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Long-term outcome of catheter ablation for atrial tachyarrhythmias in patients with atrial septal defect

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Abstract

Purpose This study aimed to evaluate efficacy, safety, and long-term outcome of radiofrequency catheter ablation (RFCA) for atrial tachyarrhythmia (ATa) in patients with atrial septal defect (ASD).

Methods Seventy-five ASD patients with ATa (52% atrial fibrillation, AF) were enrolled. Electrophysiological study and RFCA were performed, with endpoints of index and multiple procedures as follows: (a) atrial tachycardia/atrial flutter (AT/AFL), absence of inducibility of any atrial arrhythmia and (b) AF, circumferential pulmonary vein ablation (CPVA, paroxysmal AF), bidirectional block of lines, and disappearance of complex fractionated atrial electrograms (persistent and long-standing persistent (LSP)-AF).

Results Cumulative success rate at 1-year follow-up was 79.9% and dropped to 59.0% at a median follow-up of 63 months (range, 14–114 months) for multiple procedures (mean 1.6 ± 0.7 [1–3]). Freedom from ATa after multiple procedures was achieved in 75% patients with AT/AFL and 43.6% patients with AF ($P = 0.006$ for comparison). In multivariate analysis, older age at ASD correction (HR, 1.033 [95% CI, 1.008–1.059]; $P = 0.01$), ASD diameter before correction (HR, 1.054 [95% CI, 1.006–1.105]; $P = 0.027$), and first-diagnosed ATa type (AF; HR, 2.25 [95% CI, 1.03–4.92]; $P = 0.042$) were significant independent predictors of ATa recurrence. Patients with more risk factors had higher risk of ATa recurrence.

Conclusions The long-term outcome of RFCA for ATa outcome was favorable for AT/AFL while mediocre for AF. ATa recurrence was more common in patients with older age at ASD correction, larger ASD diameter before correction, and first-diagnosed AF. Patients with more risk factors had higher ATa recurrence risk.

Keywords Atrial septal defect · Radiofrequency catheter ablation · Atrial tachyarrhythmia · Long-term outcome

1 Introduction

Atrial septal defect (ASD) constitutes the major part of congenital heart disease, which is one of the most prevalent forms of major birth defects [1]. Atrial tachyarrhythmias (ATa) are common among these patients due to structural heart disease or as a result of the interventional or surgical procedures. Overall, the 20-year risk of developing atrial arrhythmia was 7% for patients aged 20, and increased to 38% for patients aged 55 [2]. And the efficacy of antiarrhythmic drug such as sotalol and dofetilide for such patients was unsatisfactory [3]. Radiofrequency catheter ablation (RFCA) has become a major treatment for atrial arrhythmias such as atrial fibrillation (AF) and atrial tachycardia (AT). However, studies on RFCA for ATa patients with ASD are lacking. Therefore, we evaluated efficacy and safety of RFCA for ATa patients with ASD.

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2 Methods

2.1 Patients

Overall, from January 2008 to December 2016, 75 consecutive patients (age, 24–71 years) with symptomatic ATa with ASD were enrolled. The inclusion criteria were as follows: (1) aged ≥ 18 years; (2) patients with history of corrected ASD by surgical repair or transcatheter occlusion; (3) patients with ATa history before ASD correction were excluded; (4) ATa patients with failure or intolerance

to antiarrhythmic drug therapy; (5) patients with cardiomyopathy or coronary artery disease were excluded by a combination of detailed histories, physical examination, and coronary angiography or coronary computerized tomography; (6) patients with acute viral myocarditis were excluded. The diagnosis of ASD was in accordance with the European Society of Cardiology criteria [4]. Standard Doppler transthoracic echocardiography and coronary angiography or coronary computerized tomography were performed in each patient before the procedure so as to evaluate cardiac function and exclude ischemic heart disease. Transesophageal echocardiography was also performed in each patient to exclude left atrial thrombus. Considering that atrial flutter (AFL) is substantially a specified macro-reentry tachycardia, it was classified into AT. AT and AFL together was expressed as AT/AFL (Fig. 1). The definitions of ATa, including AT/AFL and AF (paroxysmal AF, persistent AF, long-standing persistent AF or LSP-AF for short), conformed to ACC/AHA/HRS criteria [5]. Anticoagulation was applied using therapeutic warfarin with international normalized ratio (INR) 2.0–3.0 plus bridging low molecular weight heparin. Antiarrhythmic drugs (AADs) were discontinued for more than 5 half-lives before procedure. Informed consent was obtained from all individual participants included in the study, and the study conformed to the ethical guidelines of the Declaration of Helsinki and was approved by institutional ethics committee.

