

CLINICAL CASE

Tachycardia-induced cardiomyopathy in children and adolescents

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ABSTRACT

Background. Idiopathic dilated cardiomyopathy is the most common form of cardiomyopathy in children; however, potentially reversible causes may occasionally be identified. Among these is a group of patients with symptoms of congestive heart failure and persistent tachycardia representing a form of nonfamilial acquired cardiomyopathy known as tachycardia-induced cardiomyopathy or tachycardiomyopathy. This is a reversible condition with effective treatment of tachycardia. These patients may be misdiagnosed, potentially leading to inappropriate treatment. Diagnosis is often late and always should be suspected in patients with congestive heart failure and unexplained persistent tachycardia.

Case reports. We describe six pediatric patients (mean age 12 ± 4 years old, range 6-16 years). Patients presented with clinical manifestations of congestive heart failure followed by dilated cardiomyopathy. Different mechanisms of persistent tachycardia were documented as the cause and total recovery of ventricular function was achieved after successful treatment of tachycardia by catheter ablation.

Conclusion. The recognition of tachycardia-induced cardiomyopathy in pediatric patients is important. Opportune diagnosis and effective treatment are necessary because tachycardia-induced cardiomyopathy is a reversible cause of heart failure.

Key words: tachycardia-induced cardiomyopathy, idiopathic dilated cardiomyopathy, incessant tachycardia, ectopic atrial tachycardia, fascicular ventricular tachycardia, permanent junctional reciprocating tachycardia.

INTRODUCTION

The majority of the cases of dilated cardiomyopathy (DCM) are defined as idiopathic. However, some may be related to a rapid heart rate. Tachycardia-induced cardiomyopathy (TCM) is defined as a condition characterized by atrial or ventricular myocardial dysfunction as a result of just a sustained and/or irregular high rate. Implicit in this definition is that there is no underlying structural heart disease.¹

Tachycardia-induced cardiomyopathy of supraventricular or ventricular origin is uncommon in children or adolescents. However, timely diagnosis and effective treatment are necessary because TCM is a reversible cause of heart failure after the heart rate is normalized. The ob-

jective of this report was to describe a group of pediatric patients with clinical manifestations of congestive heart failure (CHF) who developed a form of DCM in which the etiological investigation documented a persistent tachyarrhythmia as the primary cause.

CLINICAL CASES

Patient selection

During a 12-year period there were 10 patients identified with clinical symptoms of congestive heart failure and persistent tachycardia. Of these, six were pediatric patients (mean age 12 ± 4 years, range 6 to 16 years). In all patients, any type of congenital heart disease was ruled

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out. The initial diagnosis in four cases was DCM secondary to myocarditis and in the other two cases idiopathic DCM. Patients were referred to the electrophysiology service for evaluation of incessant or persistent tachycardia refractory to different antiarrhythmic drugs (mean 3.1 ± 0.7) despite optimal drug therapy for CHF.

Postablation evaluation and follow-up

Continuous monitoring of the heart rhythm was done for 18 h in the Pediatric Cardiology Intensive Care Unit, then in the Pediatric Cardiac Room for a minimum of 24 h until discharge under treatment with oral effervescent acetylsalicylic acid (300 mg once daily) for 8 weeks. Outpatient follow-up was done monthly with 12-lead electrocardiogram (12-ECG), chest x-ray and echocardiogram. Minimum follow-up was for 1 year before definitive discharge.

Case 1

This is the case of a female child from the city of Puebla. She began at 2 years 6 months of age with progressive dyspnea up to New York Heart Association functional Class III (NYHA). She was evaluated in another hospital where persistent tachycardia and radiological cardiomegaly were detected, establishing a clinical diagnosis of idiopathic DCM. At 4 years of age, impaired left ventricular ejection fraction (LVEF) of 38% was determined by echocardiogram and 24% by radionuclide ventriculography with severe generalized hypokinesia. At 6 years of age the patient was referred for electrophysiological study (EPS) due to persistent tachycardia refractory to the antiarrhythmics propafenone, propranolol and amiodarone and optimal treatment for heart failure (digoxin, furosemide, spironolactone and captopril).

The 12-ECG showed incessant narrow QRS tachycardia characteristically with a long RP' interval–short P'R interval with negative P waves in leads II, III and aVF (Figure 1A). EPS was consistent with a re-entry macrocircuit mediated by a concealed accessory pathway of slow retrograde conduction, also known as a permanent junctional reciprocating tachycardia (PJRT) or *Coumel tachycardia*. Electrophysiological mapping identified the insertion of the accessory pathway in the right posteroseptal region of the tricuspid annulus near the coronary sinus ostium (Figure 1B). A second transcatheter radiofrequency (RF) energy pulse interrupted the accessory pathway conduction restoring sustained sinus rhythm (Figure 1C).

Follow-up with echocardiogram documented total recovery of ventricular function with LVEF 65%.

Case 2

This is the case of a female adolescent from Mexico City. Onset was with asymptomatic frequent palpitations with an evolution time not well defined. At 12 years of age she began to experience fatigue, exercise intolerance and progressive dyspnea up to orthopnea. She was referred to the cardiology service where a diagnosis of CHF, NYHA functional class III was clinically established with third degree radiological cardiomegaly with pulmonary capillary wedge hypertension and echocardiogram reported severe global hypokinesia with a LVEF 32%, suggestive of DCM. Medical treatment included digoxin, furosemide, spironolactone, enalapril, propranolol and amiodarone. Subsequently, treatment was indicated with prednisone and azathioprine for presumptive diagnosis of viral myocarditis and the patient was proposed for possible heart transplant. At 14 years of age due to persistent and refractory tachycardia, the patient was referred to EPS.

The 12-ECG showed regular wide QRS tachycardia with heart rate 160-170 bpm with right bundle branch block pattern qR in V1, R in V2-3, RS transition in V4 and rS in V5-6 and left superior axis deviation with atrioventricular dissociation, establishing the diagnosis of left posterior fascicular ventricular tachycardia (Figure 2A). Intracardiac mapping confirmed a re-entry mechanism with the critical site in the apical region of the left inter-ventricular septum where a second pulse of RF energy at the target site guided by recording of the Purkinje potential interrupted the tachycardia (Figures 2B-2D). During follow-up there was progressive improvement up to normalization of heart function with a LVEF 68%. Figure 2E shows the radiological postablation sequence.

