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REVIEW ARTICLE

Aortic Regurgitation as a Complication of Electrophysiologic Ablation Techniques: A Narrative Review

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Abstract: ***Background***: Radiofrequency catheter ablation is a well-established treatment for several cardiac arrhythmias. Arrhythmias originating from the left side of the heart including ventricular and supraventricular tachycardia and ectopy can be successfully ablated through either transseptal or retrograde aortic approach. Although these techniques have a generally low rate of complications, aortic valve injury is a potential complication of ablation at the left cardiac side that warrants more investigation.

Objective: The purpose of this review is to evaluate the incidence of iatrogenic aortic valve regurgitation and explore the potential mechanisms and risk factors that might contribute to aortic valve injury during radiofrequency ablation. Additionally, the course and progression of aortic regurgitation in the reported cases will be described.

Methods: Authors searched PubMed for articles using the keywords “ablation” AND “aortic insufficiency” OR “aortic valve injury” OR “aortic regurgitation”. Case reports and series as well as retrospective and prospective studies were included, and relevant review articles and editorial comments were used as a supplementary source of data. A total of 19 references were used and a detailed description of patient characteristics, procedural techniques, and incidence, predictors, and fate of aortic regurgitation were reported by 11 clinical studies.

Results: There is a small risk of significant iatrogenic aortic regurgitation after radiofrequency ablation of left-sided cardiac arrhythmias, especially techniques performed *via* a retrograde aortic approach.

Conclusion: Although the risk is not confined to procedures applying direct energy to the aortic cusp region, a more aggressive ablation applied in the vicinity of the valvular complex seems to be associated with a higher risk. Routine post-procedural surveillance should be adopted to detect *de novo* aortic valve injury following radiofrequency ablation techniques.

Keywords: Aortic regurgitation, aortic valve injury, radiofrequency ablation, arrhythmia, aortic insufficiency, tachycardias.

1. INTRODUCTION

1.1. Background

Several ventricular arrhythmias (VA; including tachycardia/ectopy) and Supraventricular Tachycardias (SVT; including atrial fibrillation, atrial flutter, atrial tachycardia, and atrioventricular tachycardias) can be effectively treated with catheter Radiofrequency Ablation (RFA) (Fig. **1**). Despite the relative safety of these procedures, peri-procedural complications such as vascular injury, cardiac tamponade, and valvular injury may occur [1, 2].

Many of the RFA techniques (as detailed below) can affect, either directly or indirectly, the aortic valve. Aortic valve complex is composed of the right Coronary Cusp (RCC), Left Coronary Cusp (LCC), and Non-Coronary Cusp (NCC) with their corresponding sinuses. The valve (with its aforementioned components) occupies the ‘aortic root’, which extends from the nadirs of the aortic valve semilunar cusps at the distal end of the Left Ventricular Outflow Tract (LVOT) to their uppermost points at the sinotubular junction [3].

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The most common site of VA originating from the LVOT is the aortic root followed by the sites underneath the coronary cusps where there is no myocardium at the aorto-mitral continuity (fibrous trigone) [2, 4, 5]. The observation that a non-coronary sinus of Valsalva aneurysm can rupture into the Right Ventricle (RV) supports the assumption that the NCC may be attached to the ventricular myocardium where VAs can arise [6]. Papillary Muscles (PMs) of both left and right ventricles are also potential ventricular arrhythmia origins [7].

Several SVTs can be ablated through the NCC due to its proximity to the left atrium (LA) and the atrial septum [8]. Given the fact that the posterior right side of the RCC and the anterior right side of the NCC are adjacent to the His bundle region [9], mid- to anteroseptal Accessory Pathway (AP) can also be ablated beneath the RCC or at the junction between the RCC and the NCC [8]. Additionally, peri-mitral annular area is a common origin for the left-sided AP and can be ablated through the retrograde aortic valve approach [10].

