Iatrogenic aortic regurgitation after radiofrequency ablation of idiopathic ventricular arrhythmias originating from the aortic valvular region

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BACKGROUND Radiofrequency ablation at the aortic root has the potential risk of aortic regurgitation (AR).

OBJECTIVE This study investigated the incidence and clinical features of iatrogenic AR after catheter ablation of idiopathic ventricular arrhythmias originating from the aortic root.

METHODS We studied 45 consecutive patients with idiopathic ventricular arrhythmias originating from the aortic cusps (ACs; AC group; n = 32 [71%]) and papillary muscles (control group; n = 13 [29%]) who underwent ablation via a retrograde aortic approach and serial echocardiography before and within 24 hours after the ablation procedure. No patients had preexisting AR.

RESULTS After ablation, mild AR occurred in 5 AC group patients and 1 control group patient. Regurgitant flow was observed at the center of the aortic leaflets in 3 patients, the left coronary cusp–noncoronary cusp commissure in 2 patients, and both in 1 patient. No patients undergoing ablation only above the aortic valve developed AR. In AC group patients, the occurrence of AR was associated with a longer ablation time (24 ± 14 minutes vs 10 ± 5 minutes; $P < .01$) and higher average output (36.6 ± 4.2 W vs 32.0 ± 3.2 W; $P = .01$). The same severity of AR still existed after 16.2 ± 3.6 months of follow-up. No patients required any additional medical management or surgical intervention.

CONCLUSION Iatrogenic mild AR after ablation in the aortic root occurred with a noticeable prevalence, which was associated with extensive ablation both above and below the ACs as well as catheter-related mechanical factors. Although it did not appear to aggravate the hemodynamic status during the mid-term follow-up, careful monitoring of AR progression should be considered.

KEYWORDS Aortic cusp; Aortic regurgitation; Complications; Idiopathic ventricular arrhythmia; Radiofrequency catheter ablation

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Introduction

Radiofrequency catheter ablation (RFCA) has been established as an effective therapy for idiopathic ventricular arrhythmias (VAs). Although RFCA of idiopathic VAs can be performed safely, periprocedural complications such as groin vascular-related complications, cardiac tamponades, and thrombotic events may occur at a rate of ~3%–5%.[1–3] The aortic root including the left ventricular outflow tract (LVOT) and aortic coronary cusps are a common site of origin of idiopathic VAs. Previous case series have demonstrated that mapping and ablation in the aortic cusps (ACs) and the left ventricle (LV) via a retrograde aortic approach has a risk of life-threatening collateral damage, including acute occlusion of the coronary arteries,[4] coronary artery spasms ,[5] LV pseudoaneurysms,[6] and atrioventricular block.[7] Only 1 case report demonstrated aortic regurgitation (AR) due to ablation of VAs originating from the LVOT.[8] However, AR occurring after RFCA of VAs originating from the LVOT and ACs has not been systematically evaluated. This study investigated the incidence and clinical and procedural characteristics of AR occurring after RFCA in the aortic root.

Methods

Study population

This study investigated 45 consecutive patients with idiopathic VAs originating from the ACs (AC group; n = 32) and the papillary muscles (control group; n = 13) in whom RFCA was performed using a retrograde aortic approach between April 2013 and December 2017 at our institute. All patients underwent serial transthoracic echocardiography before and within 24 hours after the ablation procedure. There was no evidence of structural heart disease based on 12-lead electrocardiography during sinus rhythm, echocardiography, and coronary angiography. No patients had AR before the ablation procedure. Written informed consent of

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ablation was obtained from all the participants. The institutional review board approved the review of these data.