Case 3

This is the case of a 14-year-old female from the state of Chiapas. The patient had a 1-year evolution with rapid and persistent palpitations with clinical manifestations of CHF, NYHA class III, third-degree radiological cardiomegaly and echocardiogram with generalized hypokinesia and LVEF 36%, suggestive of idiopathic DCM. She received treatment with furosemide, digoxin and captopril and progressed with class II symptoms and hospitalizations due to relapses in NYHA class III. She was referred for EPS

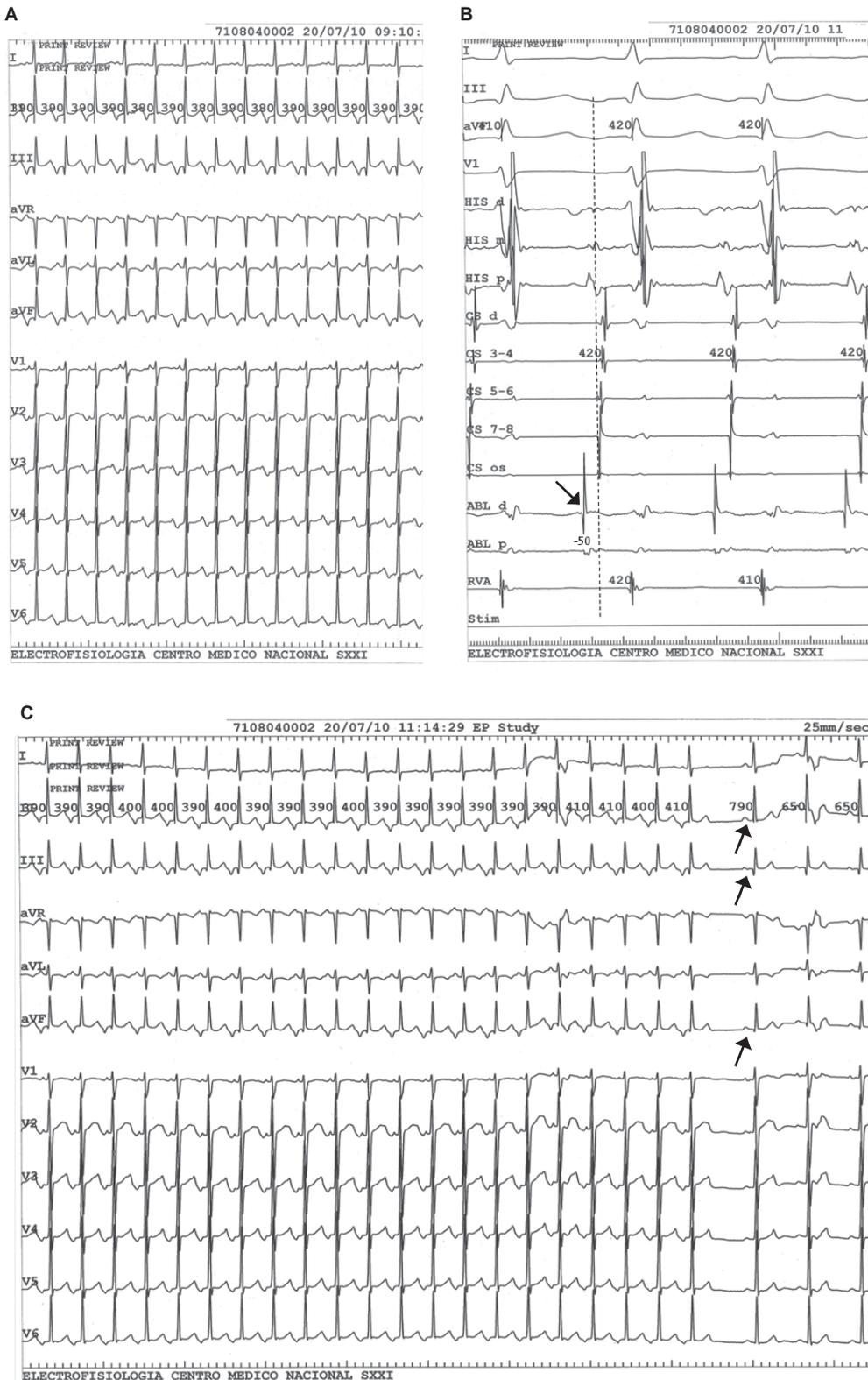


Figure 1.

Permanent junctional reciprocating tachycardia. (A) 12-lead ECG. Negative P' waves in II, III, aVF with RP' interval longer than P'R. (B) Intracardiac recordings, cycle length of TC 420 msec, earlier depolarization in the right posteroseptal region (arrow) at -50 msec of coronary sinus ostium electrogram (EGM) (CSos, dotted line). Leads I, III, aVF and V1, EGM of the distal, mid- and proximal bundle of His (HISd, HISm, HISp), distal coronary sinus to ostium (CSd-CSos), distal and proximal ablation mapping site (ABLd-ABLp), and right ventricular apex (RVA). (C) Interruption of the tachycardia. 12-lead ECG during the RF energy application at the earliest site of the anterior figure with conversion to sinus rhythm. Change of P wave morphology and axis (arrows)

due to incessant tachycardia refractory to the antiarrhythmics disopyramide, propafenone, metoprolol, digoxin and amiodarone. 12-ECG showed narrow, sustained, irregular QRS tachycardia with heart rate 140-220 bpm with bimodal P' waves, negative-positive in II, III, aVF, positive in aVR, negative in I, aVL, positive in V1-V3, with 2:1 AV conduction block (Figure 3A).

EPS confirmed the clinical suspicion of ectopic atrial tachycardia (EAT) due to abnormal automaticity mechanism originating in the left atrium (Figure 3B). The second RF application interrupted the ectopic focus restoring sustained sinus rhythm (Figure 3C). Radiological sequence was obtained for 3 months with echocardiography LVEF normalization to 70% (Figure 3D).

Case 4

This is the case of a 14-year-old male from Mexico City. The patient had a 3-month evolution with palpitations, fatigue, exercise intolerance, and progressive dyspnea up to acute pulmonary edema. Thorax x-ray showed third degree cardiomegaly and echocardiogram with generalized hypokinesia with LVEF 33%. A possible diagnosis of DCM secondary to myocarditis was proposed. The patient was treated with digoxin, furosemide, spironolactone, enalapril and immunosuppressors with prednisone and azathioprine. He continued to progress with NYHA class IV symptoms and was referred for EPS due to persistent tachycardia despite antiarrhythmic treatment with disopyramide, propafenone, digoxin and amiodarone.

