

Original article

Structural and Functional Inverse Cardiac Remodeling After Cavotricuspid Isthmus Ablation in Patients With Typical Atrial Flutter

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Article history:

Received 15 February 2012

Accepted 30 March 2012

Available online 26 July 2012

Keywords:

Atrial flutter

Cavotricuspid isthmus

Radiofrequency ablation

Structural remodeling

ABSTRACT

Introduction and objectives: The purpose of the present study is to determine the structural and functional cardiac changes that occur in patients at 1-year follow-up after ablation of typical atrial flutter.

Methods: We enrolled 95 consecutive patients referred for cavotricuspid isthmus ablation. Echocardiography was performed at ≤ 6 h post-procedure and 1-year follow-up.

Results: Of 95 patients initially included, 89 completed 1-year follow-up. Hypertensive cardiopathy was the most frequently associated condition (39%); 24% of patients presented low baseline left ventricular systolic dysfunction. We observed a significant reduction in right and left atrial areas, end-diastolic and end-systolic left ventricular diameters, and interventricular septum. We observed substantial improvement in right atrium contraction fraction and left ventricular ejection fraction, and a reduction in pulmonary hypertension. Changes in diastolic dysfunction pattern were observed: 60% of patients progressed from baseline grade III to grade I; at 1-year follow-up, this improvement was found in 81%. We found no structural differences between paroxysmal and persistent atrial flutter at baseline and 1-year follow-up, exception for basal diastolic function.

Conclusions: In patients with typical atrial flutter undergoing cavotricuspid isthmus catheter ablation, we found inverse structural and functional cardiac remodeling at 1-year follow-up with much improved left ventricular ejection fraction, right atrium contraction fraction, and diastolic dysfunction pattern.

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Remodelado cardiaco inverso estructural y funcional en pacientes con aleteo auricular típico sometidos a ablación del istmo cavotricuspideo

RESUMEN

Introducción: y objetivos El propósito de este trabajo es evaluar los cambios cardíacos estructurales y funcionales que se producen tras 1 año de seguimiento de pacientes sometidos a ablación del aleteo auricular típico.

Métodos: Se ha analizado de manera consecutiva a 95 pacientes remitidos para ablación del istmo cavotricuspideo. Se realizó un ecocardiograma en las 6 h posteriores al procedimiento y al cabo de 1 año de seguimiento.

Resultados: Completaron el estudio 89 pacientes. La cardiopatía hipertensiva fue la asociada más frecuentemente (39%). Presentaban disfunción sistólica ventricular izquierda el 24% de los pacientes. Se observó una reducción estadísticamente significativa en el área de la aurícula derecha, el área de la aurícula izquierda, los diámetros telediastólico y telesistólico del ventrículo izquierdo y el septo interventricular. Hubo una mejoría significativa en la fracción de contracción de la aurícula derecha y la fracción de eyección del ventrículo izquierdo, así como en la reducción de hipertensión pulmonar. Se observó un cambio significativo en el patrón de disfunción diastólica, que pasó de grado III (60% basal) a grado I (el 81% en el seguimiento). No se encontraron diferencias estructurales basales ni en el seguimiento entre los pacientes con aleteo auricular paroxístico o persistente, excepto en la función diastólica basal.

Conclusiones: La ablación con catéter del istmo cavotricuspideo en el aleteo auricular típico produjo al cabo de 1 año de seguimiento un remodelado cardiaco inverso estructural y funcional, con mejoría de la fracción de eyección del ventrículo izquierdo, la fracción de contracción de la aurícula derecha y el patrón de disfunción diastólica.

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Palabras clave:

Aleteo auricular

Istmo cavotricuspideo

Ablación por radiofrecuencia

Remodelado estructural

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Abbreviations

AFib: atrial fibrillation
 AFL: atrial flutter
 CTI: cavotricuspid isthmus
 LA: left atrium
 LVEF: left ventricular ejection fraction
 RA: right atrium

INTRODUCTION

Cavotricuspid isthmus (CTI) ablation is a front-line option in the treatment of typical recurrent atrial flutter (AFL), especially in patients with poor clinical tolerance and in those who develop AFL following treatment of atrial fibrillation (AFib) with class I or III drugs.^{1–4} Improvements in symptoms and quality of life have been reported following CTI ablation,^{5–9} but post-ablation structural and functional changes have not been studied.

