Diagnostic Approach and Treatment Strategy in Tachycardia-induced Cardiomyopathy

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ABSTRACT

Background: Due to the absence of differential guidelines for heart failure with tachyarrhythmia, it is difficult to diagnose tachycardia-induced cardiomyopathy (TIC) at the initial visit. Furthermore, clinical outcomes of rate versus rhythm control in TIC are unclear.

Hypothesis: Because the etiology of TIC is different from dynamic cardiomyoplasty (DCMP), differential parameters may be present.

Methods: We assessed 21 patients with TIC (15 men; mean age, 50 ± 14 years) and 21 control patients with idiopathic DCMP. We assessed clinical courses, echocardiographic parameters, as well as outcomes by treatment.

Results: In the TIC group, the related tachyarrhythmias were atrial fibrillation (n = 12), atrial flutter (n = 5), atrial tachycardia (n = 3) and paroxysmal supraventricular tachycardia (n = 1). After treatment, all patients became asymptomatic and the ejection fraction (EF) improvement (ΔEF $\geqslant$ 15%) was observed in all patients (left ventricular ejection fraction [LVEF], $30 \pm 11\%_{initial}$ versus $58 \pm 6\%_{last}$). In the idiopathic DCMP group, no patient showed EF improvement (EF increase $\leqslant$ 5%), and 4 patients (19%) underwent heart transplantation. Left ventricle (LV) mass indices, volumes adjusted by BSA, and dimensions were smaller in the TIC group than in the idiopathic DCMP group. Of those, LV end-diastolic dimension was the only independent predictor of TIC in multiple regression analysis (odds ratio [OR] 0.742 per 1 mm, 95% confidence ratio [CI] 0.618 to 0.891, p = 0.001). The Association of University Cardiologists (AUC) was 0.908 on receiver-operating characteristic (ROC) curve analysis and LV end-diastolic dimension $\leqslant$ 61 mm could predict TIC with a sensitivity of 100% and a specificity of 71.4%. After restoration of sinus rhythm (n = 8), one experienced recurrent TIC after discontinuation of amiodarone. After control of heart rate (n = 13), one experienced recurrent TIC due to poor control of heart rate (log-rank test, p = 0.808). There were no differences in the echocardiographic parameters between the 2 groups before and after treatment except for the larger initial LV volumes in the rhythm control.

Conclusions: In patients presented as heart failure with tachyarrhythmia, initial echocardiographic parameters, especially LV end-diastolic dimension, help to differentiate TIC from idiopathic DCMP. Rate control was as effective as rhythm control for EF improvement and prognosis.

Key words: dynamic cardiomyoplasty, tachycardia-induced cardiomyopathy, echocardiography, left ventricle end-diastolic dimension, clinical outcomes, rate control, rhythm control

Introduction

Tachycardia-induced cardiomyopathy (TIC) is a rare reversible disease entity of dilated cardiomyopathy described first by Gossage et al. in 1913.[1] Tachycardia-induced cardiomyopathy can be defined as a condition of atrial or ventricular systolic and diastolic dysfunctions induced by increased atrial or ventricular rates, in the absence of prior history of structural heart disease.[2] Tachycardia-induced cardiomyopathy is highly dependent on the heart rate, and that higher heart rates induce TIC earlier.[3–5] Adequate control of heart rate[6,7] or restoration of sinus rhythm[8,9] is necessary for improving cardiac function and symptoms.

There are no differential diagnostic guidelines for identifying TIC in patients who present with decreased left ventricle (LV) ejection fraction (EF) and tachyarrhythmia at the initial visit. Furthermore, it is unclear whether improved clinical outcomes result from rate or rhythm control of TIC. We performed this study to develop a diagnostic approach for identification of TIC at the initial visit, and to compare the clinical outcomes after heart rate control versus restoration of sinus rhythm.