12-ECG showed heart rate 160-200 bpm with narrow QRS tachycardia with bimodal positive P' waves in I, aVL, bimodal negative in II, III, aVF, negative in aVR and isodiphasic ± in V1-V3 and positive in V4-V6 with 1:1 AV conduction (Figure 4A). EPS confirmed the clinical suspicion of EAT due to abnormal automatism mechanism originated in the lower right atrium (Figure 4B). The fourth application of RF energy interrupted the ectopic focus restoring sustained sinus rhythm (Figure 4C) with normalization of the LVEF to 68% (Figure 4D).

Case 5

This is the case of a 16-year-old female from the state of Guanajuato. She presented with progressive dyspnea of 2 years evolution in NYHA functional class III-IV. Echocardiographic evaluation demonstrated grade II mitral and tricuspid functional regurgitation and generalized hypoki-

nesis with LVEF 34% and FAc 16%, suggestive of DCM. Her treatment included digoxin, furosemide, spironolactone, enalapril and immunosuppressors prednisone and azathioprine due to suspected probable viral myocarditis. She was referred for EPS due to initial diagnosis of persistent sinus tachycardia. Antiarrhythmic treatment included disopyramide, propafenone, flecainide, verapamil and amiodarone. 12-ECG showed narrow QRS tachycardia with heart rate 160-210 bpm with positive P' wave in I, II, III, aVF, negative in aVR, aVL, V1-V2 and positive in V3-V6 with 1:1 AV conduction (Figure 5A).

EPS confirmed the presence of EAT by abnormal automaticity mechanism originated in the upper right atrium at the level of the crista terminalis (Figure 5B). A second catheter ablation procedure with 11 pulses of RF energy interrupted the ectopic focus restoring sustained sinus rhythm (Figure 5C) with recovery of ventricular function with LVEF 66% and FAc 33% (Figure 5D).

Case 6

This is the case of an 8-year-old female from Mexico City. The patient was evaluated initially due to an isolated syncope episode with findings of supraventricular tachycardia, thorax-x ray cardiomegaly, and NYHA functional class III due to dyspnea and persistent palpitations. Initial diagnosis of DCM was established based on the echocardiogram, with left end-systolic diameter 55 mm, left end-diastolic diameter 52.8 mm, LVEF 10%, grade II mitral and tricuspid functional regurgitation and generalized hypokinesia. Treatment was begun for heart failure with digoxin, spironolactone, propranolol, furosemide and enalapril. Treatment with prednisone was initiated due to suspected viral myocarditis. After 2 years of follow-up she continued in NYHA functional class II-III with incessant tachycardia refractory to antiarrhythmics propafenone, metoprolol and amiodarone with secondary effect of thyroiditis, for which she was referred to EPS.

12-ECG showed incessant tachycardia, heart rate 100-180 bpm with narrow QRS with negative P' waves in I, aVL, positive in II, III, aVF, positive in aVR, positive in V1, biphasic -++ of V2 to V6 and intermittent 1:1 and 2:1 AV conduction (Figure 6A).

EPS confirmed the clinical suspicion of EAT due to abnormal automaticity mechanism originated in the left atrium (Figure 6B). The patient underwent transseptal catheterization technique and 3D CARTO electroanatom-