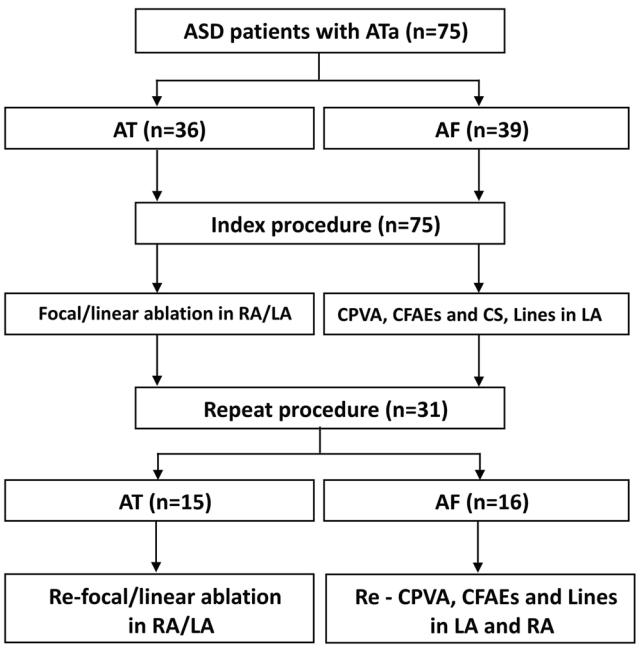

Fig. 1 Ablation strategy in the study. ASD, atrial septal defect; ATa, atrial tachyarrhythmia; AT, atrial tachycardia; AF, atrial fibrillation; LA, left atrium; RA, right atrium; CPVA, circumferential pulmonary vein ablation; CFAEs, complex fractionated atrial electrograms; CS, coronal sinus; ABL, ablation

2.2 Electrophysiological study

A 6-F decapolar diagnostic catheter for pacing and recording was positioned in the coronary sinus (CS). For AT/AFL, a 3.5-mm open irrigated-tip ablation catheter (Navistar-Thermocool, Biosense-Webster, Diamond Bar, CA, USA) was used for mapping and ablation. If AT/AFL originated from the left atrium (LA), mapping and ablation was implemented after transseptal puncture. In patients with transcatheter ASD closure history, atrial septal puncture was performed through an ASD occluder or adjacent to the rim of the ASD occluder (Fig.3e–h), depending on the occluder size and patient's atrial size. And all the procedures were performed successfully without complication. For AF, a Navistar catheter and a circular mapping catheter (Lasso, Biosense-Webster, Diamond Bar, CA, USA) were inserted through two transseptal punctures. Unselective angiography of all accessible pulmonary veins (PVs) was performed before ablation with injection of 5–10-ml contrast via the sheath. Anticoagulation was initiated with 50 IU/kg heparin adjusted as required to maintain activated clotting time ranging 250 to 300 s.

2.3 Catheter ablation

2.3.1 Index procedure

As was described in our previous study [6], a three-dimensional mapping system (Carto, Biosense-Webster, Diamond Bar, CA, USA) was applied to guide the ablation. For AT/AFL, the mechanisms of AT/AFL were identified by activation and entrainment mapping. RFCA was then performed according to the mechanisms of AT/AFL in each patient. Scar was defined as the presence of a signal with amplitude < 0.05 mV. The atriotomy was defined as a linear lesion with double atrial potentials separated by an electrical line indicating conduction block. For paroxysmal AF, circumferential pulmonary vein ablation (CPVA) was performed and pulmonary vein isolation (PVI) was monitored during the procedure. PVI was confirmed via a circular mapping catheter in each PV. For persistent and LSP-AF, CPVA was the first step. Secondly, three lines were ablated as follows: (1) the roof line, between the 2 PV circles; (2) the mitral isthmus (MI) line, between the annulus and the left inferior PV; (3) the right cavo–tricuspid isthmus (CTI) line if AT/AFL was CTI-dependent. Thirdly, complex fractionated atrial electrograms (CFAEs) were mapped and ablated in LA. The definition of CFAEs included (1) atrial electrograms with fractionation and composed of two deflections or more and/or with continuous activity of the baseline with continuous deflection of a prolonged activation complex over a 10-s period or (2) atrial electrograms with a very short cycle length, ≤ 120 ms. Irrigated radiofrequency energy was delivered on the

endocardium during CPVA with a flow rate of 20 ml/min and a maximum power of 30 W. While inside the CS, the irrigation rate was limited to 25 ml/min and the maximum ablation power was 25 W. If AF was not terminated after the latter steps, cardioversion was applied to restore sinus rhythm (SR). Under SR, PVI was reconfirmed and additional linear ablation undertaken if necessary to obtain bidirectional block of lines.