RFA induces myocardial injury by electrical heating. The histologic appearance of the myocardial lesion is consistent with coagulation necrosis, with contraction bands in the sarcomeres, nuclear pyknosis, and basophilic stippling consistent with intracellular calcium overload [11]. By eight weeks after ablation, the necrotic zone is replaced with fatty tissue, cartilage, and fibrosis and can be surrounded by chronic inflammation [12]. The chronic RFA lesion eventually evolves into a uniform scar that often shows a significant contraction with healing (Fig. **2A** and **B**) [13].

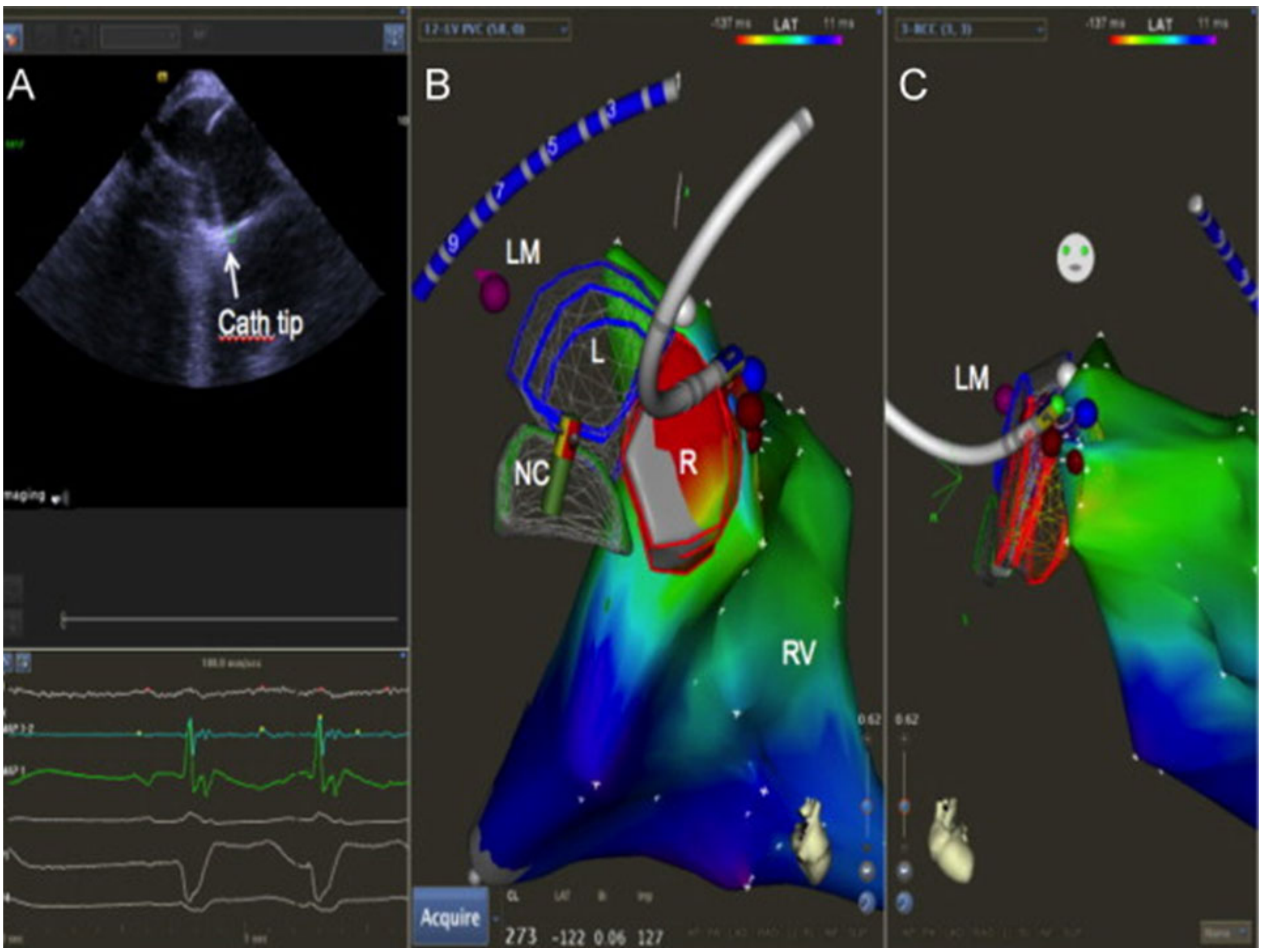

Fig. (1). Imaging during a right cusp premature ventricular contraction ablation.
(**A**) A longitudinal view of the left ventricular outflow tract as seen with phased-array intracardiac echocardiography from the right atrium at the level of the fossa ovalis. The catheter tip (arrow) is visualized at the level of the aortic cusps (sinuses of Valsalva) below the level of the right coronary artery ostium. (**B** and **C**) CartoSound images (B: right lateral