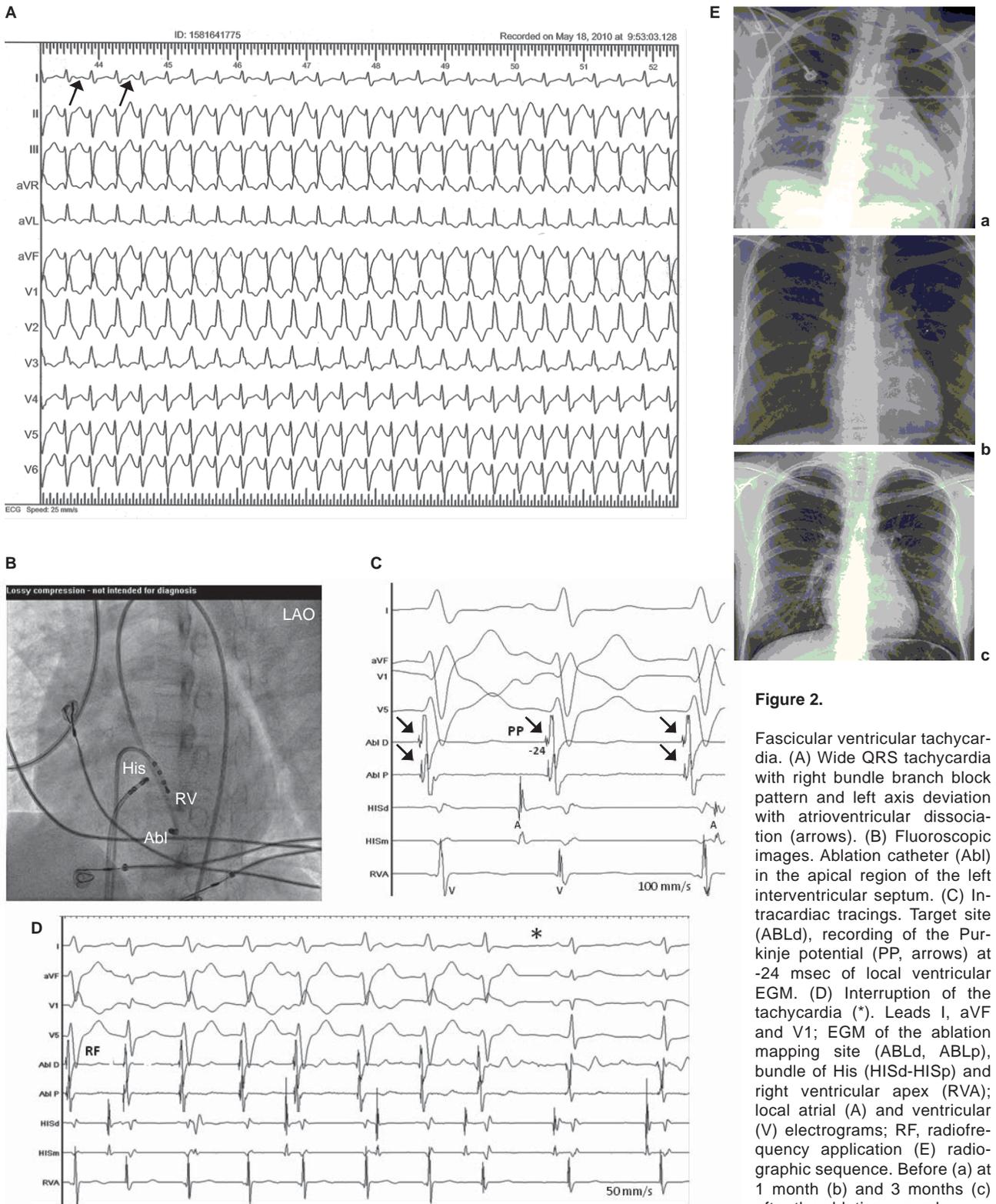


Figure 2.

Fascicular ventricular tachycardia. (A) Wide QRS tachycardia with right bundle branch block pattern and left axis deviation with atrioventricular dissociation (arrows). (B) Fluoroscopic images. Ablation catheter (Abl) in the apical region of the left interventricular septum. (C) Intracardiac tracings. Target site (ABLd), recording of the Purkinje potential (PP, arrows) at -24 msec of local ventricular EGM. (D) Interruption of the tachycardia (*). Leads I, aVF and V1; EGM of the ablation mapping site (ABLd, ABLp), bundle of His (HISd-HISp) and right ventricular apex (RVA); local atrial (A) and ventricular (V) electrograms; RF, radiofrequency application (E) radio-graphic sequence. Before (a) at 1 month (b) and 3 months (c) after the ablation procedure.

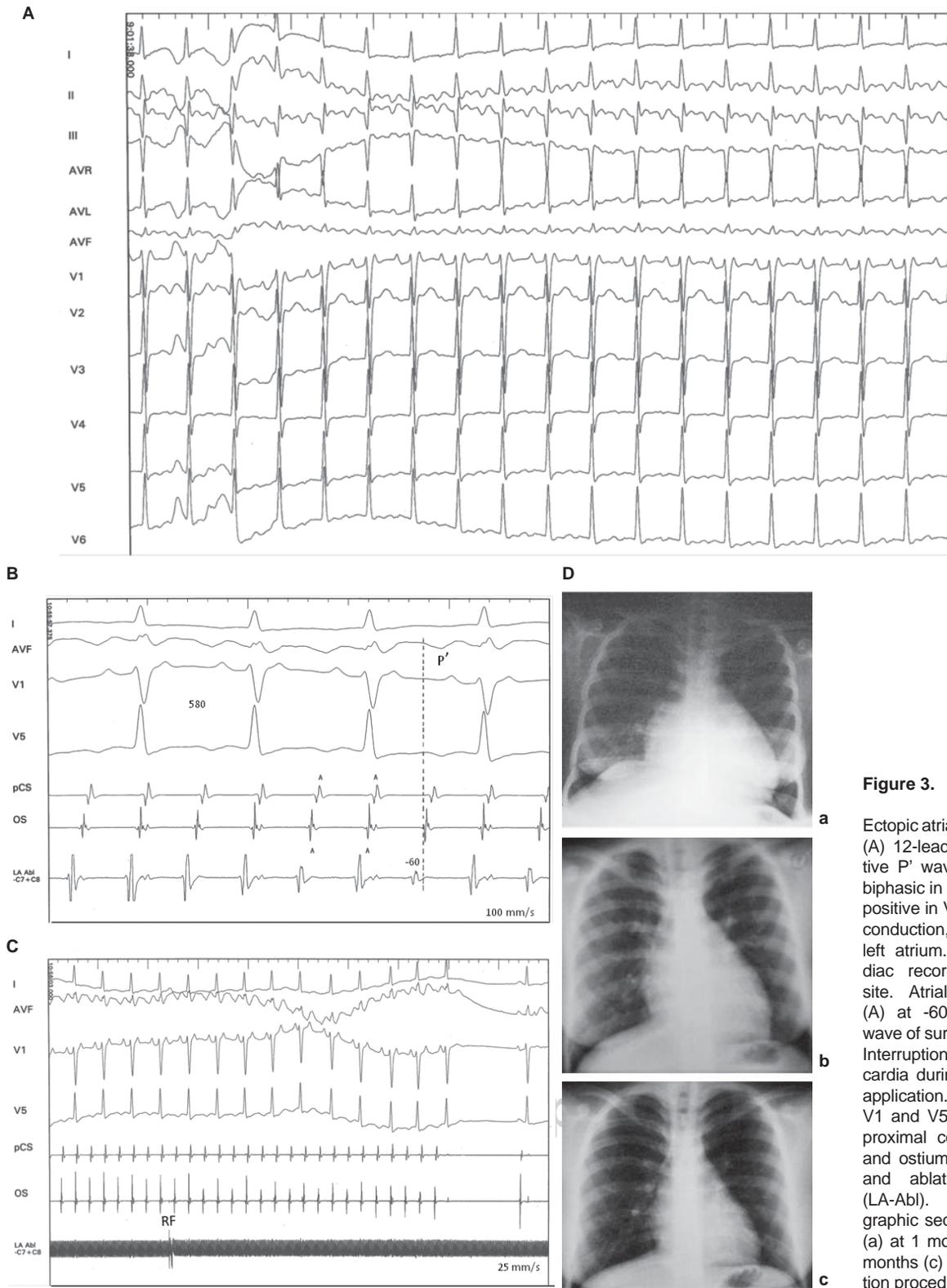


Figure 3.

Ectopic atrial tachycardia. (A) 12-lead ECG. Negative P' waves in I, aVL, biphasic in II, III, aVF and positive in V1 with 2:1 AV conduction, origin in the left atrium. (B) Intracardiac recordings. Target site. Atrial electrogram (A) at -60 msec of P' wave of surface ECG. (C) Interruption of the tachycardia during RF energy application. Leads I, aVF, V1 and V5; EGM of the proximal coronary sinus and ostium (CSp-CSos), and ablation mapping (LA-Abl). (D) Radiographic sequence before (a) at 1 month (b) and 3 months (c) after the ablation procedure.