Echocardiography evaluation

Transthoracic 2-dimensional Doppler studies were performed by the same experienced sonographers. The details of ablation were blinded to the sonographers. The wall thickness, chamber size, and aortic root diameter were measured in the standard manner. The severity of AR was diagnosed by color Doppler and was semiquantitatively graded to non-AR, mild AR, moderate AR, and severe AR according to the ratio of the AR color jet area in the LVOT in accordance with the guidelines of the American Society of Echocardiography.[9]

Electrophysiology study and ablation procedure

An electrophysiology study was performed after discontinuation of all antiarrhythmic drugs. A 3-dimensional electroanatomic mapping system (CARTO3, Biosense Webster, Diamond Bar, CA) was used in all patients. An open irrigated catheter (ThermoCool Surround Flow or ThermoCool Smart Touch, Biosense Webster) was inserted via the right femoral artery and retrogradely advanced into the LV for mapping and ablation. Intravenous heparin was used to achieve an activated clotting time of $\geq$200 seconds. Coronary angiography was performed to delineate the ostium and course of the left coronary artery before and after the ablation procedure. In a subset of patients, intracardiac echocardiography (SoundStar, Biosense Webster) and the CartoSound system (Biosense Webster) were used to confirm the relationship between the cardiac anatomy and the location of the ablation catheter and to monitor the tissue-catheter contact. If few clinical VAs were observed before the radiofrequency (RF) applications, burst pacing from the right ventricular apex and/or right atrium was performed with the addition of intravenous isoproterenol (1–3 μg/min) to provoke clinical VAs.

RF applications were given at the site showing the earliest ventricular activation during the arrhythmia and/or a good pace map where the QRS morphology reproduced by pacing was similar to the clinical VA's morphology ($\geq$11/12-lead concordance). If the RF application elicited a suppression or elimination of the VA within the initial 30 seconds, the application was maintained for $\leq$120 seconds, targeting an impedance drop of 15 Ω with a maximum temperature of 45°C and a maximum power of 35 W above the ACs and 45 W on the LV endocardium below the ACs. The RF output was adjusted according to impedance drop, temperature, and contact force at the operator's discretion. The following parameters were recorded for each ablation application: average RF output, maximum RF output, average impedance drop, maximum impedance drop, and duration of ablation. After the ablation procedure, atrial and ventricular burst pacing with and without intravenous isoproterenol was used to assess arrhythmia inducibility. *Acute success* was defined as noninducibility of the clinical VA at the end of the procedure.

Follow-up

Patients were monitored for at least 24 hours in the hospital before discharge. No antiarrhythmic drugs were prescribed after the ablation procedure. Patients were prospectively followed up at 1, 3, 6, and 12 months after the procedure, with 12-lead electrocardiography at each visit and Holter monitoring every 6 months. Echocardiography was planned 1, 6, and 12 months after the catheter ablation procedure in all patients. Arrhythmia recurrence was assessed by patient interviews, 12-lead electrocardiograms, and Holter recordings.

Statistical analysis

Continuous variables were expressed as mean ± SD. Continuous data were compared using unpaired Student *t* tests and Mann-Whitney *U* tests for normally and nonnormally distributed variables, respectively. Categorical variables were expressed as number and percentage and were compared using χ^2 or Fisher exact tests, as appropriate. All tests were 2-tailed, and *P* values $<$.05 were considered statistically significant.

Results

Study population

The baseline characteristics of this study population are summarized in Table 1. No patients had any preexisting AR in either group. There were no significant differences in the clinical characteristics including age, sex, LV ejection fraction, VA burden, and brain natriuretic peptide level between the AC and control groups. The ablation procedure was the first attempt in all patients.

Incidence and characteristics of iatrogenic AR

In the AC group, the sites of RF applications were the left coronary cusp (LCC) in 22 patients (68%), LCC-RCC junction in 8 (25%), and right coronary cusp (RCC) in 2 (7%), as confirmed by the fluoroscopic views with aortograms and/or intracardiac echocardiography. New-onset AR occurred in 5 patients (15%). In the control group, the sites of RF applications were the posterior papillary muscle in 10 patients and anterior papillary muscle in 3. New-onset mild AR occurred in 1 patient (patient 6) after the ablation procedure at the posterior papillary muscle.

Figure 1 demonstrates a case of mild AR at both the center of the aortic leaflets and the NCC-LCC commissure (patient 1). The echocardiographic images after the RF procedure in other patients with AR (patients 2–6) are shown in Figure 2 and Supplemental Videos 1 and 2. Regurgitant flow was observed at the NCC-LCC commissure area in patient 2 (Figure 2A and Supplemental Video 1) and patient 3 (Figure 2B) and at the center of the aortic leaflets in patient 4 (Figure 2C and Supplemental Video 2) and patient 5 (Figure 2D). In the control group, AR at the center of the aortic leaflets was observed in patient 6 (Figure 2E). The severity of AR was mild in all these patients.