view and C: right anterior oblique) showing the location of the right (R), left (L), and noncoronary (NC) cusps. The left main (LM) ostium is labeled with a purple dot. Ablation lesions are labeled within the right cusp in red, with the location of 12/12 matching pace maps in blue. RV = right ventricle. Reproduced with permission from Hoffmayer *et al.* Heart Rhythm 2014;11(7):1117-21. (*A higher resolution / colour version of this figure is available in the electronic copy of the article*).

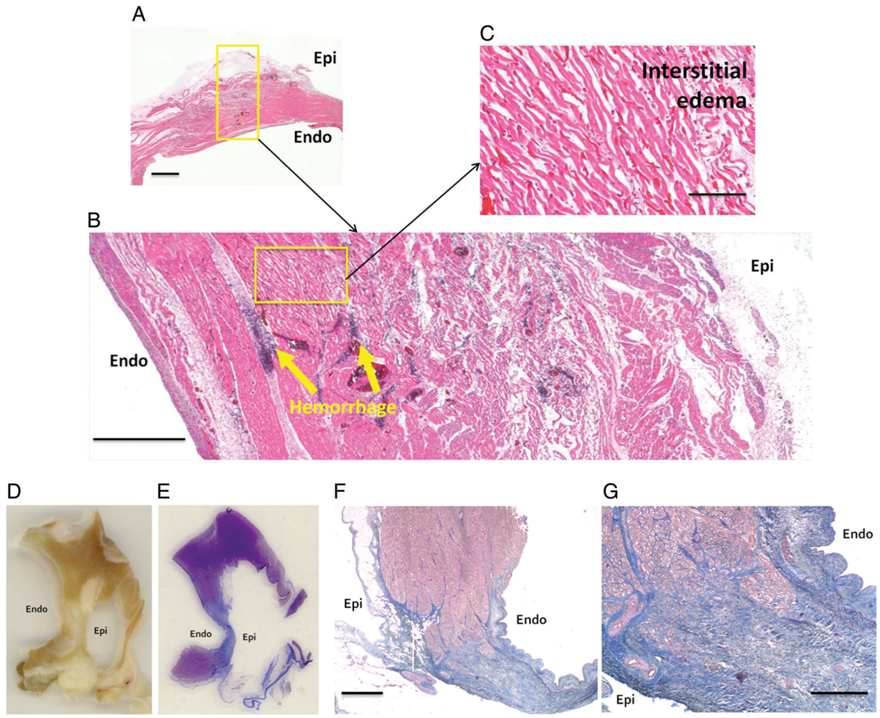

Fig. (2A). Acute and chronic cardiac tissue response to RFA.
Microscopic histology (**a**-**c**) of an acute ablation line demonstrating transmural injury with coagulative necrosis, hemorrhage, and interstitial oedema. Macroscopic (**d** and **e**) and microscopic (**f** and **g**) cross-sections through a chronic ablation line. Microscopic histology demonstrates the transmural replacement of normal atrial wall with fibrous scar tissue. (*A higher resolution / colour version of this figure is available in the electronic copy of the article*).