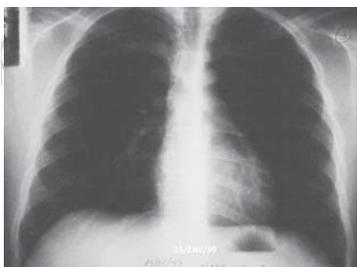
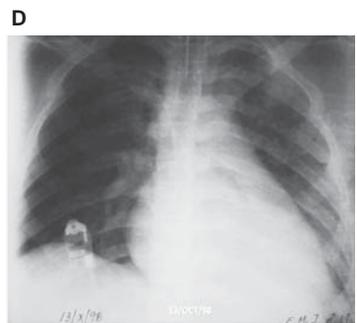
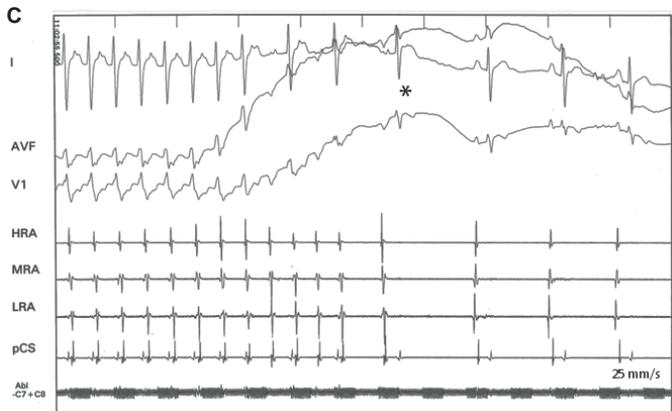
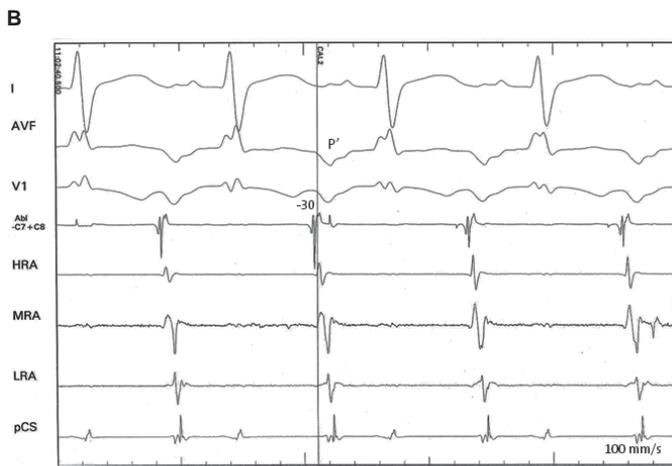
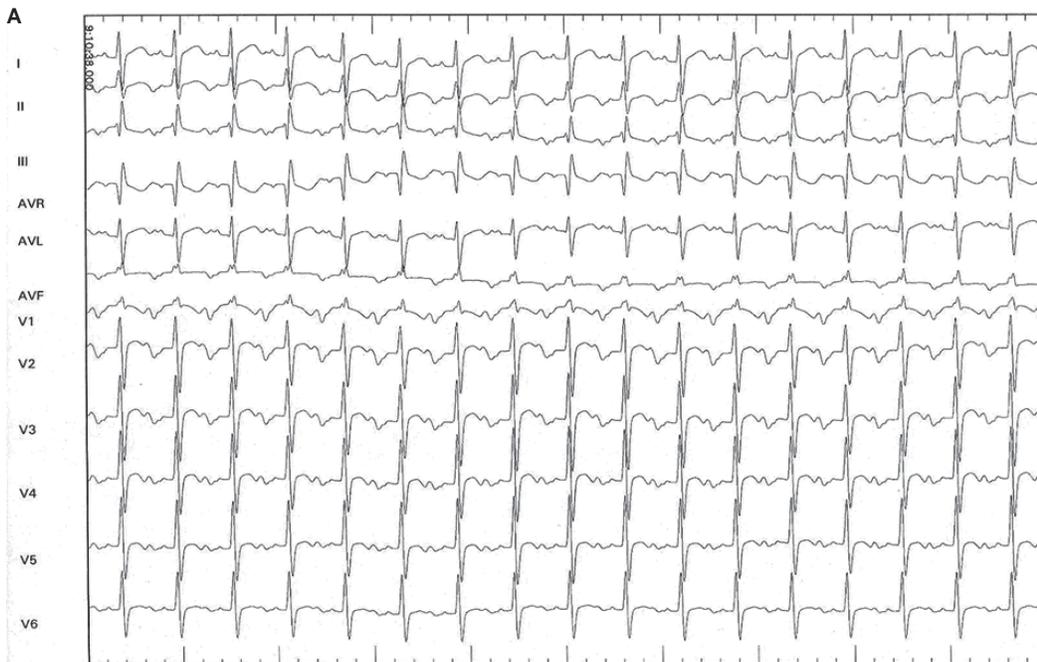
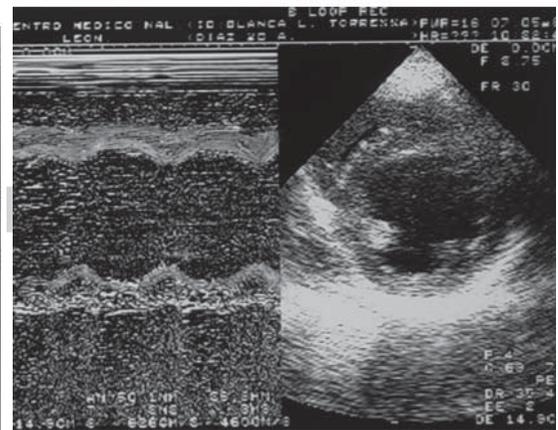
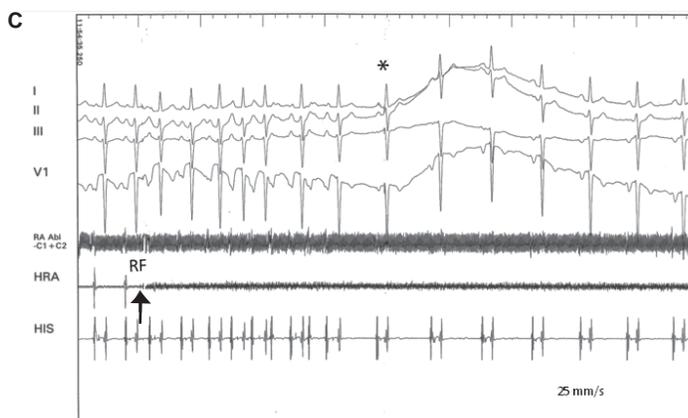
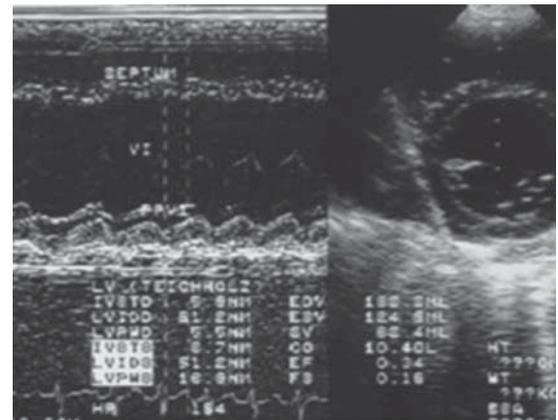
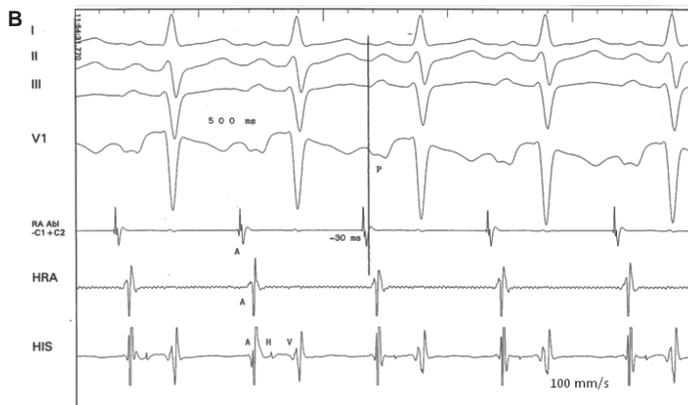
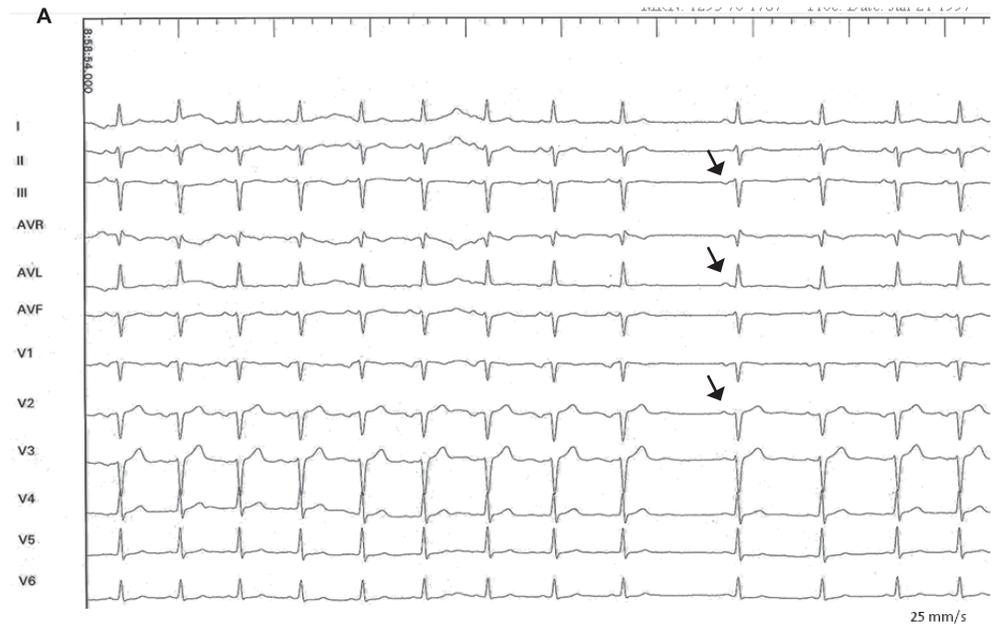


