readmissions. Although the relatively small number of patients is a limitation in this regard, some other series seem to confirm this finding.[3]

In conclusion, our series suggest that percutaneous treatment of isolated PVL seems to be a valid alternative to surgery. Nonetheless, surgical correction should always be considered, as it might be the only option with favorable outcomes in patients with PVLs not suitable for percutaneous repair, after failed percutaneous procedures, or for those patients in need of additional surgical interventions. Larger series will be necessary to confirm these findings.

CONFLICTS OF INTEREST

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Long-term Outcome of Patients With Tachycardia-induced Cardiomyopathy After Recovery of Left Ventricular Function

Evolución a largo plazo de pacientes con taquimiocardiopatía tras la recuperación de la función ventricular

To the Editor,

Tachycardia-induced cardiomyopathy (TIC) is a heart disease characterized by ventricular dysfunction and dilatation secondary to sustained tachyarrhythmia that is reversible with heart rate control. It is diagnosed after exclusion of other causes of cardiomyopathy and recovery in left ventricular ejection fraction (LVEF) of at least 15% after heart rate control. The ventricular dysfunction generated by TIC is sometimes extremely serious, leading to heart failure, arrhythmias, and sudden death.[1] TIC is frequently associated with atrial fibrillation. Because TIC is generally considered a benign and reversible condition, it is probably underdiagnosed. However, recent studies indicate that it may cause persistent subclinical damage.[2–4] The true prognosis of the disease is unknown, as well as the mechanisms underlying its reversibility and whether it causes an irreversible subclinical condition.

The present study analyzes the baseline clinical, electrocardiographic, and cardiac imaging characteristics of patients with TIC, their long-term outcomes, and the association of these characteristics with adverse events during follow-up. The study comprises a retrospective analysis of a series of patients diagnosed with TIC and evaluated and followed up in our center between March 2006 and March 2016. Patients with other heart diseases and/or possible triggers were excluded. Clinical treatment was provided according to clinical practice guidelines and at the discretion of the treating physician. LVEF relapses (an LVEF $< 50\%$ or a reduction $\geq 15\%$) during follow-up were analyzed after their complete or partial recovery, as well as their association with prognostic factors. Delayed relapses were those that occurred from the fifth year of follow-up onward. Statistical comparisons between groups were performed using a chi-square test, the Student *t* test, and the Mann-Whitney *U* test; survival analysis was performed using a Cox regression model and Kaplan-Meier estimator. $P < .05$ was considered statistically significant.

In total, 36 patients (23 men) were evaluated with a mean follow-up of 3.2 ± 2.9 years (Table). The most frequent cause of TIC was atrial fibrillation (72%). In 70% of the patients, their symptoms were not directly attributable to their arrhythmia. Eleven LVEF reductions were detected during follow-up (30% of patients; median time from treatment initiation to relapse, 3.08 [0.32-8.03] years) due to arrhythmic relapse or poor control of the original arrhythmia; of these relapses, 5 were delayed (14%). In patients who had a relapse, there were no significant differences in LVEF at treatment initiation or after ventricular function recovery (Figure A). Nonetheless, these patients did show slower LVEF recovery from disease initiation (0.39 [0.21-0.75] vs 1.13 [0.36-4.10] years; $P = .041$) (Figure B) and their clinical follow-up was significantly longer (2.1 ± 2.0 vs 5.6 ± 3.1 years; $P = .007$). In contrast, patients treated with ablation of the triggering arrhythmia were nonsignificantly less likely to have a relapse ($P = .076$), regardless of the type of arrhythmia ablated. There were no significant differences in the relapse-free survival curves between patients with atrial fibrillation and those with other arrhythmias (Figure C). Nonetheless, Cox regression analysis showed that atrial fibrillation multiplied the relapse risk during follow-up by 2.42, although the difference was again not statistically significant (95% confidence interval, 0.29-20.4; $P = .416$). Only 1 death occurred, from noncardiovascular causes.