The patient characteristics and ablation data of the 6 patients who developed mild AR postablation are summarized

Table 1 Baseline characteristics of patients

Characteristic	Aortic valve-VA (n = 32)	Control (n = 13)	*P*
Age (y)	61.8 ± 15.1	55.1 ± 16.8	.19
Male sex	25 (78)	7 (54)	.10
Hypertension	19 (59)	4 (31)	.08
Diabetes	4 (13)	3 (23)	.37
eGFR (mL/(min•1.73 m^2))	78.4 ± 15.4	82.8 ± 15.7	.43
BNP level (pg/dL)	69.5 ± 84.5 (21–84)	55.3 ± 70.7 (15–44)	.62
Echocardiography			
LVEF (%)	62.2 ± 7.3	66.0 ± 4.2	.09
LVDd (mm)	52.7 ± 6.1	49.2 ± 4.9	.07
EDV (mL)	134 ± 37	113.8 ± 28.4	.08
AOD (mm)	33.6 ± 5.7	34.1 ± 4.3	.78
LVOT (mm)	24.1 ± 5.1	21.5 ± 1.7	.10
Clinical VA			
VPC	22 (68.8)	8 (61.6)	
VPC, nonsustained VT	7 (21.8)	3 (23.0)	
VPC nonsustained/sustained VT	3 (9.4)	2 (15.4)	
Ventricular arrhythmia burden (%)	22.5 ± 11.7 (13.5–31.8)	24.3 ± 18.7 (13.3–36.3)	.71

Values are presented as mean ± SD (range) or as n (%).

AOD = aortic diameter; BNP = brain natriuretic peptide; EDV = end-diastolic volume; eGFR = estimated glomerular filtration rate; LVDd = left ventricular diastolic dysfunction; LVEF = left ventricular ejection fraction; LVOT = left ventricular outflow tract; VPC = ventricular premature contraction; VA = ventricular arrhythmia; VT = ventricular tachycardia.

in Table 2. All patients had a normal aortic diameter with no anomalies of the ACs. Of note, all 3 patients who developed AR from the NCC-LCC commissure underwent RF applications both above and below the LCC-RCC junction. On the contrary, none of the patients who underwent mapping and ablation only above the aortic valve developed AR. AR in all patients was found to be mild by evaluating the qualitative and quantitative parameters of echocardiography (the mean value of vena contracta: 0.18 ± 0.04 cm; jet width in the LVOT: 7.1% ± 1.6%; regurgitant fraction: 7.6% ± 1.3%). Table 3 shows the relationship between the location of the ablation site and the incidence of AR after the ablation procedure. While no AR occurred in patients undergoing ablation only above the valve, mild AR occurred in 5 of 21 patients undergoing ablation below the ACs (24%). There was no difference in the QRS morphology of the VA between patients who underwent ablation only above the valve and those who needed ablation below the valve (Supplemental Table 1).

Comparison of pre- and intraprocedural data between patients with and without AR

The mean number of RF applications was 11.0 ± 6.6, with a total duration of RF applications of 12.2 ± 8.5 minutes. The