Figure 4.

Ectopic atrial tachycardia. (A) 12-lead ECG. Positive bimodal P' waves in I, aVL, negative in II, III, aVF, and isodiphasic ± in V1 to V3 with 1:1 AV conduction. Origin in the lower right atrium. (B) Intracardiac recordings. Target site. Atrial electrogram at -30 msec of the P' wave of the surface ECG (vertical line), cephalo-caudal activation sequence (HRA, MRA, LRA, pCS). (C) Interruption of the tachycardia (*) during RF energy application. Leads I, aVF and V1; EGM of the high right atrium (HRA), mid (MRA) and low (LRA), proximal coronary sinus (pCS), and the ablation mapping site (Abl). (D) Radiographic sequence before (a), at 1 month (b) and 3 months (c) after the ablation procedure.

Figure 5.

Ectopic atrial tachycardia. (A) 12-lead ECG. Positive P' waves in I, aVL, II, III, aVF and negative in aVR, V1-V2, positive from V3 to V6. Origin in the superior region of the crista terminalis. Beats 10 and 11 are spontaneous in which a normal P waves morphology and axis were observed (arrows) with resumption of the tachycardia. (B) Intracardiac recordings. Target site. Atrial electrogram (A) at -30 msec of the P' wave of the surface ECG (vertical line), cephalo-caudal activation sequence (HRA, HIS). EGM of Bundle of His (H) and ventricle bundle (V). (C) Interruption of the tachycardia (*) during RF energy application. Leads I, II, III and V1; electrograms of the high right atrium (HRA), bundle of His (HIS), and ablation mapping site (RA-Abl). (D) Short-axis M-mode echocardiogram at the level of papillary muscles before ablation. (E) Short-axis M-mode echocardiogram at 3 months postablation.



ic mapping, a second pulse of RF energy using an irrigated catheter *NaviStar ThermoCool* (Biosense, Webster, TX, USA) interrupted the ectopic focus in the left atrial septum restoring sustained sinus rhythm (Figure 6C). Outpatient follow-up was done with normalization of the LVEF to 60% and FAc 33%.