Methods

Study Population and Design

The TIC group consisted of 21 patients with heart failure (left ventricle ejection fraction [LVEF] $\leqslant$ 45%) and tachyarrhythmia who visited our department complaining of symptoms of heart failure. All showed remarkable improvement in LVEF (EF increase $\geqslant$ 15%) after treatment. None of these patients had evidence of ischemic heart disease or

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other structural heart disease on the history, physical examination, laboratory data, echocardiography, stress test or coronary angiography. Eight patients underwent coronary angiography (3 patients) or thallium single photon emission computed tomography (SPECT) (5 patients), and there were no evidences of significant coronary artery disease. Other patients were confirmed without stress test by complete recovery of their global hypokinesia after treatment for heart failure. All patients' symptoms and LVEFs were normalized within 3 months after rhythm or rate control of tachyarrhythmia during follow-up period. As a control group, we analyzed the data from 21 age-, sex-, and EF-matched patients with idiopathic dilated cardiomyopathy (DCMP) group diagnosed during the same period. All had concomitant atrial fibrillation and no evidence of ischemic heart disease or other structural heart disease like TIC group. Their symptoms and LVEF were not improved after rate control of atrial fibrillation contrary to TIC group.

Data Collection

We retrospectively collected data: (i) age, sex, duration of symptoms before the diagnosis of heart failure, (ii) symptoms at the initial and all follow-up visits, (iii) TIC treatment modality, (iv) occurrence of recurrent tachycardia or TIC, (v) long-term clinical outcomes by treatment modality, and (vi) echocardiography and electrocardiogram results at the initial and all follow-up visits. Transthoracic echocardiography was performed to measure LV end-systolic volume indicies (ESVI) and end-diastolic volume indicies (EDVI) which were standardized by body surface area (BSA) using modified Simpson's method, and LVEF. The LV end-diastolic and end-systolic internal dimensions were measured and LV mass was estimated from LV linear dimensions using M-mode echocardiography.[10] We also defined critical cases as initial LVEF $\leqslant$ 30% and marked dyspnea, and collected the data of critical cases separately.

Statistical Analysis

Data were expressed as means $\pm$ SD for continuous variables and frequency (%) for categorical variables. A Wilcoxon signed-ranks test was used to compare echocardiographic parameters before and after rate or rhythm control. Log-rank test was used for comparisons of clinical outcomes for the rate and rhythm control groups. The Mann-Whitney test was used for comparisons between the study and control groups. Exact-distribution test was used because of small sample size. Logistic regression models were applied to study the independent association of variables with the diagnosis. Receiver-operating characteristics (ROC) analysis was performed to evaluate the optimal cut-off value for LV diastolic dimension. The SPSS version 12 system for Windows (SPSS, Inc., Chicago, Ill., USA) was used for statistical analysis. All p-values were 2-sided, and a p-value <0.05 was considered statistically significant.

Results

Demographic Characteristics

The TIC group consisted of 15 men and 6 women, mean age of 50 $\pm$ 14 years (range, 21–73 years) and mean LVEF of 30 $\pm$ 11% (range, 10–45%) at the initial visit (Table 1). Twelve patients had severe dyspnea (New York Heart Association [NYHA] functional class III or IV) and 9 patients had mild dyspnea (NYHA II). The duration of dyspnea or palpitation before diagnosis ranged from 3 days to 6 years. Dyspnea was resolved after control of tachyarrhythmia in all patients, but the time necessary for the symptom to improve was diverse. Although the heart rates were widely variable, all heart rates were more than 100 beats per min. In addition, their heart rates were within 100 beats per min after controls.

As shown in Table 2, the prevalence of hypertension and diabetes mellitus (DM) did not differ between the 2 groups. In spite of meticulous treatment for heart failure like TIC group, all patients of DCMP group showed no EF improvement (EF increase $\leqslant$ 5%) and 4 patients (19%) had to undergo heart transplantation due to disease progression.

Usefulness of Initial Echocardiographic Parameters for Diagnosis of Tachycardia-induced Cardiomyopathy

The LV mass indices, volume indices and dimensions were smaller in the TIC group than in the idiopathic DCMP group (Table 2). The LV volume indices and dimensions differed significantly between the 2 groups ($p<0.001$).

