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Tachycardiomyopathy: Clinical Profile, Treatment and Outcome of a Series of Clinical Cases

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Authors' contributions

This work was carried out in collaboration among all authors. Author RMFLS designed the study, performed the statistical analysis and wrote the first draft of the manuscript. Authors SMB, IFJ, GOC, MAG, RAPM and ICR collected data and managed the analysis of the study. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Background: Tachycardiomyopathy is a non-familial cause of heart failure that can be reversible. A high index of suspicion is necessary for its diagnosis and for a rational approach in the management of patients.
Aims: To describe the clinical profile and verify the treatment and outcome of a series of cases of patients with tachycardiomyopathy.
Methodology: Among patients without previous ventricular dysfunction, those with tachycardiomyopathy were identified. They underwent clinical evaluation, complementary diagnostic tests, treatment and clinical follow-up.
Results: 10 patients had tachycardiomyopathy, mean age 64.5 ± 11.5 years, 6 women. The arrhythmias were atrial fibrillation with a high heart rate (80%); atrial tachycardia with high heart rate and frequent ventricular premature beats. Mean baseline heart rate was 110.2 bpm and the ejection fraction was 36.8%. For heart rate control, all were treated with beta-blockers, in association with digoxin in 6 patients. It was necessary to implant a pacemaker in VVI mode

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(V: ventricle is stimulated, V: ventricle sensed; I: inhibiting response) and ablation of the atrioventricular node in 2 patients with persistent atrial fibrillation. After a mean 27.6 months, the mean heart rate was 76.5 bpm (p <0.0001, paired t-test) and there was clinical improvement of all patients. There was an average increase of 9.6% in the left ventricular ejection fraction. Despite this, 4 patients still had ventricular dysfunction (3 of them developed comorbidities).

Conclusion: In this series, the main cause of tachycardiomyopathy was atrial fibrillation with high heart rate. Similar to the description of literature, there was remission of symptoms with restore normal heart rhythm or control heart rate.

Keywords: Tachycardiomyopathy; tachycardia-induced cardiomyopathy; heart failure; atrial fibrillation; reversible; tachycardia.

1. INTRODUCTION

Tachycardiomyopathy is a condition of left ventricular systolic dysfunction induced or mediated by persistent arrhythmia, with high heart rate and/or with ventricular asynchrony [1,2]. Therefore, this non-familial cause of dilated cardiomyopathy may occur in patients without heart failure or may worsen ventricular dysfunction in those with concomitant heart disease [3,4]. The first description was in 1913 by Gossage [5] in a patient with atrial fibrillation with high heart rate, however the first experimental model of pacing-induced heart failure was published in 1962 by Whipple et al [6]. Its incidence is estimated between 8% and 28% (for supraventricular tachycardias) and up to 34% (for ventricular extrasystoles > 10 or 24% of the total QRS complexes per day). Ventricular dysfunction may be reversible upon months after treatment of arrhythmia or heart rate control [1,7]. A high index of suspicion is necessary for its diagnosis and for a rational approach in the management of patients. There are few articles with a small number of patients with tachycardiomyopathy on their evolution [1]. The purpose of this article is to describe a series of cases of tachycardiomyopathy, their treatment and their outcome.

2. CASE PRESENTATION

Among patients without previous ventricular dysfunction, 10 were identified with tachycardiomyopathy, who underwent clinical evaluation, complementary diagnostic tests, treatment and clinical follow-up. The diagnosis of tachycardiomyopathy was made by exclusion.

The mean age was 64.5 ± 11.5 years, ranging from 52 to 85 years, 6 women. The arrhythmias responsible for ventricular dysfunction were atrial fibrillation with a high heart rate in 8 patients (80%); atrial tachycardia with high heart rate (> 50% per day) in a patient and frequent ventricular premature beats in another patient (12% of the total QRS complexes/24h) with QRS of 160 ms in duration. The other clinical and echocardiographic data of the patients are shown in Table 1.

The mean serum creatinine level was 0.85 ± 0.16 mg/dL, with clearance by the Cockcroft-Gault equation of 79.2 ± 33.8 mL/min.

Table 1. Clinical and echocardiographic data of the patients

Variables (units)	Mean ± SD	Range
Body mass index (kg/m^2)	25.6 ± 5.3	17.3 - 32.2
Baseline heart rate (bpm)	110.2 ± 11.4	88.0 - 124.0
Baseline blood pressure systolic/diastolic (mmHg)	113.2 ± 30.3/67.8 ± 12.0	80 - 182/50 - 92
Left atrial diameter (mm)	45.3 ± 10.4	27 - 61
Left ventricular diastolic/systolic diameters (mm)	54.0 ± 9.0 /41.6 ± 8.9	39 - 65/30 - 54
Left ventricular ejection fraction % (Teichholz method)	36.8 ± 5.1	30.0 – 47.0
Pulmonary artery systolic blood pressure (mmHg)	30.0 ± 7.5	15 - 40

SD: Standard deviation; bpm: beats per minute

3. MANAGEMENT AND OUTCOME

All patients were treated with angiotensin-converting enzyme. For heart rate control, all were treated with beta-blockers and 6 in combination with digoxin. Amiodarone was used by 5 patients. Two patients with persistent atrial fibrillation underwent pacemaker implantation in VVI mode (the ventricle is stimulated, sensed and the pulse generator inhibits the stimulation output in response to a detected ventricular event) with ablation of the atrioventricular node due to the failure of pharmacological treatment and/or failure in cardioversion and ablation.