The present study was designed to determine the structural and functional cardiac changes that occur at 1 year in patients with typical AFL following CTI ablation.

METHODS

Population

We analyzed 95 consecutive patients referred to the cardiac electrophysiology laboratory between 2003 and 2005 who met the following requirements: *a)* age ≥ 18 years; *b)* ≥ 1 AFL episodes documented in 12-lead electrocardiogram (ECG) in the previous 6 months; *c)* a history of isolated or predominant AFL if presenting with concomitant AFib, or AFL after antiarrhythmic treatment with type I or III drugs for AFib prevention, and *d)* electrophysiologic confirmation of CTI-dependent AFL or CTI permeability if the ablation procedure was performed in sinus rhythm, in which case, the clinical episode ECG had to show this was of the common type.

Exclusion criteria were: *a)* non CTI-dependent AFL; *b)* cardiac surgery or interventional cardiac procedure (coronary angioplasty or pacemaker implantation) in the previous 30 days; *c)* implantable cardioverter-defibrillator recipient; *d)* life expectancy < 1 year, and *e)* inability to complete the quality of life questionnaire (Figure).

Definitions

Tachycardia-induced cardiomyopathy: left ventricular (LV) myocardial dysfunction secondary to AFL with high frequency ventricular response and total recovery on achieving normal cardiac rhythm. We defined LV systolic dysfunction as $< 50\%$ left ventricular ejection fraction (LVEF). Patients who did not achieve normal LVEF in the follow-up were not considered to have tachycardia-induced cardiomyopathy.

AFL types were defined by analogy with AFib:

- Persistent: incessant AFL which does not cease spontaneously, continuing over > 1 month and documented in several ECGs.
- Paroxysmal: intermittent AFL with episodes of < 48 h that remit spontaneously.

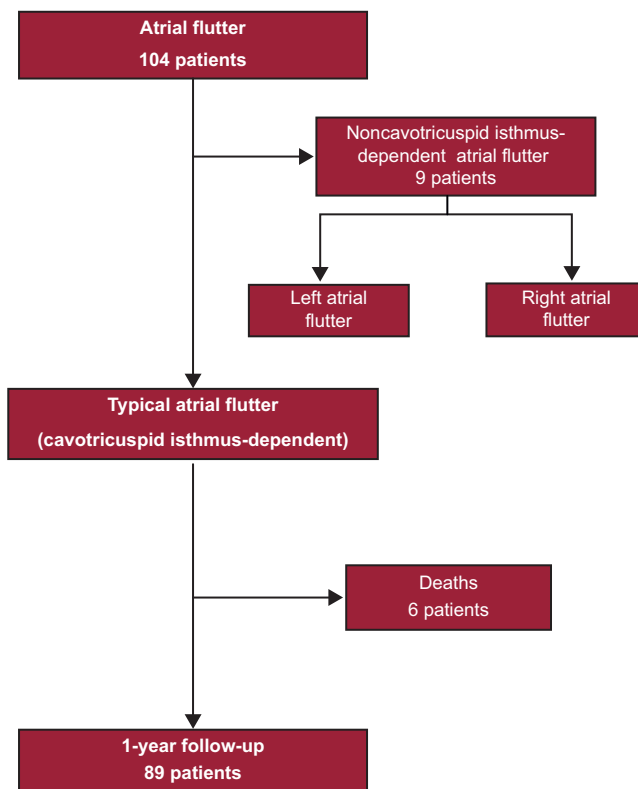


Figure. Flow-chart of the study population.

Ablation Procedure

We used a standard quadripolar catheter (Usci-Bard Inc.) to map the His bundle region, a decapolar catheter (Usci-Bard Inc.) to map the coronary sinus, and a duodecapolar Halo XP catheter (Cordis-Webster Inc.) to map activation of the right atrium (RA) anterolateral wall. Radiofrequency energy was applied for a period of 60 s at each point. CTI-dependency was confirmed by entrainment when the rhythm at the start of the electrophysiology study was AFL or when AFL was induced in the laboratory. If the patient was in sinus rhythm, bidirectional CTI permeability was confirmed prior to ablation. The objective of the procedure was to achieve bidirectional CTI conduction block.^{10,11}

Follow-up

Clinical follow-up was programmed for all patients at 3, 6 and 12 months after ablation. Any visit to either a cardiologist or the emergency department was recorded in the patient's online clinical history. At 6 months post-procedure, a 7-day Holter monitor was used to assess asymptomatic events.