In univariate analysis on echocardiographic parameters, predictors of TIC were LV end-systolic volume index (ESVI) (odds ratio [OR] 0.943 per 1 mL/m^2, 95% confidence interval [CI] 0.907 to 0.980, $p = 0.003$), LV end-diastolic volume index (EDVI) (OR 0.944 per 1 mL/m^2, 95% CI 0.911 to 0.978, $p = 0.001$), LV end-systolic dimension (OR 0.797 per 1 mm, 95% CI 0.694 to 0.915, $p = 0.001$) and LV end-diastolic dimension (OR 0.742 per 1 mm, 95% CI 0.618 to 0.891, $p = 0.001$). In stepwise multiple logistic regression, LV end-diastolic dimension was the only independent predictor of TIC (OR 0.742 per 1 mm, 95% CI 0.618 to 0.891, $p = 0.001$). The ROC curve analysis was performed to check the predictive value of LV diastolic dimension. The AUC was 0.908 and LV end-diastolic dimension $\leqslant$ 61 mm could predict TIC with a sensitivity of 100% and a specificity of 71.4% (Figure 1). In critical cases (LVEF $\leqslant$ 30%: TIC group, $n = 12$ versus DCMP group, $n = 11$), the AUC was 0.913 and LV diastolic dimension $\leqslant$ 66 mm could predict TIC with a sensitivity of 100% and a specificity of 83.4%.

Causes of Tachycardia and Clinical Courses

The arrhythmic causes of TIC were atrial fibrillation in 12 patients, atrial flutter in 5, atrial tachycardia in 3, and paroxysmal supraventricular tachycardia (PSVT) in 1 (Table 1). Patients were followed up for a mean of 35 ± 32 months. Tachycardia-induced cardiomyopathy was treated by rate control (digoxin, beta blocker, or calcium channel blocker) or rhythm control (antiarrhythmic drug, radiofrequency

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TABLE 1: Clinical characteristics of patients with TIC

Cause	Age, year	Sex	Symptom duration before Dx	Initial HR (bpm)	Initial EF (%)	Last EF (%)	Treatment method*	Rhythm change	NSR restoration method†	Recurrent TIC	Follow-up, month
pAF/WPW	67	M	3 days	85–160	21	62	Rhythm	pAF	—	No	17
pAF	35	M	3 days	80–140	10	46	Both	pAF	—	No	16
pAF	48	M	3 mos	65–115	28	60	Rate	pAF	—	No	13
cAF	51	M	2 mos	110–175	44	59	Rate	cAF	—	No	12
cAF	59	F	4 mos	80–150	18	46	Both	NSR	RFCA	No	5
cAF	70	F	2 mos	95–190	40	59	Rate	cAF	—	No	9
cAF	66	M	2 mos	80–150	27	58	Rate	cAF	—	No	9
cAF	43	M	1 yr	90–150	29	55	Rate	cAF	—	Yes	117
cAF	69	F	6 yrs	80–170	40	67	Rate	cAF	—	No	33
cAF	51	M	26 mos	85–140	38	57	Rate	cAF	—	No	86
cAF	49	M	3 yrs	150–200	23	62	Both	cAF	—	No	18
cAF	50	M	1 mos	90–190	45	63	Both	NSR	DCCV	No	20
AFL	44	M	10 days	75–150	22	64	Both	NSR	AAD	No	19
AFL	46	M	1 yr	90–150	22	57	Rhythm	NSR	RFCA	No	31
AFL	52	M	2 mos	85–145	45	62	Rhythm	pAF	—	No	24
AFL	42	M	3 days	100–140	38	63	Rhythm	AFL	—	No	31
AFL	55	M	7 yrs	60–140	41	58	Rhythm	AFL	—	No	35
AT	27	F	6 yrs	150	20	50	Both	NSR	AAD	No	90
AT	73	F	2 mos	135	34	46	Rhythm	NSR	AAD	No	85
AT	21	F	5 mos	100–150	24	60	Both	NSR†	AAD	Yes	38
PSVT	36	M	3 mos	160	26	66	Rhythm	NSR	RFCA	No	19
Mean	50 ± 14				30 ± 11	58 ± 6					35 ± 32