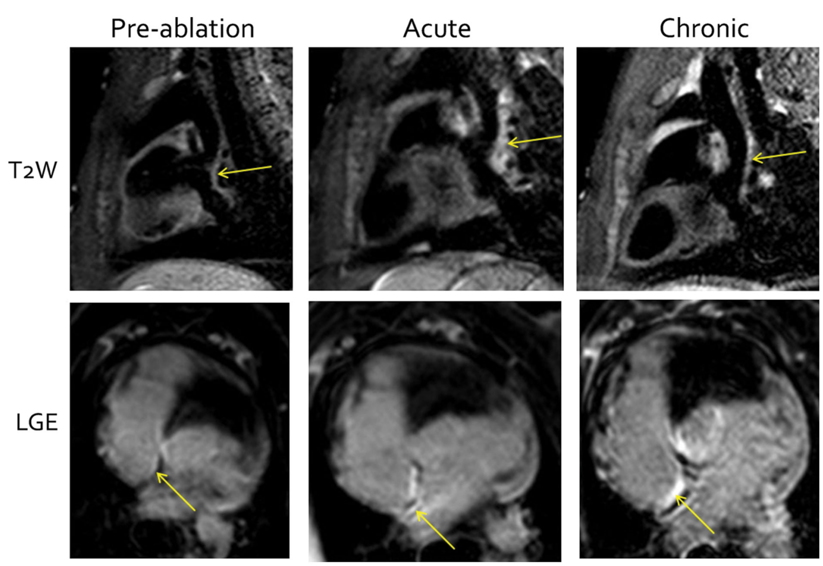

Fig. (2B). Acute and chronic cardiac tissue response to RFA.
Magnetic resonance 3D T2W and LGE scans. While no appreciable pre-ablation T2W or LGE enhancement was seen, T2W and LGE enhancements were seen post-ablation. Chronically, T2W enhancement had reduced, while LGE enhancement remained. (Reproduced from Harrison *et al.* Eur Heart J. 2014;35(22):1486-1495).

Aortic valve injury has been reported as a complication of RFA techniques performed through a retrograde aortic approach or RFA at the aortic root [14]. In the present report, we sought to review the prevalence, the possible mechanisms, and the natural history of aortic valve injury after RFA techniques for cardiac arrhythmias.

2. INCIDENCE OF RFA-RELATED AORTIC REGURGITATION

Although RFA has been reported as a safe and effective technique for the management of many cardiac arrhythmias [7, 8, 15], some studies have reported new aortic regurgitation (AR) as a complication of RFA techniques (Table **1**) [16-18]. The true incidence of RFA-related AR has not been established, possibly due to lack of routine echocardiographic screening post-ablation leading to failure to document mild and asymptomatic AR [16]. Some studies of the safety of RFA reported no cases of iatrogenic AR post-RFA [7, 8, 15], while others reported an incidence of AR complicating retrograde left-sided VA ablation ranging from 2.3% to 13% [16, 18]. Additionally, other studies have investigated the incidence of AR after left side AP ablation procedures and reported an AR rate of 1% to 30% [10, 17, 19]. In younger patients (<18 years) undergoing left-sided AP ablation, relatively higher rates of RFA-related AR (up to 30%) have been reported, albeit the number of patients studied was small [10, 17]. In a study that investigated the safety of VA ablation at the periaortic region in patients with biological/mechanical aortic valve prosthesis, no change in aortic valve function nor worsening of preexisting AR was reported after ablation [20].

Overall, most studies documented an asymptomatic clinical course of iatrogenic AR, which tends to be mild in the majority of cases as documented by echocardiography with an average vena contracta of 1.8 mm, a jet width/LVOT of 7.1% and a regurgitant fraction of 7.6% [16]. On the other hand, Kis *et al.* reported a case of aortic valve rupture involving the NCC-LCC commissure requiring surgical intervention after VA ablation below the LCC [14]. Nevertheless, none of the relevant studies (Table **1**) reported worsening of preexisting AR.

3. MECHANISM AND RISK FACTORS OF RFA-RELATED AR

Several patterns of valvular injury have been reported, such as central transvalvular regurgitant, commissural regurgitation between NCC/LCC related to a loose small coagulum attached to the commissure, and aortic LCC rupture detected on transesophageal echocardiography 24 hours after left side ablation of ventricular ectopy [14, 21]. Likewise, different presumed mechanisms of valvular injury after RFA have been postulated, including 1) repeated passage of catheter tip across the valve, 2) stretching and compression of valvular tissue by vigorous manipulation and/or prolonged placement of the catheter across the valve, and 3) extensive ablation at/adjacent to aortic cusps (especially in children and adolescents with smaller ventricular outflow tract) [8, 10, 16, 18, 19, 21].