An echocardiographic study with standard equipment (Siemens Sequoia C 256 AG; Munich, Germany) was made at ≤ 6 h after the electrophysiology study and at 1-year follow-up. M-mode and bidimensional mode measurements were made in line with American Society of Echocardiography recommendations.¹² RA and left atrium (LA) areas were calculated by mapping the internal borders of the atrium in the apical 4-chamber plane to obtain maximum atrium size at ventricular end-systole. The same method was used to calculate minimum RA area at ventricular end-diastole and RA contraction fraction (RAcf) using the following formula:

$$\text{RAcf} = (\text{diastolic RA area} - \text{systolic RA area}) / \text{diastolic RA area}$$

The pressure gradient between the right ventricle (RV) and the RA during systole was measured using the simplified Bernoulli equation. Doppler transmitral flow was used to determine diastolic function together with maximum E wave and maximum A wave velocity, E/A ratio, E wave deceleration time (E decT), and isovolumic relaxation time (IVRT). To minimize the respiratory variation of these variables, we took the mean of 5 measurements. Diastolic dysfunction was classified at 3 levels: grade I, IV relaxation alteration (E/A<0.7 and E decT>250 ms); grade II, pseudo-normal (E/A 0.7–1.5 and E decT 150–250 ms), and grade III, restrictive alteration (E/A>1.5 and E decT<150 ms). Tissue Doppler imaging of the posterolateral region of the mitral annulus was used (E'/A' < 1 and E'<8.5 cm/s) to confirm a pseudo-normal pattern.^{13,14}

To quantify the severity of mitral regurgitation (MR) and tricuspid regurgitation (TR), the ratio between maximum regurgitant jet obtained from the color Doppler flow image and atrial area was used: < 15% (grade I); 15%–30% (grade II); 30%–50% (grade III) and > 50% (grade IV).¹⁵

Interobserver variability was 2.3%–4.5% for RA area, LA area, LA diameter, end-diastolic diameter (EDD), end-systolic diameter (ESD), interventricular septum (IVS), posterior wall (PW) and LVEF and was 6%–11.5% for E wave, A wave, E/A ratio, E decT and IVRT.

Intraobserver variability was 0.8%–5.5% for RA area, LA area, LA diameter, EDD, ESD, IVS, PW and LVEF and was 4%–10.4% for E wave, A wave, E/A ratio, E decT and IVRT.

Statistical Analysis

To compare structural variables, we used the Student *t* test and the nonparametric Mann-Whitney test according to whether or not the data followed a normal distribution. The Kolmogorov-Smirnoff test was used to verify whether the data distribution was normal and the Levene test to assess the homogeneity of the variances. The Wilcoxon test for paired samples was used to compare all structural variables, diastolic function and valve alterations between baseline and follow-up.

Ethical Considerations

The study was conducted in accordance with the principles of the Declaration of Helsinki (1975) and was approved by the Clinical Research Ethics Committee of Galicia. All patients gave their informed written consent.

RESULTS

Patient Clinical Characteristics

Baseline patient characteristics are shown in Table 1. Mean age was 64 years; 82% were men. Hypertensive heart disease was the most frequently associated condition (38%). We found LVEF<50% in 25% of patients, whereas 22% showed no structural heart disease. Some 45% had a history of AFib.

Characteristics of the Procedure and Follow-up

The procedure was performed in sinus rhythm in 28 patients (29.5%) and in AFI in 67 (70.5%). Acute procedure success was 100%; bidirectional CTI conduction block was achieved in all patients. No in-hospital deaths occurred. Six patients died during follow-up. The causes of death were lung cancer, severe aortic stenosis, respiratory failure due to severe chronic obstructive pulmonary