Abbreviations: AAD = antiarrhythmic drug; AFL = atrial flutter; AT = atrial tachycardia; Cause = responsible arrhythmia; cAF = chronic atrial fibrillation; DCCV = direct current cardioversion; Dx = diagnosis; EF = ejection fraction; HR = heart rate; initial EF = LVEF before rate or rhythm control; last EF = LVEF checked last after rate or rhythm control; LVEF = left ventricle ejection fraction; NSR = normal sinus rhythm; pAF = paroxysmal atrial fibrillation; PSVT = paroxysmal supraventricular tachycardia; RFCA = radiofrequency catheter ablation; TIC = tachycardia-induced cardiomyopathy; WPW = WPW syndrome. * Rate, rate control methods include the use of digoxin, beta blocker or calcium channel blocker. Rhythm, rhythm control methods include the use of an antiarrhythmic agent, cardioversion or ablation. † This patient achieved the NSR conversion using amiodarone, which was maintained for 6 months, but TIC recurred after discontinuation of the drug. RFCA was tried without success.

continued

TABLE 2: Baseline characteristics and initial echocardiographic parameters in the TIC and DCMP groups

	TIC group (n = 21)	DCMP group (n = 21)	p
Baseline characteristics			
Men	15 (71%)	15 (71%)	1.000
Age at initial visit (years)	50 ± 14	50 ± 14	0.970
Duration of follow-up (mos)	38 ± 31	55 ± 28	0.002
Hypertension	10 (48%)	6 (29%)	0.209
DM	5 (24%)	2 (10%)	0.220
IHD	0 (0%)	0 (0%)	1.000
Concomitant atrial fibrillation	12 (57%)	21 (100%)	0.650
Echocardiographic parameters			
EF (%)	30 ± 11	30 ± 10	0.984
36–45%	9 (43%)	9 (43%)	0.954
26–35%	5 (24%)	5 (24%)	1.000
⩽ 25%	7 (33%)	7 (33%)	1.000
LVMI (g/m²)	152 ± 59	196 ± 73	0.035
ESVI (mL/m²)	46 ± 18	81 ± 33	<0.001
EDVI (mL/m²)	67 ± 21	115 ± 38	<0.001
LVIDs (mm)	45 ± 7	58 ± 8	<0.001
LVIDd (mm)	58 ± 7	71 ± 8	<0.001

Abbreviations: AF = atrial fibrillation; DCMP = dynamic cardiomyoplasty; DM = diabetes mellitus; EDVI = end-diastolic volume index; EF = ejection fraction; ESVI = end-systolic volume index; IHD = ichemic heart disease; LVIDd = LV end-diastolic dimension; LVIDs = LV end-systolic dimension; LVMI = LV mass index; TIC = tachycardia-induced cardiomyopathy.

TABLE 3: Echocardiographic changes in the TIC group

	Initial presentation	After treatment	P
EF (%)	30 ± 11	58 ± 6	<0.001
LVMI (g/m²)	152 ± 59	129 ± 32	0.048
ESVI (mL/m²)	46 ± 18	24 ± 8	<0.001
EDVI (mL/m²)	67 ± 21	56 ± 13	0.025
LVIDs (mm)	45 ± 7	36 ± 4	<0.001
LVIDd (mm)	36 ± 4	53 ± 4	0.004
SWT (mm)	9.1 ± 1.2	9.7 ± 1.7	0.144
PWT (mm)	9.6 ± 1.3	9.8 ± 1.4	0.565
TR Vmax (m/sec)	2.7 ± 0.5	2.4 ± 0.3	0.042

Abbreviations: EDV = ediastolic volume; EF = ejection fraction; ESVI = end-systolic volume index; LVIDd = LV end-diastolic dimension; LVIDs = LV end-systolic dimension; LVMI = LV mass index; PWT = LV posterior wall thickness; SWT = interventricular septum wall thickness; TIC = tachycardia-induced cardiomyopathy; TR Vmax = transtricuspid peak velocity.

TABLE 4: Echocardiographic changes in the rate and rhythm control groups

	Rhythm control group (n = 8)		Rate control group (n = 13)	
	Initial presentation	After treatment	Initial presentation	After treatment
EF (%)	26 ± 9	57 ± 8†	33 ± 11	59 ± 5†
LVMI (g/m²)	179 ± 86	136 ± 39	135 ± 24	125 ± 29
ESVI (mL/m²)	60 ± 19*	24 ± 9†	38 ± 11	24 ± 8†
EDVI (mL/m²)	81 ± 23*	56 ± 16†	58 ± 14	56 ± 12
LVIDs (mm)	49 ± 8	35 ± 2†	43 ± 6	36 ± 4†
LVIDd (mm)	61 ± 7	53 ± 5†	55 ± 7	53 ± 4