Few studies have explored the validity of those presumed mechanisms. In a study by Shinoda *et al.* involving patients who underwent retrograde transaortic RFA of VAs at aortic cusps or Left Ventricular (LV) papillary muscles, significant differences in total RFA duration (24±14.1 *vs.* 9.9 ± 4.6 minutes, p<0.01), average RFA output (36.6 ± 4.2 *vs.* 32.0 ± 3.2W, p<.01), and number of RFA applications (18.4 ± 10.1 *vs.* 9.7 ± 4.8, p=0.01) were noted between patients with and without new AR (16). In this study, two patterns of AR were observed; central and commissural (at the "NCC-LCC" commissure).

The former pattern was observed not only in patients who had aortic cusp ablation, but also in patients who had VA originating from the LV papillary muscles and, thus, had no RFA delivered directly to the aortic cusp region [16]. This observation suggests a mechanical mechanism of AR related to catheter interaction with the aortic valve. Notably, new AR has been documented even after quite short ablations with a low number of aortic passages, possibly due to compression on the leaflet by the ablation catheter [16, 21]. To minimize mechanical injury of the aortic valve caused by catheter passage/compression, some maneuvers have been suggested such as using soft and pliable distal catheter shaft to minimize damage, minimizing the number of aortic valve crossings, allowing the catheter tip to prolapse before crossing the aortic arch and then advancing to aortic root with decreasing deflection control first, rotation of prolapsed catheter tip at the valve until it drops passively without forcing through leaflets, and reposition only after returning to "neutral" position within the LV [17].

Lesh *et al.* investigated younger patients who underwent RFA of left-sided AP around the mitral annulus through a retrograde aortic or transseptal approach [19]. Ablation time for retrograde procedures was longer than transseptal procedures. Factors associated with difficult manipulation through the aortic valve in retrograde aortic procedures included: small ventricles in children, hypertrophic ventricle restricting catheter placement within the LV, aneurysmal aorta preventing torque-transmission to the catheter tip [19]. Because of the higher rate of technical difficulties and hazards with retrograde access in children and in patients with significant aortic valve disease or hypertrophic ventricles, transseptal access may be preferred in those patients [19].

A strategy of intra-cardiac echocardiography (ICE)-guidance can help decide LV access (retrograde across aortic valve *vs.* transseptal) according to aortic valve morphology and function, opting for a transseptal approach when the aortic valve is deemed "susceptible" for injury [20, 21]. Additionally, ICE guidance allows direct visualization of the ablation catheter tip, aortic cusps, and surrounding anatomy. 3D electro-anatomical mapping can also help reduce procedural duration and the number of RFA applications [18], and thus reduce the risk of aortic valve injury. It has also been suggested that cryoablation should be considered if ablation is in the vicinity of the aortic valve or adequate electrophysiological mapping could not be achieved. Alternatively, low energy RFA (10 W, 50°C) can be applied initially till detecting the right site then regular (higher) power and temperature (*e.g.* 50W, 60°C) could be applied [8].

Table 1. Characteristics and main findings of studies of AR complicating RFA techniques for cardiac arrhythmias.

Table 1A. Baseline characteristics.