Table 1

Baseline Characteristics of the Patients (n=89) who Completed 1-Year Follow-up

Age, years	64.2±10.7
Men, no. (%)	73 (82)
COPD, no. (%)	16 (18)
HT, no. (%)	43 (48.3)
Diabetes mellitus, no. (%)	17 (19.1)
Obesity, no. (%)	22 (24.7)
LVEF < 50%, no. (%)	22 (24.7)
Hypertensive cardiopathy, no. (%)	34 (38.2)
Valvular heart disease, no. (%)	18 (20.2)
Ischemic heart disease, no. (%)	13 (14.6)
Heart failure, no. (%)	14 (15.7)
Dilated cardiomyopathy, no. (%)	16 (18)
After heart surgery, no. (%)	11 (12.4)
Without heart disease, no. (%)	20 (22.5)
AfI type, no. (%)	
Paroxysmal	50 (56.2)
Persistent	39 (43.8)
Ventricular cycle (ms)	664±223
Paroxysmal	640±260
Persistent	685±192
First AfI episode	38 (42.7)
Duration of AfI (months)	18 [4–48]
AfI episodes, no.	2 [1–7]
ECV, no. (%)	22 (24.7)
PCV, no. (%)	14 (15.7)
Previous OAC, no. (%)	53 (59.6)
Subsequent OAC, no. (%)	61 (68.5)
Previous Afib, no. (%)	40 (44.9)
AfI CI or amiodarone, no. (%)	14 (15.7)
Amiodarone	11 (11.6)
Flecainide	3 (3.1)
Tachycardia-induced cardiomyopathy, no. (%)	16 (17.9)

AFib, atrial fibrillation; AfI, atrial flutter; AfI CI or amiodarone, atrial flutter related to the administration of class I or III antiarrhythmic drugs; COPD, chronic obstructive pulmonary disease; ECV, electrical cardioversion; HT, hypertension; LVEF, left ventricular ejection fraction; OAC, oral anticoagulation therapy; PCV, pharmacologic cardioversion.

We present data on the 89 patients who completed follow-up out of the 95 patients who started follow-up.

Quantitative data are expressed as mean±standard deviation or median [p25–p75].

disease, respiratory infection, and sudden out-of-hospital death in 2 patients (1 due to pulmonary thromboembolism).

We recorded 13 cases of typical AfI recurrence (14.6%) at 1-year follow-up. In 12 of these, CTI ablation was performed, and 1 patient underwent electrical cardioversion; 24 patients (25%) showed Afib episodes during follow-up. At 1 year, 82 patients (92%) were in sinus rhythm and 7 (8%) were in Afib.

Echocardiographic Characteristics

The baseline echocardiographic characteristics of the patients are shown in Table 2. A notable finding was an increase in RA and LA areas, together with RAcf depression (0.29). Mean LVEF was 55% and LV diameters were within the normal range. Some 28% of the patients had RV dilatation. Most patients (80%) had baseline diastolic dysfunction and the restrictive form was predominant (58%). Some 58% of patients had TR, most of them with mild

repercussions (72% grade I). Some 20% of patients had baseline pulmonary hypertension (PHT) and 67% had MR, most also with mild repercussions (73% grade I).

The patients' echocardiographic characteristics at 1-year follow-up are shown in Table 2. Statistically significant differences were found for the reduction of RA and LA areas, EDD, ESD and IVS. Right atrium contraction fraction and LVEF improved significantly. Most patients (66%) continued to show diastolic dysfunction during follow-up, which predominantly took the form of relaxation alteration (grade I) (82%). Some 58% of the patients had TR, most (81%) with mild repercussions; 52% had MR, mostly with mild repercussions (85% grade I).

We also found an inverse remodeling in RV with a significant fall in the number of patients with RV dilatation: from 25 at baseline (28%) to 14 (16%) in the follow-up ($P=.008$). The number of patients with PHT fell from 18 (20%) at baseline to 9 (10%) in the follow-up ($P=.03$) (Table 2).

The changes in diastolic function consisted of a significant reduction in maximum E wave velocity, a significant increase in maximum A wave velocity, a significant fall in the E/A ratio, and a significant increase in E decT (Table 2).

Some 80% of patients had altered diastolic function at baseline. In the follow-up, the number of patients with diastolic dysfunction fell significantly (66%), although this difference was not statistically significant ($P=.33$). The most frequent diastolic dysfunction pattern was grade III (60%) at baseline and grade I (81%) in the follow-up. The fall in diastolic dysfunction grade showed a statistically significant improvement ($P<.001$).

We found no differences in the presence of TR in the follow-up vs baseline. However, we did identify a significant reduction in the grade of TR in the follow-up with respect to baseline ($P=.02$) (Table 2).