Abbreviations: EF = ejection fraction; EDVI = end-diastolic volume index; ESVI = end-systolic volume index; LVIDd = LV end-diastolic; LVIDs = LV end-systolic; LVMI = LV mass index. *p<0.05 versus corresponding value of rate control group. †p<0.05 brtdud corresponding value of initial presentation data.

ablation, or direct current cardioversion) at doctor's discretion. In addition, they all received conventional treatment for heart failure, including angiotensin-converting enzyme inhibitors or angiotensin receptor blocker, beta-blocker, digitalis, and diuretics if needed. After treatment, all patients became asymptomatic with improvement of the LVEF.

Rhythm control was used in 14 patients, 8 (57%) of whom showed restoration of sinus rhythm (rhythm control group). Restoration of sinus rhythm was achieved in 2 patients with atrial fibrillation (1 by ablation and 1 by cardioversion), 2 with atrial flutter (1 by ablation and 1 by antiarrhythmic drug), 3 with atrial tachycardia (all by antiarrhythmic drug), and 1 with PSVT (by ablation). One patient with initial atrial tachycardia, who showed maintenance of sinus rhythm for months while on amiodarone, experienced recurrent TIC after discontinuation of amiodarone (Figure 2). In another 13 patients, only heart rate control could be achieved (rate control group). One patient with initial

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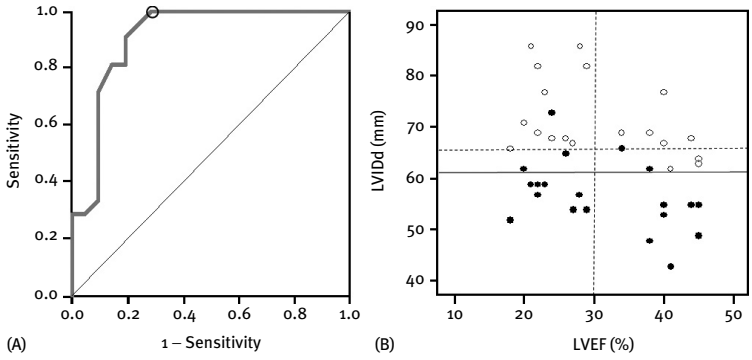

Figure 1: The ROC curve analysis (A) and distribution (B) of LV end-diastolic dimension according to EF in the TIC (●) and DCMP (○) Groups. *Abbreviations*: LVEF = LV ejection fraction; LVIDd = LV end-diastolic dimension; ROC = receiver-operating characteristic. LV diastolic dimension ⩽ 61 mm predicted TIC with a sensitivity of 100% and a specificity of 71.4%. In patients with EF ⩽ 30%, LV diastolic dimension ⩽ 66 mm predicted TIC with a sensitivity of 100% and a specificity of 83.4%.

atrial fibrillation experienced recurrent TIC due to poor medication compliance. Recurrence of TIC did not differ between the rhythm and rate control groups (log-rank test, $p = 0.808$).

Serial Changes of Echocardiographic Parameters

In the TIC group, LVEF increased from $30\% \pm 11\%$ (range, 10%–45%) to $58\% \pm 6\%$ (range, 46%–67%) after treatment (Wilcoxon signed-ranks; $p<0.001$). Left ventricle volume indices, dimensions, mitral regurgitation, and tricuspid regurgitation decreased significantly after treatment (Table 3). Left ventricle mass indices and transtricuspid peak velocity showed a tendency to decrease after treatment. Even though some patients had hypertension (48%), there was no evidence of LV hypertrophy on echocardiography and showed no interval change after therapy.

At initial visit, LV volume indices were larger in the rhythm control group than in the rate control group (Table 4). However, LV volume indices after treatment did not differ between these groups. Other echocardiographic parameters showed similar values in the 2 groups before and after treatment. In general, changes of LV volume indices and dimensions were remarkable in the rhythm control group.

Discussion

The major finding of this study is that the TIC group showed smaller LV dimensions and volume indices than the idiopathic DCMP at the initial visit, a finding that may be helpful in the differential diagnosis of TIC from idiopathic DCMP with tachyarrhythmia. Our finding suggested that LV diastolic dimension may be the best index to predict TIC. We also found that rate control of pure TIC was as effective as the rhythm control for EF improvement, relief of symptoms and prevention of TIC recurrence.