Authors	Shinoda *et al.*	Edward *et al.*	Styczkiewicz *et al.*	Park *et al.*	Hoffmayer *et al.*	Frias *et al.*	Olsson *et al.*	Lesh *et al.*	Pires *et al.*	Minich *et al.*
Study period	2013-2017	2008-2018	2005 -2017	2009-2010	2011-2014	1992-1998	1998	1990-1992	1995	1990-1991
Patient number	45	149	103	19	35	27	179	106	355	41
Supraventricular arrhythmia	-	-	-	SVT=12 WPW=7	-	WPW=27	WPW=144 AF=18 AT= 1	WPW=106	WPW=214 SVT=159	WPW=41
Ventricular arrhythmia	PVCs = 30 PVCs+VT= 15	PVCs= 95 VT=51 PVCs+ VT= 3	PVCs=99 VT=30	-	PVCs=28 VT=7	-	VT=6	-	-	-
Age (yr)	ACs=61.8±15.1 *vs.* PM=55.1±6.8	61±13	56(34-64)	46.9±21.9	58±13	13.4(4 -18)	43±17	33±0.2	37±21	12(2-12)
Female (n)	13	44	50	7	9	12	61	44	175	-
LVEF % at baseline	62.2±7.3	45±1	58.3±8.5	NR	NR	≤45 in 3 patients	NR	NR	62±10	NR
Baseline AR	0	Mild =4, Moderate=3	NR	NR	NR	0	Mild=5 Moderate=3	0	Mild=2	NR
Baseline AS	0	NR	Moderate=2	NR	NR	0	2	0	NR	NR

Table 1B. Procedural characteristics.

Authors	Shinoda *et al.*	Edward *et al.*	Styczkiewicz *et al.*	Park *et al.*	Hoffmayer *et al.*	Frias *et al.*	Olsson *et al.*	Lesh *et al.*	Pires *et al.*	Minich *et al.*
Access	Retrograde aortic	Retrograde aortic or trans-septal	Retrograde aortic	Retrograde aortic	Retrograde aortic	Retrograde aortic=29, Transseptal=5, Coronary Sinus=1	Retrograde aortic	Retrograde aortic=89, Transseptal=32	Retrograde aortic, Transseptal, or Anterograde TV	Retrograde aortic=13, Anterograde TV=30
Ablation site	ACs=32, PM=13	ACs=84, LV PM=60, RV PM=5	ACs: AMC/LCC=27(26%), LCC=50(48%), LCC/RCC=11 (11%), RCC=9(9%), NCC=6(6%)	ACs: NCC=12, RCC=6, LCC=1	ACs: LCC=17 (49%) RCC=9(26%), LCC & LCC junction=8(23%), RCC & NCC junction=1 (3%)	Left sided AP	Left side AP=144, AV junction=29, LV=6	Left sided AP around MA	AP=214, AVN slow pathway=120, AV junction=39	Around Mitral valve=30, Tricuspid valve=13
Total RFA duration	12.2± 8.5 min	878±696 s	351(191.5-590) s	AT: 6.2±3.1 s, AP: 9.1±4.4 s	NR	NR	58±34 min	RA: 69.2± 10.5 min, TS: 43.4 ± 9.3 min	10-40 s	NR
Number of RFA applications,	11.0 ± 6.6	NR	9(5-15)	AT: 3.1±2.3, AP: 7.0±7.1	NR	NR	10±9	RA: 7.1±0.9, TS: 7.7±1.3	NR	NR
Intraprocedural imaging	ICE+fluoroscopy with aortography	3D-EAM+ ICE	Fluoroscopy = 11% Fluoroscopy+ 3D-EAM = 54% 3D-EAM = 35%	Fluoroscopy + 3D-EAM	ICE +3DEAM =32 ICE+CA=3	Fluoroscopy =26 Fluoroscopy+TEE=1	Fluoroscopy	Fluoroscopy	NR	NR

Table 1C. Aortic regurgitation.