The presence of MR fell significantly in the follow-up (52%) vs baseline (67%) ($P=.02$). The reduction in follow-up MR grade vs baseline was also significant ($P=.03$) (Table 2).

Patients with persistent AFL had larger baseline RA areas than did those with paroxysmal AFL, although this difference was not statistically significant ($P=.06$). Maximum E wave velocity was higher, maximum A wave velocity was lower, and E decT was shorter in patients with persistent AFL than in those with paroxysmal AFL (Table 3). None of the structural variables showed significant differences between AFL types in the follow-up. Similarly, the baseline differences in diastolic function variables disappeared (Table 3).

Tachycardia-induced Cardiomyopathy

Essentially, tachycardia-induced cardiomyopathy was found in patients with persistent typical AFL and structural heart disease (Table 4). The most frequent structural heart disease was dilated cardiomyopathy in patients with tachycardia-induced cardiomyopathy (31%) and was hypertensive heart disease in those without tachycardia-induced cardiomyopathy (43%). There were no differences in the duration of AFL, the number of episodes, or the presence of previous AFib.

DISCUSSION

The most important finding in this study of patients with typical AFL undergoing CTI ablation is the improvement in structural and functional characteristics in both right and left heart cavities at 1-year follow-up.

Table 2

Structural Variables at Baseline and at 1-Year Follow-up of the Population

Variables (n=89)	Baseline	Follow-up	P
Diastolic RA area, cm ²	23±5.6	19.2±4.9	<.01
Systolic RA area, cm ²	16.9±4.9	12.6±4.1	<.01
RAcf	0.29±0.11	0.45±0.1	<.01
LA area, cm ²	23.9±5.2	22±5.4	.04
LAd, cm	4.57±0.7	4.53±0.6	.36
LVEF, %	55.8±14.2	64.3±8.7	<.01
HR, bpm	70.6 (13.9)	62±11.6	.07
Diastolic function, no. (%)			.33
Normal	16 (18)	23 (25.8)	
Abnormal	71 (79.8)	59 (66.3)	
Unreadable	2 (2.2)	7 (7.9)	
Altered diastolic function, no. (%)			<.01
Grade I	15 (21.1)	48 (81.4)	
Grade II	13 (18.3)	6 (10.2)	
Grade III	43 (60.6)	5 (8.4)	
RV dilatation, no. (%)	25 (28)	14 (16)	<.01
E, m/s	0.90±0.29	0.80±0.23	.01
A, m/s	0.52±0.28	0.81±0.29	<.01
E/A	2.42±1.96	1.1±0.51	<.01
E decT, ms	176±44	207±49.9	<.01
IVRT, ms	90.4±21.9	88±22	.55
IVT, cm	1.10±0.22	1.02±0.21	.02
EDD, cm	5.43±0.65	5.18±0.57	.01
ESD, cm	3.79±0.85	3.33±0.52	<.01
PW, cm	1.01±0.25	0.98±0.18	.17
Tricuspid valve failure, no. (%)	52 (58)	52 (58)	1
Tricuspid valve failure, no. (%)			.02
Grade I	37 (71)	42 (81)	
Grade II	9 (17)	10 (19)	
Grade III	4 (8)	0	
Grade IV	2 (4)	0	
PHT, no. (%)	18 (20)	9 (10)	.03
Mitral valve stenosis, no. (%)	2 (2)	2 (2)	1
Mitral regurgitation, no. (%)	60 (67)	46 (52)	.02
Mitral regurgitation, no. (%)			.03
Grade I	44 (73)	39 (85)	
Grade II	13 (22)	6 (13)	
Grade III	3 (5)	1 (2)	
Aortic stenosis, no. (%)	2 (2)	2 (2)	1
Aortic regurgitation, no. (%)	12 (13)	18 (20)	.73
Aortic regurgitation, no. (%)			.91
Grade I	10 (86)	14 (80)	
Grade III	2 (14)	4 (20)	
Pericardial effusion, no. (%)			.76
Small	3 (3)	2 (2)	

A, maximum A wave velocity; E/A, E/A ratio; E, maximum E wave velocity; E decT, E wave deceleration time; EDD, end-systolic left ventricular diameter; ESD, end-diastolic left ventricular diameter; HR, heart rate; IVRT, isovolumic relaxation time; IVS, interventricular septum; LA, left atrium; LAd, left atrium dimension; LVEF, left ventricular ejection fraction; PHT, pulmonary hypertension; PW, left ventricular posterior wall; RA, right atrium; RV, right ventricle; cf, contraction fraction.