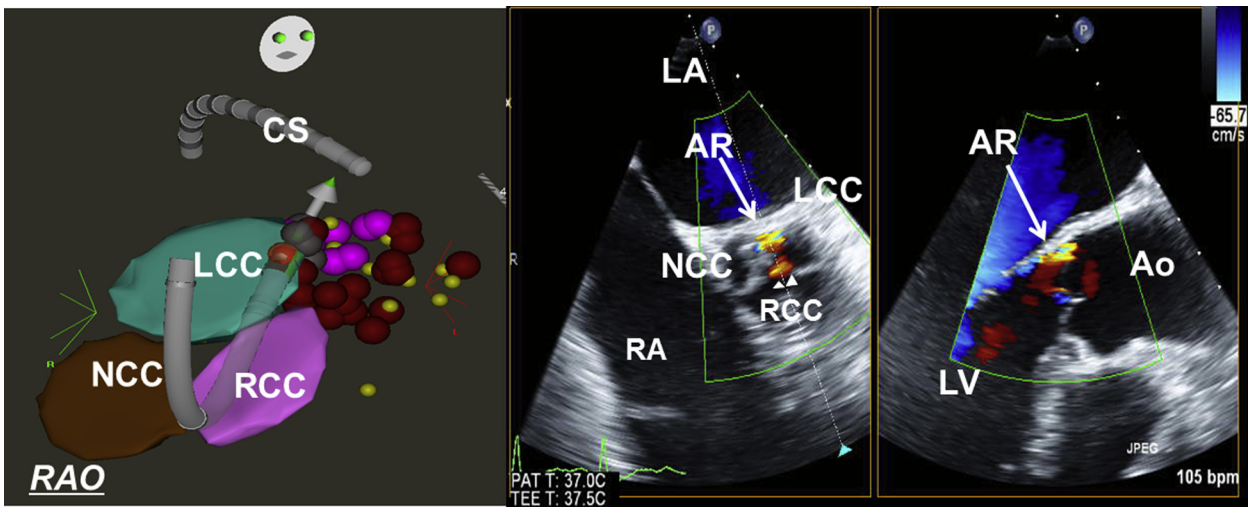

Figure 1 A case of AR at the LCC-NCC commissure. **A:** Patient 1: A 3-dimensional anatomic mapping with an intracardiac echocardiography system shows that the location of radiofrequency applications was above and below the LCC-RCC junction. In addition, the ablation catheter was inserted into the LV through the NCC-RCC commissure. **B:** AR at the center of the aortic leaflets (*arrow head*) and NCC-LCC commissure (*arrow*) was observed on transesophageal echocardiography 1 day after the ablation procedure. Ao = aorta; AR = aortic regurgitation; CS = coronary sinus; LA = left atrium; LCC = left coronary cusp; LV = left ventricle; NCC = noncoronary cusp; RA = right atrium; RAO = right anterior oblique; RCC = right coronary cusp.

