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Tachycardia-Induced Cardiomyopathy Mimicking End-Stage Heart Failure: A Case Report

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Abstract

Background: Tachycardia-induced cardiomyopathy is a potentially reversible subtype of dilated cardiomyopathy which can arise in ventricular and supraventricular tachyarrhythmia settings.

Case Report: A 48-year-old male with a history of hypertension and paroxysmal atrial fibrillation with a recently diagnosed dilated cardiomyopathy of unknown origin with an ejection fraction of 13% presented feeling unwell with palpitations and congestion symptoms two months after initiating heart failure and antiarrhythmic therapy. A full differential cardiac workup, which included electrocardiography, cardiac enzymes, coronary angiography, cardiac echocardiography, and cardiac magnetic resonance imaging was performed. Other than a persistently low ejection fraction of 13% on echocardiography and atrial fibrillation on electrocardiography, all requested tests were inconclusive for any specific etiology. The patient was cardioverted into sinus rhythm and reported rapid symptomatic relief followed by a substantial increase in his ejection fraction the following day (13% to 53%). Tachycardia-induced cardiomyopathy was subsequently diagnosed retrospectively based on a recorded ejection fraction normalization following sinus rhythm restoration, presence of longstanding paroxysmal atrial fibrillation and only after excluding ischemic heart disease and other possible causes. Following a recurrence of atrial fibrillation and a decrease in ejection fraction to 45% two years later while on amiodarone and beta blockers, the patient underwent radiofrequency ablation with no further reported symptoms and atrial arrythmia recurrences.

Conclusion: Tachycardia-induced cardiomyopathy should be suspected in patients with chronic tachyarrhythmias and unexplained left ventricular systolic dysfunction. If not counter indicated, catheter ablation must be prioritized as a safe and effective method to treat patients with atrial fibrillation-mediated tachycardia-induced cardiomyopathy.

Keywords: Tachycardia-induced cardiomyopathy; Heart failure; Atrial fibrillation; Radiofrequency catheter ablation therapy; Cardiac magnetic resonance imaging

Introduction

Heart failure continues to be an important health and socioeconomic issue for patients as its prevalence continues to climb despite therapeutical advancements and a better understanding of disease management [1]. Tachycardia Induced Cardiomyopathy (TIC) is a type of arrhythmia induced cardiomyopathy that arises in settings of supraventricular and ventricular tachyarrhythmias. It characterized by ventricular dysfunction and dilatation and presents with heart failure symptoms which regress after sinus rhythm restoration [2,3]. TIC can develop over periods ranging from 1 month to years of registered sustained tachyarrhythmias [4]. Nevertheless, cases of acute TIC have been previously documented [5] and experimental studies in animals show that hemodynamic changes can manifest as early as 24 h after pacing [6]. Although TIC has been described to arise as a result of virtually any ventricular or supraventricular tachycardia, Atrial Fibrillation (AF) is believed to be its most common cause with recent studies showing that up to one third of patients with

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AF can exhibit certain features of this disease [7-9]. TIC is treated by targeting the culprit tachyarrhythmia [3]. Multiple management approaches, including various rhythm and rate control techniques, are currently being used in the treatment of AF [10,11]. However, and especially in AF-mediated TIC, Radiofrequency Ablation (RFA) is proving to be more efficacious when compared with the other approaches in terms of recurrences, mortality and hospitalization rates, and EF restoration [12-16]. To emphasize the importance of early recognition and treatment of TIC and the superiority of RFA in the management of its AF-mediated subtype, we present the following case.

Case Presentation

A 48-year-old male was referred for advanced heart failure treatment and presented feeling unwell with palpitations, shortness of breath and peripheral oedema. The patient was previously admitted to a local hospital and diagnosed with acutely decompensated heart failure after developing congestion symptoms and experiencing palpitations three months before coming to our hospital. During his previous stay at the local hospital, atrial fibrillation of an unknown duration was noted and a transthoracic echocardiogram revealed a dilated left ventricle with EF of 13%, mild to moderate mitral regurgitation and moderate pulmonary hypertension. Coronary angiography was also performed and patent coronary arteries were observed. A diagnosis of dilated cardiomyopathy was established. The patient was prescribed and up-titrated ACE inhibitors (ACEi) (perindopril), beta-blockers (bisoprolol), spironolactone, digoxin, Direct Oral Anticoagulants (DOAC), amiodarone, and loop diuretics. Hypotension did not allow for an initial substantial increase in the dosages, however, three months into the treatment plan, ACEi and beta-blockers doses were about 50% to 75% of targeted values. The patient was still symptomatic three months after treatment initiation and a transthoracic echocardiography showed no improvement in his EF (13%) (Table 1). The patient was referred for advanced treatment and was admitted to our hospital. Heart auscultation revealed

and 2 years on amiod	darone and beta	-blockers.	er DC cardioversion,
Date	Reference	1 day after DC cardioversion	2 years on amiodarone and

		cardioversion	beta-blockers
LVEF %	13	53	62
EDV, ml	224	174	135
ESV, ml	193	95	52
SV, ml/beat	31	79	83
LVEDD, mm	63	58	53
LVESD, mm	41	38	35