Quantitative data are expressed as mean±standard deviation.

Table 3

Differences in Structural and Functional Variables Between Paroxysmal and Persistent Atrial Flutter

Variables (n=89)	Baseline			Follow-up		
	Paroxysmal (n=50)	Persistent (n=39)	P	Paroxysmal (n=50)	Persistent (n=39)	P
Diastolic RA area, cm ²	21.7±5.9	24.6±4.8	.06	18.3±5.4 ^a	19.4±7	.15
Diastolic RA area, cm ²	16.7±5.9	17.2±3.4	.68	12.3±5.3 ^a	13.6±4	.24
RAcf	0.30±0.11	0.27±0.13	.45	0.47±0.16 ^a	0.47±0.16	.63
LA area, cm ²	23.5±4.9	24±5.7	.77	22.8±7.4 ^b	22.6±6.9	.83
LAd, cm	4.50±0.6	4.6±0.8	.69	4.46±0.6 ^b	4.48±0.6	.87
LVEF, %	55.3±12.1	56.3±16.6	.82	64.9±14.2 ^a	64.1±15.7	.81
HR, bpm	69.8±15.3	73.3±15.5	.42	67.2±13.3 ^b	68.6±13.2	.62
RV dilatation, no. (%)	11 (22)	14 (35.8)	.69	6 (12) ^c	8 (20.5)	.23
PHT, no. (%)	9 (18)	9 (23.1)	.71	6 (12) ^b	3 (7.7)	.69
E, m/s	0.75±0.20	1.07±0.29	<.01 ^d	0.79±0.17	0.8±0.19	.78
A, m/s	0.62±0.29	0.39±0.20	<.01 ^d	0.81±0.26 ^a	0.85±0.32	.62
E/A	1.49±0.75	3.58±2.36	<.01 ^d	1.09±0.8 ^c	0.98±2.4	.90
E decT, ms	193±44	154±36	<.01 ^d	220±57 ^c	204±52	.97
IVRT, ms	89±22	91±21	.75	89±28 ^b	85±31	.06
IVT, cm	1.07±0.19	1.13±0.25	.43	1±0.20 ^b	1.03±0.28	.22
EDD, cm	5.49±0.55	5.35±0.74	.14	5.30±0.38 ^c	5.07±0.70	.18
ESD, cm	3.85±0.68	3.72±1.03	.13	3.45±0.65 ^a	3.26±0.90	.44
PW, cm	0.99±0.21	1.04±0.30	.47	0.96±0.2 ^b	0.97±0.3	.81
Ventricular cycle, ms	641±260	686±192	.87			
Counterclockwise AFI cycle, ms	260±48	244±31	.67			
Clockwise AFI cycle, ms	250±23	245±35	.72			

A, maximum A wave velocity; E/A, E/A ratio; E, maximum E wave velocity; E decT, E wave deceleration time; EDD, left ventricular end-diastolic diameter; ESD, left ventricular end-systolic diameter; HR, heart rate; IVRT, isovolumic relaxation time; IVS, interventricular septum; LA, left atrium; LAd, left atrium dimension; LVEF, left ventricular ejection fraction; PHT, pulmonary hypertension; PW, left ventricular posterior wall; RA, right atrium; RV, right ventricle; cf, contraction fraction.

^a $P<.001$ for the differences between follow-up and baseline data for paroxysmal atrial flutter.

^b Not significant.

^c $P<.05$.

^d $P<.05$ for the differences between persistent and paroxysmal atrial flutter at baseline and at follow-up.

Structural Changes

The improvement in LVEF (a mean 9-point increase) is highly important since it demonstrates that LV contractile function depression is more frequently related to tachyarrhythmias than expected. Moreover, this improvement indicates the presence of a “tachycardia-induced cardiomyopathy-like” component in many patients without LV systolic dysfunction—in whom interrupting the tachycardia facilitates contractile function improvement—as well as in patients with LV systolic dysfunction, who show significant improvement, even though LV systolic function may not achieve normality. Structural changes also affect the RV with an inverse remodeling and a significant reduction in PHT.