2.3.2 Repeat procedure

If first-diagnosed AT/AFL presented with recurrent AT/AFL, activation and entrainment mapping were used to identify underlying mechanisms and to guide the following ablation. After terminating AT/AFL, short-duration burst pacing from RA, CS, and PVs was applied to induce AT/AFL under isoproterenol infusion. For first-diagnosed AF patients, if the patients presented with recurrent AT/AFL, PVI and bidirectional block of three lines were reconfirmed after AT/AFL was terminated. For patients with recurrent AF, firstly, PVI was reconfirmed with a circular mapping catheter at each PV and CPVA was applied to eliminate recovery of PV potentials. Secondly, CFAEs were remapped and ablated in the LA and the RA. Thirdly, after restoring SR by ablation or cardioversion, bidirectional block of three lines was reconfirmed.

2.4 Follow-up

All patients were assessed with 12-lead electrocardiograph (ECG) and 24-h Holter monitoring during follow-up. Patients were anticoagulated with warfarin with a target INR of 2–3 after the procedure. Patients with evidence of fluid retention were prescribed diuretics. And a 3-month blanking period was observed, during which ATa recurrence was managed with AADs. Patients were instructed to obtain an ECG when symptoms occurred. The primary study outcome was rate of survival free from recurrence of ATa, which was defined according to consensus guidelines as any documented electrocardiographic episodes of ATa lasting at least 30 s with or without symptoms [7].

2.5 Statistical analysis

Normally distributed continuous variables were expressed as mean ± standard deviation, abnormally distributed continuous variables as median (range), and discrete variables as percentages. Comparisons between variables were conducted by χ^2 test for categorical variables, independent samples *t* test for continuous variables with normal distribution, and Mann–Whitney *U* test for continuous variables with abnormal distribution. Event-free survival was estimated by the Kaplan–Meier method and compared by the log-rank test. Cox regression was employed to determine the relation between baseline characteristics and recurrence of ATa. A P value < 0.05 was considered statistically significant.

3 Results

Clinical characteristics are presented in Table 1. All of the 75 patients enrolled had ostium secundum ASD, including 36 AT/AFL patients (48%) and 39 AF patients (52%). Among

Table 1 Clinical characteristics of the patients

Variable	$N = 75$
Age, years (median, IQR)	56 (24–71)
Male gender (%)	36
Age at ASD correction (years)	38.8 ± 16.3
ASD diameter (mm)	19.3 ± 7.9
ASD correction (%)	
Transcatheter occlusion	22 (29.3)
Surgical closure	53 (70.7)
ATa duration, months (median, IQR)	33 (3–48)
ATa type (%)	
Atrial tachycardia	36 (48)
Paroxysmal AF	18 (24)
Persistent AF	15 (20)
Long-standing persistent AF	6 (8)
NYHA functional class	
I	5 (6.7)
II	66 (88)
III	4 (5.3)
Diabetes	10 (13.3)
Stroke	3 (4.7)
LA diameter (mm)	41.8 ± 7.9
LV end-diastolic diameter (mm)	46.4 ± 5.8
LV ejection fraction (%)	60.8 ± 4.3
Pulmonary artery pressure (mmHg)	36.9 ± 10.2
RA enlargement (%)	43 (57.3)
MR	
Non (%)	19 (25.3)
Mild (%)	34 (45.3)
≥ moderate (%)	22 (29.3)
AR	
Non (%)	71 (94.7)
Mild (%)	4 (5.3)
TR	
Non (%)	19 (25.3)
Mild (%)	31 (41.3)
≥ moderate (%)	25 (33.4)

Values are expressed as mean ± SD or *n* (%). *ATa* indicates atrial tachycardia arrhythmias; *AF*, atrial fibrillation; *IQR*, interquartile range; *LA*, left atrium; *LV*, left ventricle; *RA*, right atrium; *MR*, mitral regurgitation; *TR*, tricuspid regurgitation; *AR*, aortic regurgitation