DISCUSSION

Despite the great interest and impact of CM, demographics and underlying causes have been difficult to establish, particularly in the pediatric population. DCM is the most common form and reason for heart transplantation in children.² DCM has been classified as idiopathic in 66% of the cases with age at the time of the diagnosis between 6 and 18 years in 37% of cases.³ One-third of patients had NYHA functional class IV symptoms with a 5-year survival of 71% for the idiopathic variety.³⁻⁵ However, these reports have limited details with respect to the causes, risk and evolution of the specific varieties of CM. Despite these data, knowledge of the causes of DCM continues to be difficult. Only 34% of pediatric patients have an identified cause.⁵ Recently, a new classification includes CM induced by tachycardia and, in it, “tachycardiomyopathy” is classified as a non-familial acquired cause of DCM.^{6,7}

TCM is an infrequent, but potentially treatable, cause of CHF. Reports on its incidence are very limited and, above all, underestimated. In a report of 673 cases of DCM, only one case was attributed to incessant tachycardia.⁸ As the arrhythmias are frequently a complication of CM, these usually goes unnoticed as a primary cause. Clinical evidence and experimental studies have clearly demonstrated that myocardial dysfunction is associated with the occurrence of a rapid heart rate and with an incessant course in the context of diverse types of supra-ventricular and ventricular tachycardia.⁹⁻²³ TCM has been reported in any pediatric age group—from fetus to adolescence.²⁴⁻²⁶

It is notable that EAT commonly has a chronic and incessant course and represents one of the most important reversible cause of left ventricular dysfunction with a picture of DCM, especially in adolescents.¹³ This clinical course was documented in four cases in our series. These cases had in common that tachycardia-related symptoms were always prominent and not taken into account by the clinician. Instead, in the case of patient 5, few symptoms

were related to tachycardia and were recognized when the patient developed symptoms of CHF. The possibility of spontaneous resolution of the EAT is very low. Most cases require antiarrhythmic therapy, but good pharmacological control is achieved only in one-third of cases. According to previous reports, EAT in pediatric patients has an incessant course in 94% of the cases, and up to 28% progress to left ventricular dysfunction when EAT is diagnosed for the first time.^{27,28} Catheter ablation has been a successful and safe procedure in children; therefore, the ablative procedure should be considered early if the EAT is associated with CM.^{13-15,29-31}

On the other hand, the main form of clinical presentation of AV reciprocating tachycardia mediated by an accessory pathway is of paroxysmal type, although there are isolated reports of AV reciprocating tachycardia with an incessant course, notably in the type referred to as PJRT or Coumel tachycardia.^{32,33} It is frequently refractory to antiarrhythmic treatment and is associated with TCM in up one-third of cases.³⁴⁻³⁶ This variety of tachycardia is uncommon and was confirmed in the first case of this series with a rapid recovery of ventricular function with successful catheter ablation.

The second case was associated with ventricular tachycardia (VT). Idiopathic fascicular VT represented an extraordinary situation because it is infrequent in childhood and when present generally has a favorable prognosis.^{37,38} Of the 37 cases published by Ohe et al., 25% were younger than 15 years of age and notably, only one case had a severe clinical course refractory to drugs.³⁹ There are only three reports in the international literature of unique cases with TCM induced by idiopathic fascicular VT, a clinical course similar to our case with normalization of the left ventricular function after successful treatment with catheter ablation.^{22,40,41}

There are no definitive answers on the precise mechanism of TCM in clinical situations.^{42,43} However; several experimental studies have helped us to understand the pathophysiology of TCM. These studies have shown that rapid and sustained stimulation with a pacemaker in the atrium and/or ventricle produces a progressive increase of ventricular volumes, cardiac output and ejection fraction showed a progressive decline after 3 to 5 weeks of pacemaker stimulation.^{44,45}

Histologically, the modifications are characterized by a disorder of the extracellular matrix due to a significant loss