Dynamic cardiomyoplasty is a disease entity consisting of diverse etiologies and characterized by cardiac enlargement and impaired systolic function of one or both ventricles.[11] More than half of all patients with DCMP have no identifiable etiologies, and are classified as idiopathic DCMP.[12] Coronary artery disease and hypertensive heart disease are considered the leading causes of DCMP. There are also some rare but reversible disease entities. TIC is a curable disease and should be suspected in any patient with decreased ventricular function in the setting of supraventricular or ventricular tachycardia. Many rhythms and causes evolve into TIC, which include atrial fibrillation,[13,14] atrial flutter,[15] PSVT,[16] ventricular tachycardia,[17,18] fascicular tachycardia,[7] ventricular ectopy,[19] persistent rapid atrial or ventricular pacing,[20] and even extracardiac causes such as thyrotoxicosis and glucagonoma.[5,21]

Heart failure and tachyarrhythmia have a strong association, especially for atrial fibrillation. In general, tachyarrhythmia is a secondary consequence following advanced heart failure. In one study, as many as 35% of the patients with heart failure had concomitant atrial fibrillation.[22] Therefore, it may be difficult to distinguish pure TIC from idiopathic DCMP with tachyarrhythmia at the initial visit.

Initial Approach to Etiologic Differential Diagnosis of Heart Failure with Tachyarrhythmia (Figure 3): A New Suggestion

In an ovine model, TIC produced not only ventricular but also atrial cardiomyopathy. The chronic stimulation of the ventricular myocardium resulted in increased LV area, mitral regurgitation secondary to annular dilatation,

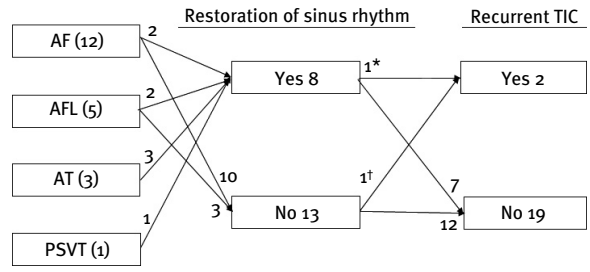

Figure 2: Sinus rhythm conversion and recurrence in the TIC group. *Abbreviations*: AF = atrial fibrillation; AFL = atrial flutter; AT = atrial tachycardia; PSVT = paroxysmal supraventricular tachycardia. *After restoration of sinus rhythm with initial AT, TIC recurred after discontinuance of amiodarone. †Recurrence due to poor medication compliance.