AF patients, there were 18 paroxysmal AF, 15 permanent AF, and 6 LSP-AF. No patients with surgically repaired ASD underwent MAZE surgery. Before RFCA, 53 patients underwent surgical correction, of which 29 patients developed AT/AFL and 24 had AF, and 22 patients underwent transcatheter occlusion, of which 7 patients developed AT/AFL and 15 patients had AF. And no significant difference was observed in the pattern of arrhythmias between patients undergoing surgical or percutaneous ASD closure ($P = 0.071$). The ASD diameter before correction was 19.3 ± 7.9 mm (range, 6–35 mm), the mean LA diameter was 41.8 ± 7.9 mm, and the LV ejection fraction was 60.5 ± 4.1%. Mitral regurgitation, tricuspid regurgitation, and RA enlargement were shown in 56 (74.7%), 56 (74.7%), and 43 (57.3%) patients, respectively. All patients were prescribed with AADs including propafenone or amiodarone after RFCA. No residual shunt was observed.

3.1 Outcomes of index procedure

In all AT/AFL patients, 43 mechanisms were identified, including 9 foci or micro-reentry (from RA) and 34 macro-reentry (23 scar-related, 7 CTI-related, and 4 CS-related AT/AFLs). As for the macro-reentry AT/AFLs, the critical isthmus was located from the scar to the superior vena cava (SVC) in 13 patients, from the scar to the inferior vena cava (IVC) in 23 patients, and from the CS to the IVC in 4 patients and cavo–tricuspid isthmus in 7 patients (Fig. 3a–d). In AF patients, CPVA and PVI were achieved in all patients with paroxysmal AF. While for those with persistent AF and LSP-AF (21 patients), CPVA and PVI were achieved in all patients and bidirectional block of the MI, roof, and CTI lines were obtained in 18 (85.7%), 19 (90.5%), and 4 (19%) patients, respectively. AF was terminated by CFAE ablation in 8 patients, while 8 patients with persistent AF and 5 patients with LSP-AF converted to SR by cardioversion when ablation failed to restore SR. All procedures were performed without major complication.

All patients completed 12-month follow-up with 12-lead ECG and 24-h ambulatory ECG monitoring. At 1 year after a single procedure, freedom from ATa persisted in 47 (62.7%) of 75 patients (25 [69.4%] of 36 patients with AT/AFL and 22 [56.4%] of 39 patients with AF; $P = 0.244$ for comparison). Thirty-one patients with recurrent ATa underwent re-ablation, including 17 (54.8%) AT/AFL patients and 14 (45.2%) AF patients. Among recurrent AT/AFL patients, 15 (88.2%) were first-diagnosed AT/AFL and 2 (11.8%) were first-diagnosed AF, and all recurrent AF patients were first-diagnosed AF. During the re-ablation procedure, in 17 patients with recurrent AT/AFL, 18 mechanisms were identified, including foci or micro-reentry in 3 AT/AFLs (3 from RA) and macro-reentry in 15 AT/AFLs (6 scar-related AT/AFLs, 6 CTI-related AT/AFLs, 2 MI-related AT/AFLs, 1 roof-related AT/AFL). In 14 patients with recurrent AF who underwent re-ablation, 10 were paroxysmal AF, among which 8 were terminated by PVI and 2 restored SR by cardioversion; the remaining 4 patients were persistent AF or LSP-AF, and all underwent cardioversion to restore SR. Overall, the secondary success rate of all patients at 1 year was 73.3% off AADs. Arteriovenous fistula at the femoral vein occurred in 1 patient, and no major complication occurred during follow-up.

3.2 Long-term follow-up

At a median follow-up of 63 months (range, 14–114 months, 3-month blanking period included) after multiple procedures (average 1.6 ± 0.7 [1–3] ablation procedures), the cumulative success rate of multiple procedures was 59% (Fig. 2a). The median of follow-up duration of patients with percutaneous and surgical ASD closure was 54 months (range, 14–99 months) and 65 months (range, 14–114 months), respectively, and no significant difference was observed between these two groups ($P = 0.514$). Long-term freedom from ATa was achieved in 43 patients (57.3%), including 27 (75%) of 36 patients with AT/AFL and 17 (43.6%) of 39 patients with AF ($P = 0.006$ for comparison).

The majority of late recurrence occurred within 60 months. There were 10 patients that underwent third procedures, including 5 with recurrent AT/AFL and 5 with recurrent AF, and all with rate-control drug failure. During follow-up after the last procedure, 31 patients had recurrent ATa, including 15 AT/AFL and 16 AF. No death occurred during follow-up.