Authors	Shinoda *et al.*	Edward *et al.*	Styczkiewicz *et al.*	Park *et al.*	Hoffmayer *et al.*	Frias *et al.*	Olsson *et al.*	Lesh *et al.*	Pires *et al.*	Minich *et al.*
Screening for new valvular dysfunction	All patients underwent pre and post-ablation TTE	All patients underwent pre and post-ablation TTE	All patients underwent pre and post-ablation TTE	Pre-operative TTE was not specified, post-RFA TTE has been done	NR	All patients underwent pre and post-ablation TTE	All patients underwent pre and post-ablation TTE	All patients underwent pre and post-ablation TTE	All patients underwent pre and post-ablation TTE.	All patients underwent pre and post-ablation TTE
Incidence of new AV injury	New AR=6(13%) 5(15%) in ACs *vs.* 1(7%) in PMs ablation Vena contracta: 0.18±0.04 cm, jet width in the LVOT:7.1±1.6%, regurgitant fraction: 7.6 ± 1.3%	No significant difference between pre and post-RFA in AR severity.	New AR=3(3%)	New AR=0%	New AR=0	New AR=1(4%)	New AR=2(1.9%; 95% CI: 0.1 to 3.7%) Central regurgitant Jet in one and peripheral jet (between NCC/LCC) related to a loose small coagulum in one No worsening of preexisting AR	New AR=1(1%)	New AR=9(2.3%) Retrograde aortic access=1, trans-septal access=8. No worsening of preexisting AR.	New AR=9(30%; 95% CI: 15 to 45%) Mild AR (small, narrow jet) No worsening of preexisting AR
Predictors of new AR	Total RF duration: 24±14 *vs.* 10±5 min, p<0.01; Average RF output: 36±4 *vs.* 32±3 W, p<0.01; Numbers of RF application: 18±10 *vs.* 10±5, p=0.01	NR	NR	NR	NR	NR	NR	NR	Poor correlation between AV injury and the ablation nature; predominantly (89%) in the absence of contact with the aortic valve.	AV injury independent of age, weight and ablation attempts.
Follow up duration	16.2±3.6 months	36±9 months	42.9±38.2 months	19.7±9.8 months	30 days	24 months	33-49 months	19.4±0.6 months	15±6.0 months	NR
Natural history of new AR	No clinical/echocardiographic worsening	No clinical/echocardiographic worsening.	No clinical/echocardiographic worsening.	NR	No evidence of new AR throughout follow up.	NR	At 49th month: no new AR and the attached structure disappeared with AR resolution.	NR	No clinical/echocardiographic worsening.	NR

Abbreviations: AC, aortic cusp; AMC, aorto-mitral continuity; AP, accessory pathway; AR, aortic regurgitation; AS, aortic stenosis; EAM, electro-anatomical mapping; ICE, intracardiac echocardiography; LCC, left coronary cusp; NCC, noncoronary cusp; NR, not reported; PM, papillary muscle; PVC, premature ventricular contraction; RCC, right coronary cusp; RFA, radiofrequency ablation; SVT, supraventricular tachycardia; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography; VT, ventricular tachycardia; WPW, Wolf-Parkinson-White syndrome.
. Data are presented as (Mean±SD) median(IQR).

4. COURSE AND PROGRESSION OF RFA-RELATED AR

With the exception of few reported cases of significant aortic valve injury [12-14], most studies (involving variable follow up periods, ranging from one month to four years) showed that post-RFA AR was mild with no significant change in the AR severity nor clinical deterioration during follow up [16, 18, 21]. Nevertheless, antibiotic prophylaxis against infective endocarditis was performed before invasive procedures have been recommended for six months post-RFA [17]. It should be noted, however, that the follow-up duration of the aforementioned studies is short relative to the young age of patients undergoing RFA for cardiac arrhythmias, especially those undergoing AP ablation. Therefore, longer-term follow-up studies are required before concluding upon the long-term fate of RFA-related AR. In one study, cardiac magnetic resonance imaging could detect post-RFA subtle aortic cusp changes including confirmation of valvular scarring in areas of cusp thickening detected on transthoracic echocardiography [16]. This approach carries significant research as well as clinical potential to deepen our understanding of the incidence, mechanisms, and fate of RFA-related AR.

CONCLUSION

Aortic valve injury and iatrogenic AR can complicate RFA techniques for several cardiac arrhythmias (especially those performed *via* a retrograde aortic approach) and are not confined to procedures applying direct energy to the aortic cusp region. Few predictors could be identified to date, but a more aggressive ablation seems to be associated with a higher risk. Although the risk of aortic valve injury during

RFA techniques is generally small, it should not be ignored when minimally symptomatic patients (especially younger and smaller patients) are considered for RFA techniques.

CONSENT FOR PUBLICATION

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CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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