The present study represents the most extensive series of patients with TIC. Our data show that these patients have a significant future likelihood of relapse. These findings might be related to studies indicating residual subclinical damage in the form of interstitial fibrosis that causes relapses and/or

Table
Characteristics of the Study Population

	Without relapse, n = 25 (69%)	With relapse, n = 11 (31%)	Total, n = 36 (100%)	*P*
Men	16 (64)	7 (63.6)	23 (63.8)	.633
Age at treatment initiation, y	60 ± 1	61 ± 9	61 ± 1	.929
Weight, kg	78.7 ± 16.3	84.0 ± 21.3	80.4 ± 17.8	.425
Hypertension	12 (48)	6 (54)	18 (50)	1
Dyslipidemia	8 (32)	7 (64)	15 (42)	.141
Diabetes mellitus	6 (24)	6 (54)	12 (33)	.073
Smokers	10 (40)	2 (18)	12 (33)	.441
Troponin T, ng/L	22 [11-41]	17 [10-139]	18 [10-139]	.591
NT-proBNP, ng/L	2,703 [324-6,270]	2,135 [606-10,742]	2,548 [324-10,742]	.498
Symptomatic arrhythmia	7 (28)	4 (40)	11 (30.5)	.689
Triggering arrhythmia				
AF	17 (69)	9 (82)	26 (72)	.688
Paroxysmal AF	8 (32)	2 (18)	10 (27)	.688
Permanent AF	9 (36)	7 (64)	16 (44)	.159
Flutter	7 (28)	2 (18)	9 (25)	.690
VE	1 (4)	0	1 (3)	1
HR at diagnosis, bpm	142 ± 30	141 ± 20	142 ± 27	.926
NYHA class at treatment initiation				
I	6 (24)	2 (18)	8 (22)	.402
II	8 (32)	3 (27)	11 (30)	.402
III	9 (36)	3 (27)	12 (33)	.402
IV	2 (8)	3 (27)	5 (14)	.402
QRS width at initiation of treatment, ms	96 ± 21	97 ± 25	97 ± 22	.929
LVEF at treatment initiation, %	32 ± 10	34 ± 8	33 ± 9	.598
LVEDD, mm	55.4 ± 7.8	57.3 ± 4.8	56.0 ± 6.9	.501
MR-LVEDV, mL	208 ± 52	193 ± 48	202 ± 49	.554
MR-RVEDV, mL	161 ± 36	146 ± 59	155 ± 44	.526
Follow-up, y	2.11 ± 2.03	5.60 ± 3.15	3.17 ± 2.87	.007
Tachyarrhythmia ablation	7 (28)	0	7 (19)	.076
AF ablation	3 (12)	0	3 (8)	.538
Flutter ablation	3 (12)	0	3 (8)	.538
VE ablation	1 (4)	0	1 (3)	1
Time to LVEF recovery, y	0.39 [0.21-0.75]	1.13 [0.36-4.10]	0.61 [0.21-1.51]	.041
Time to relapse from treatment initiation, y		3.08 [0.32-8.03]		

AF, atrial fibrillation; LVEDD, left ventricular end-diastolic diameter; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; MR, magnetic resonance; NT-proBNP, N-terminal pro-B type natriuretic peptide; NYHA, New York Heart Association functional class; RVEDV, right ventricular end-diastolic volume; VE, frequent ventricular extrasystole.
Values represent No. (%), mean ± standard deviation, or median [range].

adverse events during follow-up, even sudden death.[1–3] This hypothesis has not been completely confirmed[4] and, in our series, apart from the relapses, there were no sudden deaths or other complications.

Our study failed to identify any clinical, electrocardiographic, or cardiac imaging findings associated with worse prognosis, except a longer time from treatment initiation to LVEF recovery. Recent magnetic resonance studies indicate the ability of interstitial fibrosis on T_1 mapping to predict poor prognosis.[2] The magnetic resonance performed in our patients did not include this technique but the other parameters studied showed no associations with prognosis.

Clinical follow-up was significantly longer in patients with relapse, probably due to a need for more exhaustive monitoring.

In conclusion, patients with TIC can have ventricular function relapses years after their recovery. Our data, although limited by the small sample size and the retrospective nature of the study, indicate that TIC is probably not as benign as thought and requires long-term follow-up with exhaustive heart rate monitoring due to the relapse risk.