After a mean 27.6 months, the mean heart rate was 76.5 ± 9.3 bpm ($p<0.0001$, paired t- test) and there was clinical improvement of all patients (New York Heart Association –NYHA - functional class from 3.9 to 1.8, $p <0.0001$). Tachycardiomyopathy recurrence occurred in 2 patients (in one due to unsuccessful ablation of atrial fibrillation and in another due to recurrence of arrhythmia with permanent atrial fibrillation). There was an average increase of 9.6% in the left ventricular ejection fraction (mean of 46.4 ± 15.8%; ranging from 23% to 73%, $p=0.05$). Despite this increase in ventricular function, 4 patients still had ventricular dysfunction during a follow-up time of 61.3 months. Two patients developed dilated cardiomyopathy due to another etiology (Chagas disease and cor pulmonale) and one patient had coronary artery disease as comorbidity. These patients did not have ventricular dysfunction before the rapid atrial fibrillation that induced the tachycardiomyopathy.

4. DISCUSSION

There have been advances in the technology of catheter ablation of atrial fibrillation and monomorphic ventricular extrasystoles that cause tachycardiomyopathy [1,4]. Success rates reach between 80% and 95% depending on the mechanism of supraventricular tachycardia that induces ventricular dysfunction [8]. The strong findings of this case series were pharmacological treatment based on the neurohormonal mechanism of tachycardiomyopathy and effectiveness of the combination of ventricular stimulation and atrioventricular node ablation for patients with refractory atrial fibrillation with high heart rate.

The classification of tachycardiomyopathy is made in type 1 (arrhythmia-induced) and type 2 (arrhythmia-mediated) [1,3,8]. Persistent atrial fibrillation and atrial flutter with a high ventricular rate are the most studied arrhythmias among those that cause tachycardiomyopathy. Ectopic atrial tachycardia is the most frequent cause of tachycardiomyopathy in the pediatric population. Therefore, this series of cases is in accordance with what has been described in the literature regarding the cause of tachycardiomyopathy. Less frequent causes of supraventricular arrhythmias that induce tachycardiomyopathy are permanent junctional re-entrant tachycardia, atrioventricular tachycardia, nodal tachycardia with simultaneous antegrade conduction through fast and slow atrioventricular nodal pathways [1,9-11]. Frequent premature ventricular contractions with burden greater than 10% or 24% of the total QRS complexes during 24 h can result in ventricular dyssynchrony and tachycardiomyopathy [1-3]. There is a greater risk of tachycardiomyopathy if these premature beats have a QRS duration greater than 150 ms [3] as with one of the patients in this case series.

The pathophysiology of tachycardiomyopathy is explained by experimental models with rapid ventricular pacing [1,3,8,12]. After the first week of tachycardia or dyssynchrony, there is an increase in metabolic demand and hemodynamic impairment. With ventricular dilation, there is neurohormonal activation. Heart failure can occur from the 3rd week. For this reason, the management of these patients includes the improvement of symptoms, with action on the neurohormonal mechanism, and the elimination or control of the arrhythmia. The recurrence of arrhythmia with tachycardiomyopathy has been reported in series of up to 24 patients, with rates of up to 25% [1,3] because of the persistence of cardiac structural changes. In the present study, the recurrence rate was similar (20%). In a larger retrospective series of 36 patients, the recurrence rate was 31% over the 5.6-year period [13]. Hence the importance of long-term follow-up of patients with a history of tachycardiomyopathy

According to data in the literature, recovery can occur within 6 months [1], less than the time in the present case series. This may be due to the delay in cardioversion or radiofrequency ablation procedures to control arrhythmia. Tachycardiomyopathy is usually a reversible cause of heart failure, with almost complete recovery from symptoms and ventricular contractility. However, improvement in symptoms or ejection fraction may not be observed [1,3].

Also for this reason, the treatment in order to prevent recurrence of the arrhythmia and control of neurohormonal activation in the long term are important in the management of these patients.

5. CONCLUSIONS

In this series, the main cause of tachycardiomyopathy was atrial fibrillation with high heart rate. Similar to the description of literature, there was remission of symptoms with restore normal heart rhythm or control heart rate.

CONSENT

The authors declare that written informed consent was obtained from the patients.

ETHICAL APRROVAL

All authors declare that this study is part of an approved research by the appropriate ethics committee and has therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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