In our series, baseline LV structural variables were found to be within the normal range, is consistent with the fact that most patients had mild or moderate hypertensive heart disease. Some 24% of patients had LVEF<50% prior to ablation and therefore our series stands midway between others (19% reported by Luchsinger et al.¹⁶ and 30% by Paydak et al.¹⁷).

This highly substantial improvement in LVEF could be due to a greater prevalence of tachycardia-induced cardiomyopathy in our series (17.9%) vs the 10% described elsewhere.¹⁶ The prevalence of tachycardia-induced cardiomyopathy was greater, even though we excluded patients who showed partial recovery of LVEF but did not show complete recovery at 1-year follow-up. In most patients, neither LVEF nor the presence or absence of LV dilatation were known prior to the onset of the arrhythmia—data that are important in diagnosing tachycardia-induced cardiomyopathy—nor was the presence or absence of previous concomitant

structural heart disease,¹⁸ which constitutes a limitation of the study.

The improvement in LVEF was similar in both AFI types, even though 28/50 patients with paroxysmal AFI (56%) were in sinus rhythm at the time of the procedure. This may be explained by the presence of multiple self-resolving episodes in paroxysmal AFI. In an animal model, alterations in calcium channel activity and sarcoplasmic reticulum calcium transport were observed as early as 24 h after rapid atrial stimulation and persisted up to 4 weeks after stimulation ceased. Calcium alterations correlate with grade VI dysfunction, which may also explain why 31% of patients with tachycardia-induced cardiomyopathy have paroxysmal AFI.¹⁹

However, in our series, persistent AFI was statistically associated with the development of tachycardia-induced cardiomyopathy independently of the ventricular cycle. The development of tachycardia-induced cardiomyopathy is probably multifactorial, depending on the type, frequency and duration of the arrhythmia, but also on age, underlying heart disease, drugs and concomitant diseases.²⁰

We are uncertain as to how long it takes to progress from the onset of the tachyarrhythmia until LV systolic dysfunction occurs, but it can range from weeks to years.²⁰ LV function generally recovers quickly (around 2 weeks) once cardiac rhythm is normal. After 6–8 months, recovery stops and the patient is left with chronic LVEF.^{20,21} A close relation between arrhythmia duration, myocardial damage and LVEF recovery time has been reported, probably because cardiac fibrosis increases as the arrhythmia progresses.²² A 1-year follow-up comparison constitutes a reasonable length of time for LVEF recovery after suppression of the arrhythmia.

Table 4

Differences in Sociodemographic Variables and Variables Related to Atrial Flutter With and Without Associated Tachycardia-induced Cardiomyopathy

Variable	Tachycardia-induced cardiomyopathy (n=16)	Without tachycardia-induced cardiomyopathy (n=73)	P
Age, years	63.4±11	64.5±10.5	.70
Sex, no. (%)			.47
Men	14 (87.5)	58 (79.4)	
Women	2 (12.5)	15 (20.5)	
HT, no. (%)	5 (31.3)	39 (53.4)	.11
Alcohol, no. (%)	3 (18.8)	8 (10.9)	.42
Obesity, no. (%)	3 (18.8)	20 (27.4)	.45
Hypertensive cardiopathy, no. (%)	3 (18.8)	32 (43.8)	.07
Ischemic heart disease, no. (%)	3 (18.8)	10 (13.7)	.38
Dilated cardiomyopathy, no. (%)	5 (31.2)	9 (12.3)	.06
Kidney failure, no. (%)	3 (18.8)	5 (6.8)	.10
Diabetes mellitus, no. (%)	3 (18.8)	15 (20.5)	.89
Type of flutter, no. (%)			.03*
Paroxysmal	5 (31.3)	44 (60.3)	
Persistent	11 (68.7)	29 (39.7)	
Duration of flutter, months	48.7±104.7	36.9±54.1	.54
Episodes of flutter, no.	2.50±3	3.95±3.8	.15
Previous AFib, no. (%)	6 (37.5)	32 (43.8)	.61
Sinus node dysfunction, no. (%)	1 (6.3)	13 (17.8)	.25
AFL CI or amiodarone, no. (%)	3 (18.8)	10 (13.7)	.62
Ventricular cycle, ms	645±227	655±224	.87
Ventricular cycle+persistent AFL, ms	623±202	691±188	.32
Without heart disease, no. (%)	1 (6.3)	19 (24.1)	.11

AFib, atrial fibrillation; AFL, atrial flutter; AFL CI or amiodarone, atrial flutter related to the administration of class I or III antiarrhythmic drugs; HT, hypertension. Quantitative data are expressed as mean±standard deviation.

