofrequency catheter-ablation. J Electrocardiol. 2007; 40:355.e1-e4.

Појава нових аритмија након катетер-аблације акцесорног пута: удаљени проаритмијски ефекат терапијске радиофреквентне лезије?

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КРАТАК САДРЖАЈ

Увод Након аблације акцесорног пута (АП) могу се јавити нове аритмије, али није разјашњено да ли ове аритмије могу настати као касна последица терапијске радиофреквентне (РФ) лезије.

Циљ рада Циљ рада је био да се утврде преваленција и предиктори појаве нових аритмија након аблације АП, те испита могућност проаритмијског ефекта РФ лезије.

Методе рада Укупно 124 болесника (88 мушкараца; средња старост 43±14 година) са Волф–Паркинсон–Вајтовим (*Wolff–Parkinson–White*) синдромом и појединачним АП клинички су праћена након успешне РФ аблације. Аритмије након аблације, које нису забележене пре процедуре, означене су као нове аритмије. Њихово исходиште је утврђивано анализом морфологије *P*-таласа и *QRS*-комплекса, а затим је упоређивано с локализацијама претходно елиминисаних АП.

Резултати Болесници су клинички праћени 4,3±3,9 година, а нове аритмије су утврђене код 20 болесника (16%). Утврђена је следећа преваленција појединих нових аритмија: атриовентрикуларни (АВ) блок 0,8%, атријалне екстрасистоле 1,6%, атријална фибрилација 5,4%, атријални флатер 0,8%, синусна тахикардија 4,8%, вентрикуларне екстрасистоле (ВЕС) 7,3%. Мултиваријантном Коксовом (*Cox*) регресионом анализом препознати су независни предиктори појаве нових аритмија: препроцедурална историја тахиаритмија које су посредоване АП дужа од десет година (*HR*=3,54; *p*=0,016) и септална локализација АП (*HR*=4,25; *p*=0,003). Претпостављена локализација фокуса нових аритмија је у четири случаја установљена у близини претходне аблативне лезије (два случаја септалних ВЕС, један типичан атријални флатер и један АВ-блок).

Закључак Нове аритмије су дијагностиковане код 16% болесника након елиминације АП. У ретким случајевима оне су могле настати због касног проаритмијског ефекта претходне терапијске РФ лезије. Чини се да би ранија интервенција могла спречити појаву нових аритмија, те се препоручује клиничко праћење болесника након аблације септалних АП.

Кључне речи: радиофреквентна аблација; катетер-аблација; акцесорни пут

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