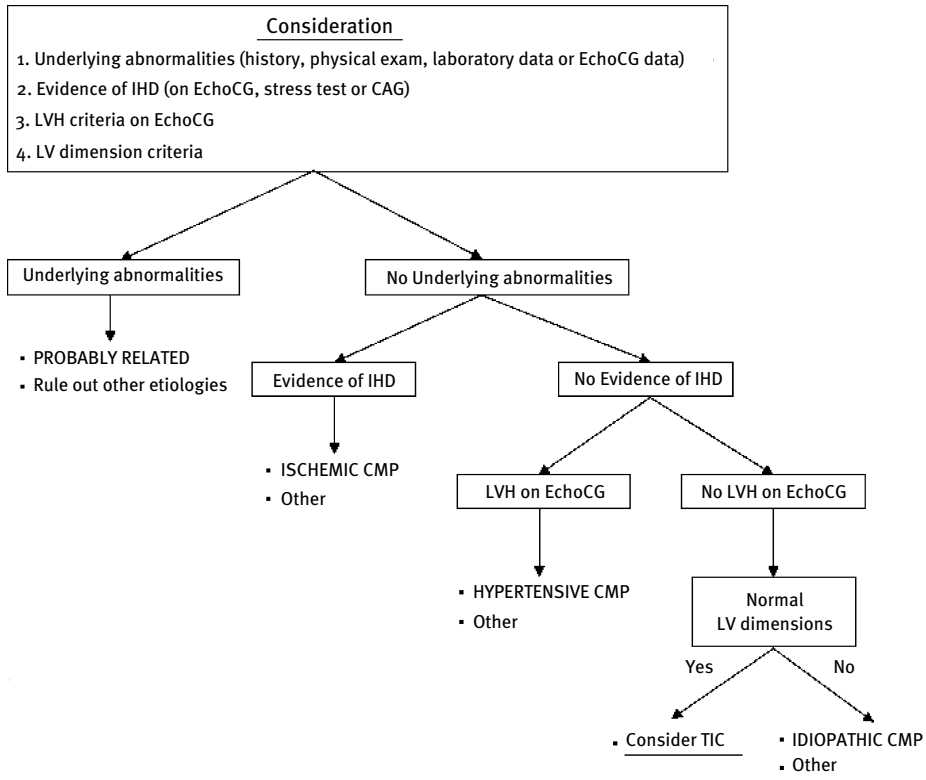

Figure 3: Initial approach to etiologic differential diagnosis of heart failure with tachyarrhythmia. *Abbreviations*: CAG = coronary angiography; CMP = cardiomyopathy; EchoCG = echocardiography; IHD = ischemic heart disease; LVH = left ventricular hypertrophy; LVIDd = LV end-diastolic dimension.

elevated LV end-diastolic pressure, and decreased LV wall thickness and systolic function.[23] In addition, dilatation and contractile dysfunction of the atrium occurred as the mechanical remodeling of atrial TIC.

We found that the TIC patients showed dilated atrium and ventricle, reduced LV contractility, and mitral and tricuspid regurgitation. However, LV mass indices, volume indices and LV dimensions were smaller in the TIC group than in the idiopathic DCMP group. Of these, LV diastolic dimension was the only independent predictor of TIC. As shown at Figure 1B, many patients showed normal LV diastolic dimension despite LV dysfunction. Ten patients (48%) showed LV diastolic dimension $\leqslant$ 55 mm and there was no overlap with idiopathic DCMP group. The causes of smaller LV dimensions and volumes in the TIC group are not clear. They may partly be attributed to the acute deterioration of LV function and rapid development of dyspnea without structural remodeling.

As outlined in Figure 3, we suggest the initial approach algorithm for etiologic differential diagnosis of heart failure with tachyarrhythmia at the initial visit. Earlier diagnosis of pure TIC may lead to earlier treatment of tachyarrhythmia. The suspected diagnosis can be confirmed by short-term echocardiographic and clinical follow-up to determine if there is a drastic improvement in LV dysfunction.

Treatment Methods and Clinical Outcomes

In case of atrial fibrillation, rate control has been shown to be not inferior to rhythm control in the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) study.[24] However, some authors reported that the "ablate and pace" modality was superior to pharmacological management in patients with LV dysfunction and tachyarrhythmia,[18,19] but the Australian Intervention Randomized Control of Rate in Atrial Fibrillation Trial (AIRCRAFT) reported that there was no significant difference in LVEF or exercise duration on treadmill testing.[25] Randomized prospective studies comparing the "ablate and pace" modality and pharmacological management in patients with LV dysfunction are rare, and patients of these studies included not only some pure TIC but also many structural heart diseases.[26,27] Present study included only pure TIC patients. We found that control of heart rate was comparable to restoration of sinus rhythm for symptoms and echocardiographic parameters.

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The conversion to sinus rhythm or control of ventricular rate has been considered to cause a good prognosis. However, Nerheim et al. recently reported that 3 of 24 TIC patients experienced sudden death despite apparent rate control, which rate control treatment for 6 mos eliminated heart failure symptoms and improved or normalized LVEF in all patients.[28] In the present study, there were no cases of death even after including two recurrent TICs. Exact clinical outcomes of TIC require long-term prospective studies in large populations.

Our study was limited by its retrospective design, the small number of patients, the nonrandom selection of patients, and insufficient data on diastolic dysfunction. The accurate duration of tachycardia before LV dysfunction and the recovery time of LV dysfunction were also difficult to determine.

Conclusion

The TIC patients showed smaller LV dimensions and volumes than patients with idiopathic DCMP at the initial visit. The LV end-diastolic dimension was the best determinant for predicting TIC at the initial visit. Rate control was as effective as rhythm control for EF improvement, relief of symptom and prevention of recurrent TIC.

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