In multivariate analysis, after multiple procedures, age at ASD correction, ASD diameter before correction, and first-diagnosed ATa type (AT/AFL or AF) were independent risk factors of recurrent ATa. Patients with age at ASD correction ≥ 45 years old had a significantly higher recurrence rate compared with those who underwent ASD correction before 45 (69.0% vs. 23.9%, $P < 0.01$), and were almost 2.8 times more likely to relapse (≥ 45 years; HR, 3.80 [95% CI, 1.81–7.98]; $P < 0.01$; Fig. 2b). Patients first-diagnosed AF were almost 1.6 times more likely to suffer ATa recurrence (AF; HR, 2.58 [95% CI, 1.18–5.62]; $P = 0.017$; Fig. 2c) compared with those first-diagnosed AT/AFL (56.4% vs. 25%, $P = 0.006$). The recurrent rate of patients with ASD diameter ≥ 15 mm before correction was almost 7 times the rate of those < 15 mm (56% vs. 12%, $P < 0.01$ for comparison; ≥ 15 mm; HR, 6.9 [95% CI, 2.09–22.94]; $P = 0.002$; Fig. 2d).

The risk of ATa recurrence increased significantly as risk factors accumulated in a patient. Compared with patients without risk factor, patients with one risk factor had a similar recurrent risk (risk factor = 1; HR, 1.84 [95% CI, 0.19–17.8]; $P = 0.599$), while those with two were almost 9 times more likely to suffer from recurrent ATa (risk factor = 2; HR, 9.91 [95% CI, 1.30–75.73]; $P = 0.027$), and patients with three risk factors had the highest recurrent risk, which was almost

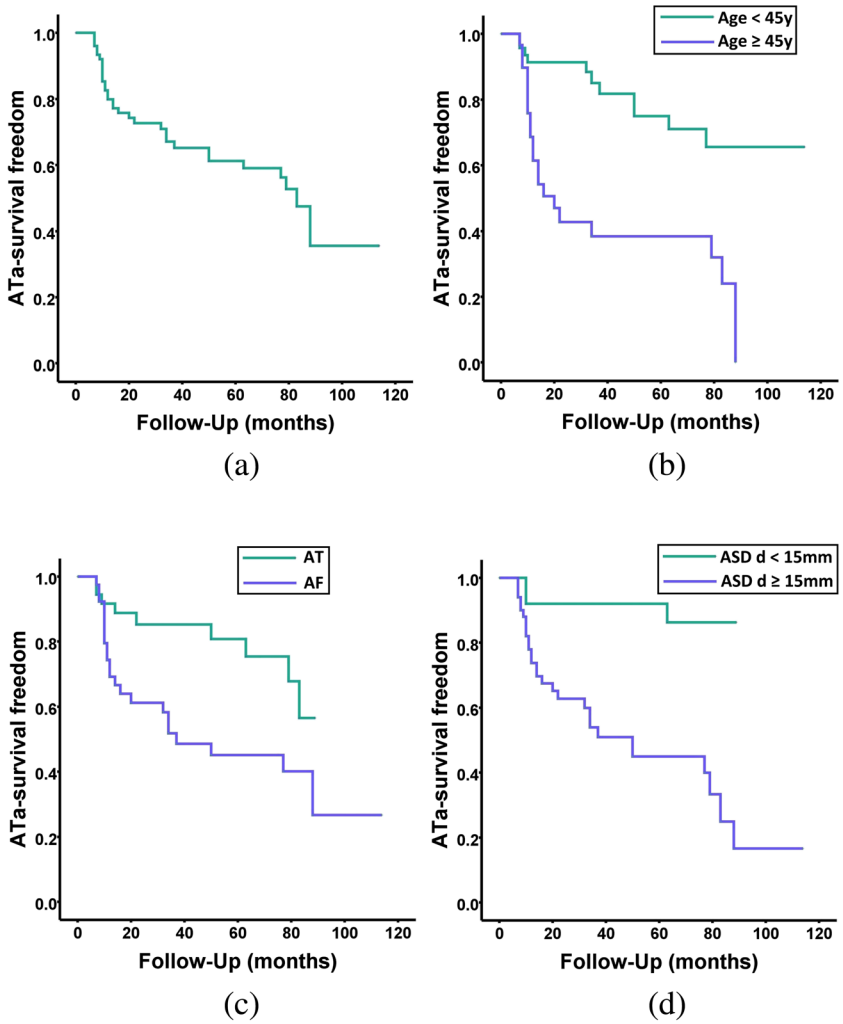

Fig. 2 Kaplan–Meier survival curve showing freedom from atrial tachyarrhythmia recurrence after multiple procedures in the total follow-up (**a**); multiple procedures according to patients' age at first ablation (**b**); multiple procedures according to first-diagnosed ATa type (**c**); and multiple procedures according to ASD diameter (**d**). ATa, atrial tachyarrhythmia; ASD, atrial septal defect