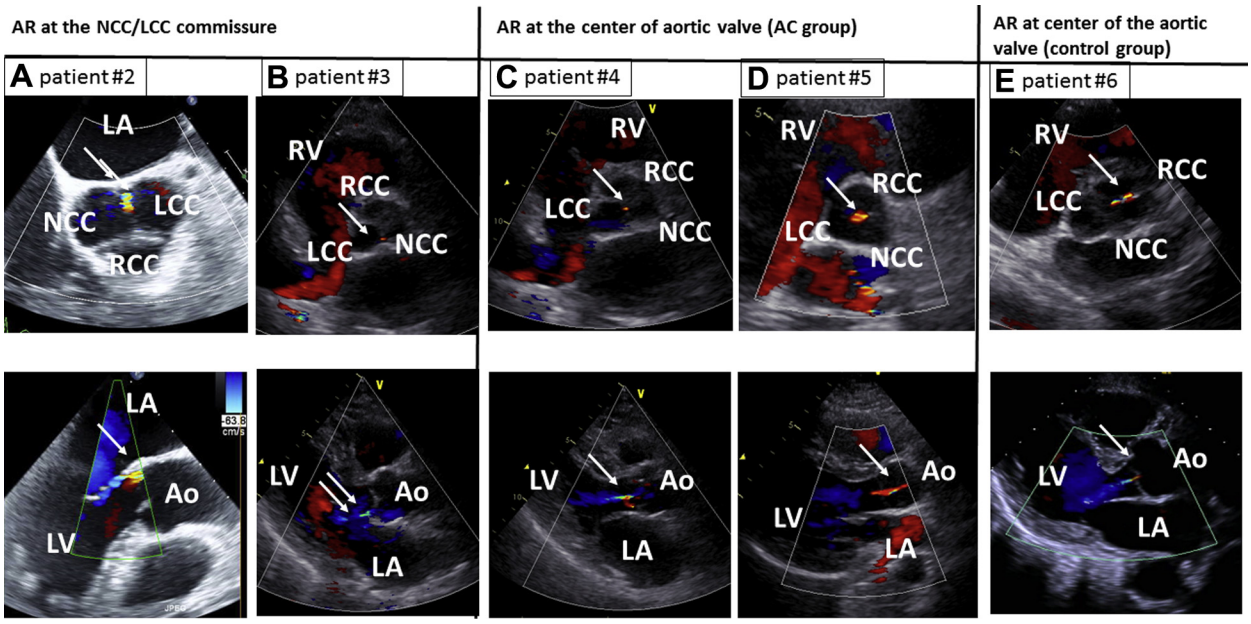

Figure 2 Echocardiography of iatrogenic AR in patients 2–6. **A:** Patient 2: AR at the NCC-LCC commissure observed on transesophageal echocardiography 1 day after the ablation procedure (*arrow*). **B:** Patient 3: AR at the NCC-LCC commissure observed on transthoracic echocardiography 1 day after the ablation procedure (*arrow*). **C–E:** Patient 4 (panel **C**), patient 5 (panel **D**), patient 6 (panel **E**): AR at the center of the aortic leaflets observed on transthoracic echocardiography 1 day after the ablation procedure (*arrow*). Ao = aorta; AR = aortic regurgitation; LA = left atrium; LCC = left coronary cusp; LV = left ventricle; NCC = noncoronary cusp; RCC = right coronary cusp; RV = right ventricle.

mean procedure time was 172 ± 52 minutes. Overall, acute success was obtained in 26 patients (81.2%). There were no significant differences in the baseline clinical and echocardiographic characteristics between patients with and without AR postablation (Table 4). Acute success was obtained in 5 patients with AR (83%) and 31 patients without AR (79%) ($P = .82$). Among AC group patients, the RF ablation data of patients with and without AR postablation are presented in Table 5. There were significant differences in total RF duration (24 ± 14 minutes vs 10 ± 5 minutes; $P < .01$), average RF output (36 ± 4 W vs 32 ± 3 W; $P < .01$), and numbers of RF applications (18 ± 10 times vs 10 ± 5 times; $P = .01$) between patients with and without AR. On the contrary, there were no significant differences in maximum impedance drop and maximum RF output throughout the procedure between patients with and without AR.

One of the patients without AR developed cardiac tamponade at the end of the procedure and required pericardial drainage. Coronary angiography after ablation revealed no coronary artery stenosis.

Follow-up outcomes

Mild AR after ablation remained for over 1 month and 1 year of follow-up on echocardiography in all patients with AR. However, the severity of AR did not change in any of the patients during a mean follow-up period of 16.2 ± 3.6 months. Furthermore, none of the patients with AR developed acute congestive heart failure or had any symptoms concerning AR, such as dyspnea, shortness of breath, and chest pain during follow-up. The serial changes in echocardiographic parameters including LV ejection fraction, LV end-diastolic volume, and aortic diameter of patients with AR are shown in Supplemental Figure 1. Fifteen patients without AR underwent echocardiography at 1 month of follow-up. None of them developed any delayed occurrence of AR. None of the patients without AR had any symptoms or complications during follow-up.

None of the patients who achieved acute success experienced any recurrence of VAs during follow-up. Of the 6 patients who failed to obtain acute success, 4 were successfully managed with antiarrhythmic drugs, and a repeat ablation procedure was planned for 2 patients.

Discussion

Main findings

To our knowledge, this study was the first to systematically investigate the incidence and characteristics of iatrogenic AR after an ablation procedure at the aortic root region. The main findings are summarized as follows:

1. Iatrogenic mild AR after ablation of idiopathic VAs originating from the aortic root occurred with a noticeable prevalence.
2. AR after ablation at the aortic root was associated with extensive ablation both above and below the ACs as well as injury caused by catheter manipulation.
3. The location of AR occurring after aortic root ablation was documented in the following 2 regions: (1) the center of the aortic valve leaflet and (2) the LCC-NCC commissure area.
4. This mild AR remained unchanged during the mid-term follow-up.