*P<.05.

Changes in Diastolic Function

We also found an “inverse remodeling” in diastolic function, consisting of a reduction in the grade of dysfunction—from grade III (restrictive), which was predominant at baseline, to grade I (relaxation alteration), in the follow-up. This change is due to the presence of atrial tachycardia-induced cardiomyopathy, which entails the phenomenon of atrial “stunning” after AFL radio-frequency ablation. This finding is consistent with other published studies, demonstrating the reversibility of stunning as early as 3 weeks after ablation.^{23,24} In our series, baseline A wave velocity (0.52 cm/s) was greater than reported elsewhere (up to 28% of patients with absence of A wave)²⁵ probably due to the inclusion of a greater proportion of patients with paroxysmal AFL (56%) and because more than half underwent ablation in sinus rhythm. The significant differences between paroxysmal and persistent AFL in baseline diastolic function variables reflect greater atrial stunning in persistent AFL. However, a component of atrial stunning exists in paroxysmal AFL with significant improvement in RAcf, A wave velocity and E decT not described in the literature because published series have focused on the persistent AFL group and patients with paroxysmal AFL have scarcely been represented.

Differences Between Types of Atrial Flutter

We found no baseline structural differences between patients with paroxysmal AFL and those with persistent AFL. We only observed differences in baseline diastolic function with a predominant grade III pattern in persistent AFL vs a predominant grade II pattern in paroxysmal AFL. These differences disappeared

at 1-year follow-up. These results contradict some published data. Da Costa et al.²⁶ found differences in the structural characteristics of right and left heart cavities measured by echocardiography between paroxysmal and persistent AFL. In persistent AFL, RA area, CTI and LV volumes were greater and LVEF was lower than in paroxysmal AFL. These authors found no differences in the incidence of structural heart disease between the two types of AFL. Cabrera et al.²⁷, using contrast angiography for measurement, found that CTI was longer and RAcf was lower in patients with persistent AFL than in those with paroxysmal AFL. However, RA diameter was similar in both. In our cohort, we found no significant differences in the structural characteristics of right heart cavities between the two forms of AFL, indicating that what determines one or other form is a different atrial remodeling mechanism. The role of the terminal crest is probably decisive both in the onset and in the persistence of AFL.^{28,29} These data are supported by Ohkubo et al.'s³⁰ anatomic observations, which demonstrate differences in the terminal crest between persistent AFL (thick, uniform terminal crest transversal conduction block in all patients) and paroxysmal AFL (thin, non-uniform terminal crest without transversal block in all patients).

Differences have also been described in electric remodeling between patients with AFL and control patients³¹ and between paroxysmal and persistent AFL,²⁸ consistent above all with the finding that the recovery time of the baseline atrial refractory period after arrhythmia ceases is greater in persistent AFL than in paroxysmal AFL. No differences have been detected between AFL types in AFL cycle length or in the atrial refractory period measured immediately after AFL ceases.³² Hence, the real difference seems to lie in electric remodeling recovery time, which is shorter in paroxysmal AFL than in persistent AFL; this difference could

possibly be due to distinct mechanisms. This study has much in common with the data in our cohort, which showed an AFL cycle of similar length for both paroxysmal and persistent AFL, whether counterclockwise or clockwise (Table 3). Baseline and follow-up structural characteristics were similar in both AFL types, and in the AFL cycle. This suggests that anatomic and electrophysiologic differences, and not structural remodeling, determine the form in which AFL presents.

Limitations

Because our study was observational, with no randomized assignment of patients to treatment or placebo groups, we cannot establish a causal relation in our findings. Follow-up echocardiography was performed blind with respect to the baseline study, although the operator knew the purpose of the study, which may have influenced their interpretation of the results.

CONCLUSIONS

CTI catheter ablation of typical AFL produced an inverse structural cardiac remodeling at 1-year follow-up in the atrium and ventricle and functional remodeling with improved LVEF and diastolic dysfunction pattern. No significant structural differences were found—except in the diastolic dysfunction pattern—between paroxysmal and persistent AFL.

CONFLICTS OF INTEREST

None declared.

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