16.5 times more than those without risk factor (risk factor = 3; HR, 16.57 [95% CI, 2.15–127.81]; $P = 0.007$).

After multiple procedures, patients who underwent transcatheter occlusion had a higher rate of freedom from recurrent ATa than those who underwent surgical repair (66.0% vs. 40.1%, $P = 0.044$ for comparison). However, in Cox regression, no significant difference in ATa recurrence was observed between the transcatheter occlusion group and the surgical repair group (HR, 1.73 [95% CI, 0.85–3.54]; $P = 0.132$). In patients first-diagnosed either AT/AFL or AF, ATa recurrence did not seem to correlate with MR, TR, LA size, RA enlargement, pulmonary artery pressure, or NYHA classification (Fig. 3).

4 Discussion

Documenting outcomes of catheter ablation for ATa in a comparatively large series of patients with ASD, this study had major finding as follows: (1) multiple ablation methods including focal ablation, CPVA, linear ablation, and CFAE ablation were sufficient to restore SR in 62.7% patients at 1-year follow-up after a single ablation procedure; (2) long-term outcomes of RFCA for AT/AFL were favorable with a 75% multiple-procedure success rate but remained mediocre for AF with a 43.6% success rate after multiple procedures; (3) age at ASD correction, ASD diameter before correction, and first-diagnosed ATa type were independent risk factors of recurrent ATa; (4) patients with more risk factors had higher risk of ATa recurrence.

ATa is prevalent in patients with ASD, with possible reasons as follows: (a) atrium dilation due to volume overload, which is associated with development of fibrosis and electrical heterogeneity, facilitating the initiation and maintenance of ATa [8]; (b) incisional scar predisposing to reentrant circuits in patients who underwent ASD surgical correction [9]; (c) blood desaturation in ASD patients with pulmonary hypertension or heart failure facilitating development of ATa [10]. Catheter ablation has been