Table 2 Ablation and echocardiographic data of patients with AR

Patient no.	Sex	Age (y)	Echocardiography data					Ablation data						Ablation success
			EF (%)	AOD (mm)	AR before RFCA	Location of AR	Jet width in the LVOT (%)	Regurgitant fraction (%)	Catheter	Location of RF	No. of RF applications	Maximum output (W)	Average output (W)	Total RF duration (min)	Maximum impedance drop (Ω)	
1	M	55	58	36	None	Center, LCC-NCC commissure	8.3	8.4	Without CF	Above/below the LR junction	28	45	40.6	46.9	14	Yes
2	M	56	53	40	None	LCC-NCC commissure	9.2	8.2	Without CF	Above/below the LR junction	10	45	38.4	27.6	15	Yes
3	M	81	69	22	None	LCC-NCC commissure	6.8	6.4	With CF	Above/below the LR junction	20	40	33.8	15.1	15	Yes
4	F	86	72	31	None	Center	4.8	5.8	With CF	Above/below the LCC	6	40	24.1	10.5	8	Yes
5	M	16	58	36	None	Center	5.6	8.1	Without CF	Below the LCC	28	40	37.1	22.1	15	No
6	F	64	71	35	None	Center	8.1	9	Without CF	PPM	10	40	27.7	36.6	14	Yes

AOD = aortic diameter; AR = aortic regurgitation; CF = contact force; EF = ejection fraction; F = female; LCC = left coronary cusp; LR = left coronary cusp–right coronary cusp; LVOT = left ventricular outflow tract; M = male; NCC = noncoronary cusp; PPM = posterior papillary muscle; RF = radiofrequency.

Incidence and possible reasons for iatrogenic AR after ablation

This study revealed a noticeable prevalence of iatrogenic AR after ablation, which might be underestimated in clinical practice because of the following reasons: (1) transthoracic echocardiography was not routinely performed just after ablation, and (2) minor valvular regurgitation did not affect the hemodynamics and symptoms. Although the severity of iatrogenic AR in this study was not severe, a previous case report demonstrated aortic valve rupture requiring surgical intervention after ablation at the LCC.[8] We should keep in mind that there is an inherent risk of iatrogenic AR after mapping and ablation of idiopathic VAs originating from the aortic root.

Previous case reports described aortic valve injury after mapping and ablation of an accessory pathway via a retrograde aortic approach.[10,11] Olsson et al[12] demonstrated that the incidence of valvular complications after left-sided RFCA using the retrograde aortic technique was 1.9%. They reported 2 cases of mild AR at the center of the valve occurring after a left lateral accessory pathway ablation procedure. Those previous reports suggested that iatrogenic AR after ablation might be due to the mechanical compression and stretching of the aortic leaflets by the catheter tip and shaft. In the present study, AR at the center of the ACs was also observed in the control group patient in whom an RF application was not delivered at the aortic valve. This observation supports the hypothesis of the mechanical effect associated with the catheter passage through the aortic valve on the occurrence of AR at the center of the valve.

AR at the NCC-LCC commissure was observed in 3 AC group patients, whereas no control group patients developed this type of AR. Kis et al[8] reported a case of an aortic valve rupture on the NCC-LCC commissure requiring a surgical intervention after ablation below the LCC. The ablation catheter is usually inserted into the LV through either the center of the ACs or the NCC-RCC commissure with the catheter bent in a "U shape"' (Figure 1), but not through the NCC-LCC commissure. A 3-dimensional anatomic mapping system using intracardiac echocardiography exhibited that the dorsal side of the catheter shaft may press the NCC-RCC commissure when the ablation target is a VA originating from the LCC and LCC-RCC commissure (Figure 1). In this study, the occurrence of AR was associated with a longer duration and higher power of RF energy delivery. These findings suggested that postablation AR originating from the NCC-LCC commissure might be caused by extensive ablation both above and below the ACs rather than catheter-related mechanical injury. However, the reason why regurgitation flow occurred at the NCC-LCC junction where no RF applications were delivered remains to be investigated.