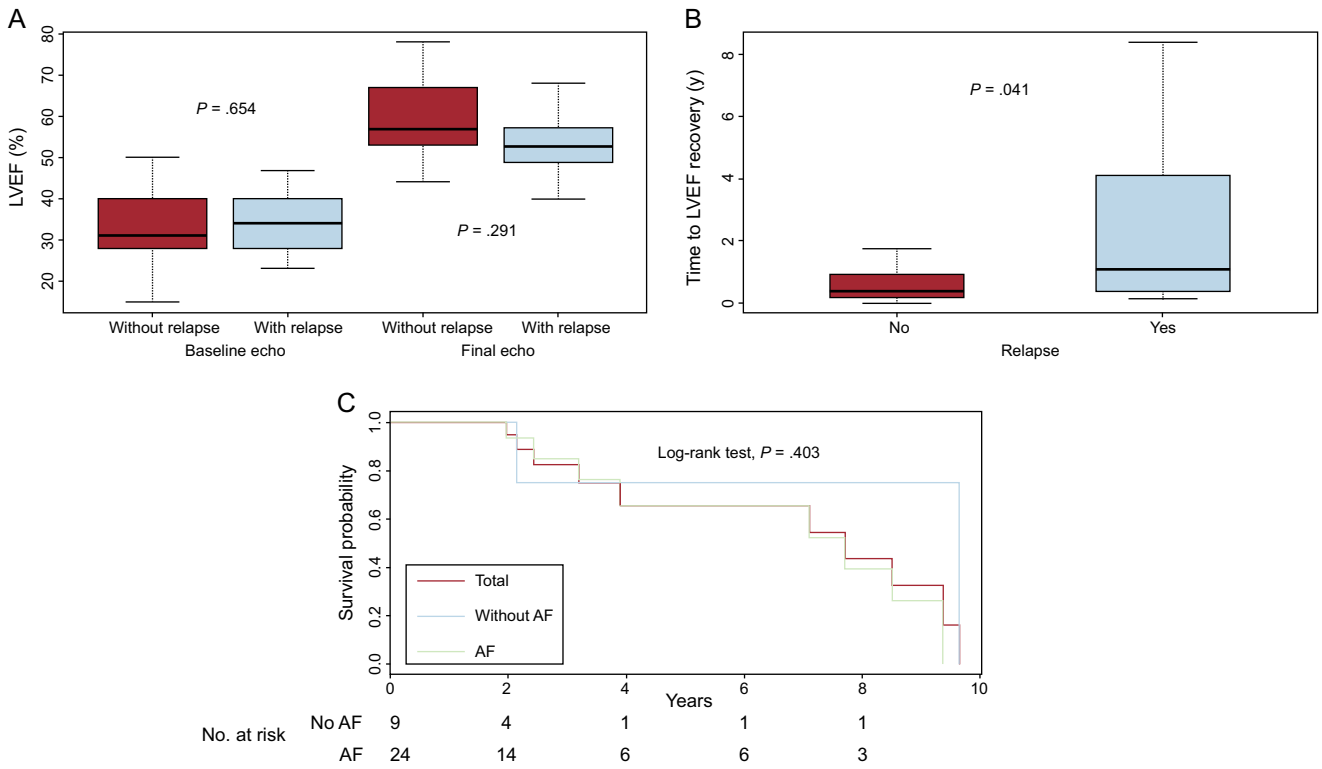

Figure. A: LVEF at initiation of treatment and after its complete recovery according to the presence of LVEF relapse during follow-up. B: Time to recovery of LVEF from initiation of treatment according to the presence of LVEF relapse during follow-up. C: LVEF relapse-free survival during follow-up in the total population (maroon) and in patients with and without AF. Log-rank test between subgroups with and without AF. AF, atrial fibrillation; Echo, echocardiography; LVEF, left ventricular ejection fraction.

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Deep Sedation With Propofol Administered by Electrophysiologists in Atrial Fibrillation Ablation

Sedación profunda basada en propofol y administrada por electrofisiólogos en la ablación de la fibrilación auricular

To the Editor,

Patient sedation is a fundamental aspect of catheter ablation procedures. In prolonged or painful procedures, such as atrial fibrillation (AF) ablation, the patient may receive "conscious sedation", which does not prevent involuntary movements or perception of pain, or general anesthetic.[1] The choice of one or the other depends on patient characteristics and anesthetist availability. "Deep sedation" with propofol has been developed as a third alternative in AF catheter ablation.[1–4] This option can achieve immobility and complete analgesia without the need for intubation or general anesthetic. We describe our experience with this technique.

We prospectively included all patients who underwent AF ablation in our hospital from July 2012 to December 2016. The study was authorized by the local ethics committee. The ablation procedure has previously been described elsewhere.[5] Briefly, via the right femoral vein, we introduced 1 decapolar catheter up to the coronary sinus, and, via a single transseptal puncture, one