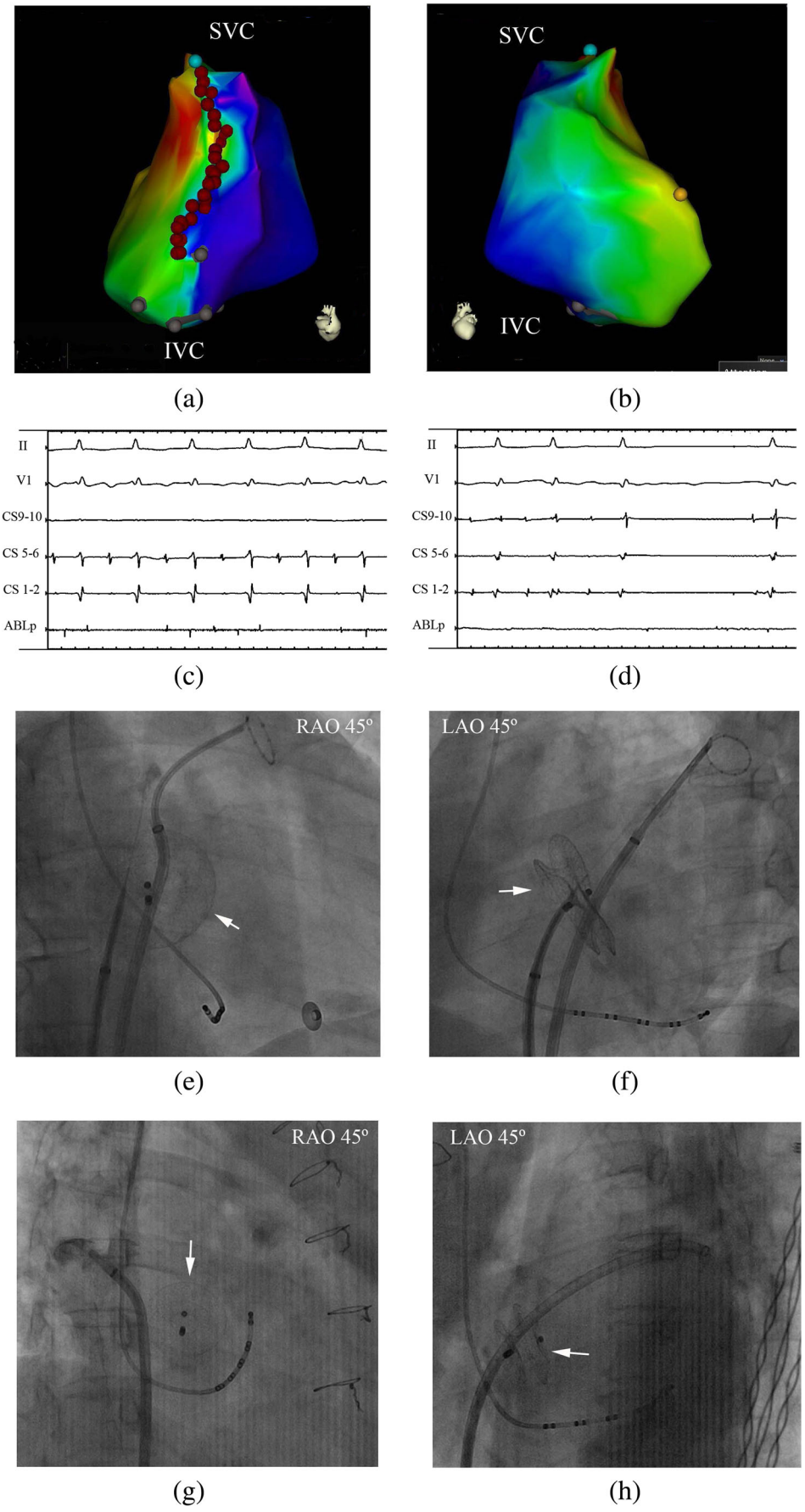

Fig. 3 Examples of electroanatomic mapping of a macro-reentrant atrial tachycardia and atrial septal puncture through and adjacent to the atrial septal occluder. **a**, **b** The activation maps showed a scar-related reentrant circuit in different views as indicated at the corner of each figure. Gery points indicate atriotomy scar at the lateral–posterior right atrial wall. The red dots indicate the linear ablation extending from the superior vena cava to the scar. The yellow dot indicates the His bundle. The color code shows the wavefront propagation around the scar. **c** Surface and intracardiac electrocardiogram of the atrial tachycardia. CS1–2, 5–6, and 9–10 recordings were from coronary sinus catheter. **d** Tachycardia interruption during linear ablation of the critical isthmus of conduction. CS1–2, 5–6, and 9–10 were recorded from coronary sinus catheter. **e,** f Atrial septal puncture through the ASD occluder in RAO 45° and LAO 45°, respectively. **g**, **h** Septal puncture adjacent to the ASD occluder in RAO 45° and LAO 45 °, respectively. Arrows indicated the ASD occluder. SVC, superior vena cava; IVC, inferior vena cava; ABL, ablation; p, proximity; RAO, right anterior oblique; LAO, left anterior oblique

proved effective and safe for ATa. However, our study documented limited benefit of RFCA for treating ATa in ASD patients (75% for AT/AFL, 43.6% for AF, and 57.3% overall). Recent studies on RFCA for ATa in patients with ASD are lacking, while Nie et al. [11] documented survival free from AF of 55.5% after a median follow-up of 20 months, and our study had cumulative success rate of 59% at a median 63-month follow-up, which seemed consistent with the former study.

In our study, more patients were diagnosed AF; however, it would seem more reasonable that more ASD patients with surgical correction develop AT/AFL rather than AF. Selection bias is one of the possible reasons for enrolling more AF patients since this is a single-center study. Besides, according to published experience [12], outcome of ASD patients operated on younger than 25 years old had satisfactory outcome. However, the average age of ASD closure was 38.8 ± 16.3 years old in our study, much older than 25. Thus, the surgical repair of the patients enrolled was rather late, allowing severer atrial remodeling and increasing propensity to develop AF.

Severity of atrial shunt was said to be determined by the size of defect and the pressure difference across the atrial septa [13]. It was reported that most ASD with diameter less than 10 mm was associated with a relatively small shunt, causing less structural remodeling of the right heart myocardium [12]. However, larger defect tends to cause enlargement of the heart structure, myocardial cell hypertrophy, and fibrosis through long-term volume and pressure overload, and associates with severer atrial remodeling. Besides, surgical closure for larger ASD tends to cause more complex structural remodeling by generating complex scar and implanted patch material; thus, larger ASD diameter is predisposed to associate with an increased risk of ATa recurrence.