Follow-up

The severity of iatrogenic mild AR remained unchanged over 1 year of follow-up. All patients have remained asymptomatic during the follow-up period. However, the long-term

Table 3 Ablation site and AR after ablation

Variable	AC (n = 32)	Control (n = 13)	Ablation site							
			LCC (n = 22)			LCC-RCC junction (n = 8)		RCC (n = 2)	Control	
			Above	Below	Above and below	Above	Above and below	Above and below	PPM	APM
No. of patients	32	13	9	2	11	2	6	2	10	3
No. of patients with AR after ablation	5 (6 sites)	1	0	1	1	0	3 (4 sites)	0	1	0
AR at the center of the valve	3	1	0	1	1	0	1	0	1	0
AR at the LCC-NCC commissure	3	0	0	0	0	0	3	0	0	0

AC = aortic cusp; APM = anterior papillary muscle; AR = aortic regurgitation; LCC = left coronary cusp; NCC = noncoronary cusp; PPM = posterior papillary muscle; RCC = right coronary cusp.

risk of AR progression in these patients needs further investigation with serial echocardiographic examinations. Previous studies demonstrated that iatrogenic AR occurring after a left-sided accessory pathway ablation procedure was still observed during follow-up in most patients, whereas it disappeared in a subset of them. According to previous reports, the time course of the symptom onset or LV systolic dysfunction in patients with asymptomatic AR is gradual, with an average event rate of 3%–6% per year.[13–15] However, as the majority of the patients in these reports had rheumatic or arteriosclerotic AR, the time course of ablation-related AR is still unclear.

Study limitations

This study had several limitations. First, the greatest limitation of this study was that it was a single-center retrospective analysis with a small number of patients. Although iatrogenic AR in this study was subclinical, there was a possibility that patients could have developed more severe AR requiring surgical intervention. In contrast, the incidence of iatrogenic AR would have been lower when the targeted arrhythmias were more easily eliminated. Further prospective studies with a higher number of patients are needed to more definitively determine the incidence and severity of iatrogenic AR.

Second, we did not perform cardiac magnetic resonance imaging before and after ablation. Cardiac magnetic resonance imaging could assess the fibrotic changes in the myocardium and characterize the cusp motion, which might have provided significant clues to assess the relationship between ablation lesions and the location of regurgitation flow.

Third, a high number and long duration of ablation applications were required to eliminate the targeted arrhythmias in this study population. This was possibly because of the relatively subepicardial site of origin (MDI [maximum deflection index] 0.55 ± 0.06). In fact, several patients who were referred to our institution had a previously failed ablation.

Table 4 Comparison of the clinical and procedural characteristics between patients with and without AR

Characteristic	AR (n = 6)	Without AR (n = 39)	*P*
Age (y)	60 ± 24.2	59.9 ± 14.3	.99
Male sex	4 (67)	28 (72)	.79
Hypertension	5 (83)	18 (46)	.08
Diabetes	0 (0)	7 (18)	.25
Echocardiography			
LVEF (%)	63.3 ± 8.1	63.5 ± 6.7	.94
AOD (mm)	33.4 ± 6.3	33.8 ± 5.2	.87
Ventricular tachycardia	2 (33.3)	13 (33.3)	>.99
Ablation procedure			
Procedure time (min)	210 ± 91	176 ± 58	.24
Using intracardiac echocardiography	3 (50)	23 (58.9)	.67
Using contact force catheter	2 (33)	19 (48.7)	.48
Catheter insertion into the LV	6 (100)	28 (71.7)	.13
Successful ablation	5 (83.3)	31 (79.4)	.82

Values are presented as mean ± SD or as n (%).

AOD = aortic diameter; AR = aortic regurgitation; LV = left ventricle; LVEF = left ventricular ejection fraction.

Table 5 RF ablation data of patients with and without AR postablation among AC group patients

Variable	AR (n = 5)	Without AR (n = 27)	*P*
Earliest activation time relative to QRS onset during VA (ms)	−27.5 ± 10.7	−27.6 ± 7.1	.98
No. of RF applications	18.4 ± 10.1	9.7 ± 4.8	.01
Total RF duration (min)	24.4 ± 14.1	9.9 ± 4.6	<.01
RF output			
Maximum (W)	42.0 ± 2.7	38.5 ± 2.8	.21
Average (W)	36.6 ± 4.2	32.0 ± 3.2	<.01
Impedance drop			
Maximum (Ω)	15.2 ± 4.7	13.7 ± 4.0	.49
Average (Ω)	9.3 ± 2.3	9.3 ± 2.6	.96

Values are presented as mean ± SD or as n (%).

AR = aortic regurgitation; RF = radiofrequency; VA = ventricular arrhythmia.