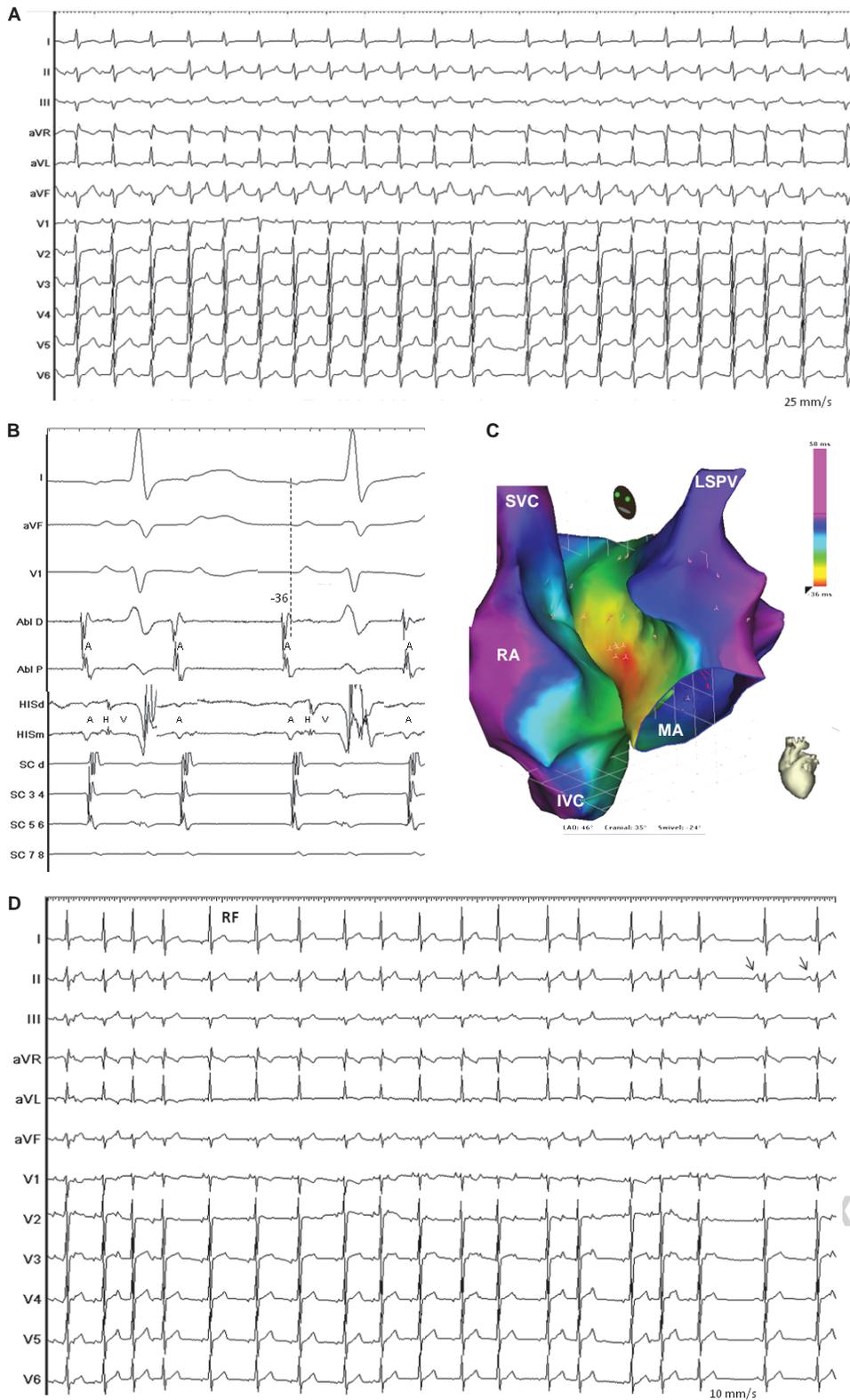


Figure 6.

Ectopic atrial tachycardia. (A) 12-lead ECG. Negative P' waves in I, aVL, positive in II, III, aVF, positive in V1, variable AV conduction. Origin in the left atrium. Typical "hot-cold" behavior of the atrial rate. (B) Intracardiac recordings. Target site. Atrial electrogram (A) at -36 msec of the P' wave of the surface ECG (dotted line), 2:1 AV conduction; EGM of local atrium (A), bundle of His (H), ventricle (V), ablation mapping site (ABLd-ABLp), and distal to proximal coronary sinus (SCd-SC7-8). (C) Electroanatomic 3-D map of local activation time. Left anterior oblique view 45° caudal 35°. Activation time codified in color with earliest activation site in the lower part of the left inter-atrial septum (red color, target site) with progressive radial distribution (yellow-green-blue-purple). RA, right atrium; SVC, superior vena cava; IVC, inferior vena cava; MA, mitral annulus; LSPV, left superior pulmonary vein; LAA, left atrial appendage. (D) Interruption of tachycardia (arrows) during RF energy application

Table 1. Clinical characteristics of the patients

Case	Age/Gender	CF NYHA	Time of evolution (months)	CF (bpm)	Tachy- cardia	AA
	(years) M/F					
1	6/F	III	54	150-166	AV PJRT	3
2	14/F	III	24	160-170	TVF	3
3	14/F	IV	12	90-180	EAT	2
4	14/M	IV	3	160-200	EAT	4
5	16/F	IV	24	125-150	EAT	4
6	8/F	III	24	100-180	EAT	3
Average (mean \pm SD)	12 \pm 4 years		23.5 \pm 17.2 months			3.1 \pm 0.75

M, male; F, female; FC NYHA, functional class New York Heart Association; CF, cardiac frequency; bpm, beats per minute; AV PJRT, permanent junctional reciprocating tachycardia (Coumel); TVF, ventricular fascicular tachycardia; EAT, ectopic atrial tachycardia; AA, antiarrhythmics.

Table 2. Echocardiogram data before and after ablative treatment

Case	Before	% LVEF	
		1 month	3 month
1	38	63	65
2	32	65	68
3	36	70	---
4	33	60	68
5	34	55	66
6	10	60	60

LVEF, left ventricular ejection fraction.

of myocyte density, cellular elongation, myofibrillar disarray, sarcomeric loss, hypertrophy and apoptosis.^{42,43,46,47} The responsible mechanisms probably involve a decrease and altered utilization of the high-energy substrates that determine myocardial remodeling, alteration of the cellular structure, decrease of the ATPase activity with increase of calcium at the level of the sarcolemma and mitochondrial alteration.^{1,48,49}

Reversibility of CM after effective treatment of tachycardia is an important characteristic and has been studied in experimental models. The pattern and recovery time that follow curative treatment of incessant tachycardia in children are not well documented and are derived from clinical observation.⁵⁰⁻⁵⁴

De Giovanni et al. evaluated the pattern of left ventricular recovery secondary to incessant tachycardia after radiofrequency catheter ablation in a group of infants and children. Age at the time of ablation was 2 months to 12.5

years (mean 4.1 years). They concluded that the time of recovery was related principally with the duration of the tachycardia and was significantly shorter in infants.¹⁹

As in previous reports,⁵⁵ the six patients presented here demonstrated an onset of symptom improvement and LVEF normalization at the end of the first month after successful treatment. Accordingly, diagnosis of TCM requires demonstration of improvement in left ventricular function after effective treatment of tachycardia. With the exception of one case, the patients in our series had a longer evolution with symptoms of palpitations and CHF of 3 months to 4.5 years (mean 23.5 \pm 17.2 months). In no case was a clinical picture of TCM suspected, and there was an interval from 3 months to 2 years from the onset of symptoms of CHF until the time of referral for EPS.

It is recognized that diagnosis of TCM is not simple. Unfortunately, there are no diagnostic guidelines to identify a TCM during the first visit with the physician.⁵⁶⁻⁵⁸ Although arrhythmias have shown to be a contributing factor in hospital admissions due to heart failure, it would be expected that the percentage of patients with a tachyarrhythmia as the primary cause of CM would be considerably low. Therefore, TCM requires a high index of suspicion. The clinician should aggressively look for this condition when left ventricular dysfunction accompanies a persistent tachycardia refractory to treatment. A review of the cases reported here indicates that they were misdiagnosed. And this was a factor in the decision to undertake an inappropriate treatment plan that included immunosuppressive therapy due to suspected myocarditis or that the patients were included in a cardiac transplant program due to suspected idiopathic DCM.

In conclusion, recognition of TCM in the pediatric age is very important because offering appropriate treatment leads to recovery of ventricular function. TCM can be one of the most common and unrecognized cause of potentially curable CHF and needs to be taken into consideration in the differential diagnosis of idiopathic DCM. These patients may be misdiagnosed, leading to potentially inappropriate treatment. Therefore, children and adolescents who present with heart failure and persistent tachycardia should be evaluated to rule out a primary cause to allow appropriate initial treatment for this reversible cause of heart failure. TCM is a diagnosis that should not be confused with other conditions.

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