It has been recognized that ASD correction could terminate or remit pre-existing abnormal hemodynamic condition, preventing or slowing further development of electrophysiological heterogeneity [14]. Right ventricular and atrial size may even reduce after ASD correction [15]. Previous studies yielded inconsistent conclusions on the effect of ASD correction on ATa occurrence. Roos-Hesselink et al. reported that early ASD closure had a lower incidence of ATa [16], while Avila et al. found that intracardiac repair increased the risk of AT/AFL but underpinned the protective effect of ASD correction on AT/AFL development [17]. In our study, electrophysiological study showed that scar-related AT/AFL was an important mechanism; however, we also observed that patients undergoing ASD correction before 45 years old had a significantly lower ATa recurrence rate than those aged above 45 at ASD correction. This is comprehensible since atrial remodeling in ASD patients deteriorates progressively [7]; thus, older patients possess more complex structural and electrophysiological atrial substrate predisposing to initiation and maintenance of ATa.

No significant difference was observed in ATa recurrence between surgical- and transcatheter-corrected patients in our study. Surgical correction was reported to reduce but not to prevent recurrence of ATa [18]. While transcatheter ASD occlusion was said to reduce risk of AF [19], the protective effect warranted further trials to confirm and it remained unclear if patients could benefit more from transcatheter occlusion than reparative surgery. No significant difference was observed in atrial size (RA and LA) between patients with different types of ATa (AT/AFL or AF) or recurrence of ATa. Indeed, it has been accepted that AF patients have a higher prevalence of larger atrial size and a higher recurrence rate. However, our study yielded different results. The possible reasons may include (1) the power of the study is limited by its small sample size; (2) most patients had surgical ASD closure, whose surgical scar was substrate for AT/AFL regardless of atrial size; (3) our study showed that ATa type (which implied complexity of the ATa mechanism), rather than atrial size, was correlated with ATa recurrence, indicating that atrial size was not entirely correlated with ATa type in ASD patients.

Another independent risk factor of ATa recurrence was first-diagnosed ATa type. In our study, patients with AT/AFL had a significantly lower recurrence rate than AF. The mechanisms of AT/AFL include focal activity, micro-reentry, and macro-reentry, and under most circumstances, such mechanisms are identifiable and thus terminated by RFCA. However, even with explanations such as focal activity [20], multiple wavelet hypothesis [21], and "rotors" [22], the exact electrophysiological mechanism of AF remains unclear, making it hard to terminate AF by targeting a specific mechanism.

Other than scar-related AT/AFLs, each AT/AFL pattern size was small (9 foci or RA micro-reentry, 7 CTI-related, and 4 CS-related AT/AFLs); thus, it was not surprising that no significant correlation was observed between these mechanisms and surgical scar or other clinical characteristics. However, we speculated that atrial volume and pressure overload caused by ASD was the primary cause of these ATs, and difference of AT patterns was possible between individuals.

In patients without any of these risk factors, freedom from ATa was comparatively high, and patients with only 1 risk factor had a similar ATa recurrent risk. On the other hand, ATa survival freedom decreased significantly as risk factors accumulated in 1 patient. Thus, active examination for ATa such as Holter monitoring in this kind of patients should be encouraged regardless of presence of palpitation.

5 Limitations

This study had several limitations. It is descriptive and nonrandomized, and had a relatively small sample size. Therefore, the results and conclusions warrant larger randomized controlled trials to confirm. Furthermore, 12-lead ECG and 24-h Holter, but not 7-day Holter monitoring or implantable loop recorders, was used to assess ATa recurrence, so the

success rate might be overestimated, especially for patients with symptom improvement.

6 Conclusions

Catheter ablation for ATa in ASD patients is safe. The long-term outcome appeared mediocre for AF; however, catheter ablation for ASD patients with AT/AFL seemed favorable. ATa recurrence was more common in patients with older age at ASD correction, larger ASD diameter before correction, and first-diagnosed AF. Patients with more risk factors had higher risk of ATa recurrence.

Compliance with ethical standards Informed consent was obtained from all individual participants included in the study, and the study conformed to the ethical guidelines of the Declaration of Helsinki and was approved by institutional ethics committee.

Conflict of interest The authors declare that they have no conflict of interest.

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