Finally, among patients developing AR in this study, intracardiac echocardiography was used during the procedure in only 1 patient. Intracardiac echocardiography allows direct visualization of the ablation catheter tip, ACs, left main coronary ostium, and surrounding anatomy,[16] which may improve the safety of ablation at the aortic root and could have avoided the valvular complications.

Conclusion

This study revealed a noticeable prevalence of iatrogenic mild AR after ablation of idiopathic VAs originating from the aortic root, which was associated with extensive ablation at the perivalvular region as well as catheter-related mechanical factors. Although it did not appear to aggravate the hemodynamic status during the mid-term follow-up, careful monitoring of AR progression should be considered. Further studies are warranted to investigate its adverse effect on the long-term clinical prognosis.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2019.03.010.

References

1. Latchamsetty R, Yokokawa M, Morady F, et al. Multicenter outcomes for catheter ablation of idiopathic premature ventricular complexes. JACC Clin Electrophysiol 2015;1:116–123.
2. Bohnen M, Stevenson WG, Tedrow UB, et al. Incidence and predictors of major complications from contemporary catheter ablation to treat cardiac arrhythmias. Heart Rhythm 2011;8:1661–1666.
3. Peichl P, Wichterle D, Pavlu L, Cihak R, Aldhoon B, Kautzner J. Complications of catheter ablation of ventricular tachycardia: a single center experience. Circ Arrhythm Electrophysiol 2014;7:684–690.
4. Roberts-Thomson KC, Steven D, Seiler J, et al. Coronary artery injury due to catheter ablation in adults. Circulation 2009;120:1465–1473.
5. Kimata A, Igarashi M, Yoshida K, Takeyasu N, Nogami A, Aonuma K. Left anterior descending artery spasm after radiofrequency catheter ablation for ventricular premature contractions originating from the left ventricular outflow tract. Heart Rhythm Case Rep 2015;23:103–106.
6. Dandamudi S, Kim SS, Verma N, Malaisrie SC, Tung R, Knight BP. Left ventricular pseudoaneurysm as a complication of left ventricular summit premature ventricular contraction ablation. Heart Rhythm Case Rep 2017;3:268–271.
7. Beukema RJ, Smit JJ, Adiyaman A, et al. Ablation of focal atrial tachycardia from the non-coronary aortic cusp: case series and review of the literature. Europace 2015;17:953–961.
8. Kis Z, Pal M, Szabo Z, Kardos A. Aortic valve rupture due to radiofrequency ablation of left ventricular outflow tract extrasystole. J Cardiovasc Electrophysiol 2016;27:992.
9. Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two dimensional and Doppler echocardiography. J Am Soc Echocardiogr 2003;16:777–802.
10. Melina G, Codecasa R, Capecchi I, et al. Successful aortic valve repair for severe aortic insufficiency caused by radiofrequency ablation. J Thorac Cardiovasc Surg 2005;130:564–565.
11. Seifert MJ, Morady F, Calkins HG, Langberg JJ. Aortic leaflet perforation during radiofrequency ablation. Pacing Clin Electrophysiol 1991;14:1582–1585.
12. Olsson A, Darpo B, Bergfeldt L, Rosenqvist M. Frequency and long term follow up of valvar insufficiency caused by retrograde aortic radiofrequency catheter ablation procedures. Heart 1999;81:292–296.
13. Bonow RO, Lakatos E, Maron BJ, Epstein SE. Serial long-term assessment of the natural history of asymptomatic patients with chronic aortic regurgitation and normal left ventricular systolic function. Circulation 1991;84:1625–1635.
14. Tornos MP, Olona M, Permanyer-Miralda G, et al. Clinical outcome of severe asymptomatic chronic aortic regurgitation: a long-term prospective follow-up study. Am Heart J 1995;130:333–339.
15. Borer JS, Hochreiter C, Herrold EM, et al. Prediction of indications for valve replacement among asymptomatic or minimally symptomatic patients with chronic aortic regurgitation and normal left ventricular performance. Circulation 1998;97:525–534.
16. Hoffmayer KS, Dewland TA, Hsia HH, et al. Safety of radiofrequency catheter ablation without coronary angiography in aortic cusp ventricular arrhythmias. Heart Rhythm 2014;11:1117–1121.