LVEF: Left Ventricular Ejection Fraction; EDV: End Diastolic Volume; ESV: End Systolic Volume; SV: Stroke Volume; LVEDD: Left Ventricular End Diastolic Dimension; LVESD: Left Ventricular End Systolic Dimension

an irregular heartbeat and no murmurs, the rest of the physical examination revealed pitting leg oedema and bibasilar crackles. A full cardiac workup was ordered. An Electrocardiogram (ECG) revealed AF with a ventricular rate of approximately 153 beats/ min (Figure 1). Troponin and N-Terminal pro-B-type Natriuretic Peptide (NT-proBNP) were negative. A coronary angiogram was nonremarkable and ruled out cardiac ischemia as the cause of dilated cardiomyopathy. Cardiac Magnetic Resonance imaging (CMR) with late gadolinium enhancement was performed and showed no features of myocarditis, noncompaction cardiomyopathy, or any other structural changes (Figure 2). A combination of persistent AF, patent coronary arteries and normal CMR results led us to suspect a diagnosis of TIC. At that point, the patient had been enduring AF for 7 months. After pre-treatment with amiodarone, direct current cardioversion was performed with sinus rhythm restoration, after which the patient reported significant symptomatic relief. A transthoracic echocardiography was performed the next day and showed an EF of 53% (Table 1) with satisfactory left ventricle range of motion. The diagnosis of TIC was confirmed. The patient was discharged and reported no further symptoms on beta blockers, dabigatran, and amiodarone and was recommended to consult



segment changes and bundle branch blocks.



Figure 2: Cardiovascular magnetic imaging with late gadolinium enhancement in the short axis (A, B, C), horizontal long axis (D) and vertical long axis (E) showing normal myocardium with no signs of structural damage.



Figure 3: Map of Left atrium with ablation points.

with a cardiac electrophysiologist for further studies regarding the possibility of ablation for conclusive AF treatment.

A follow-up transthoracic echocardiography 2 years later showed normal left ventricular range of motion with no evidence of hypokinesia and an EF of 62% (Table 1). A 25-h Holter-monitor registered a sinus rhythm (median HR 62 beats/min) with occasional premature atrial and ventricular contractions and no sign of any AF episodes.

About two years later, after receiving a dose of Sputnik V (COVID-19 vaccination), an AF recurring episode was noted. After a few days of recorded persistent arrhythmia, transthoracic echocardiography showed a drop of the patient's EF down to 45%. The patient developed heart failure symptoms that were classified as class II NYHA and underwent direct current cardioversion with normal sinus rhythm restoration.

In a patient with recurrent symptomatic persistent atrial

fibrillation and a history of tachycardia induced cardiomyopathy, we decided to perform radiofrequency catheter ablation (Indications class I, Level of evidence B according to ESC Guidelines) [11].

The procedure was performed during sinus rhythm. Isolation of pulmonary veins was performed with an RFA catheter (THERMOCOOL SMARTTOUCH, Biosense Webster) and confirmed by Decapolar catheter (Lasso, Biosense Webster). After PV isolation (cavotricuspid isthmus - dependent) atrial flutter was induced by burst pacing protocol. Atrial flutter transformed into atrial fibrillation spontaneously. RFA of fragmented potentials was performed with transformation of AF back to flutter, Cavotricuspid isthmus ablation was performed with sinus rhythm restoration (Figure 3). The patient was followed for 6 months after ablation. 24-h ECG monitoring revealed no signs of sustained atrial arrhythmia and no clinical recurrence of arrhythmia was noted by the patient. Amiodarone was stopped 2 months after ablation.

Discussion

TIC is a subtype of dilated cardiomyopathy that is potentially reversible if diagnosed in a timely manner. Sustained tachycardia can cause ischemia, loss of myocytes, sarcomere destruction, oxidative stress and intracellular electrolyte disbalance, all of which are believed to be implicated in the development of ventricular dysfunction [17,18].

Its incidence might be significantly underreported due to the fact that cardiomyopathies as a causative factor themselves are associated with the development of several well-studied tachyarrhythmias and the diagnosis of TIC can be overlooked due to this [19,20].

AF is the most common cause of TIC with studies showing that it can contribute to 77% of cases and finding that certain elements of left ventricular dysfunction being present in up to one-third of patients with AF [6-8,21].

Similar to other diagnoses of exclusion, TIC should be suspected in HF patients with no apparent cause, presenting with a history of tachyarrhythmias after excluding structural cardiac muscle abnormalities through thorough cardiac workup. It should be noted that the ability of cardiac MRI studies to safely assess cardiac muscle tissue for scarring and necrosis makes it an ideal tool in the differential diagnosis of TIC [6,22].

TIC is treated using the combination of standard anti-failure therapy, anticoagulation for patients with atrial fibrillation and CHA_2DS_2 -VASc score ≥ 1 , and sinus rhythm restoration. The latter can be achieved by antiarrhythmic drugs or through radiofrequency ablation [23].

The decision to choose rate or rhythm control should be individualized based on the presentation and workup results of the patient. According to recent guidelines, rhythm control is recommended with the main indication being to improve symptoms and Quality of Life (QOL) in younger patients, patients with a short history of AF, no or few cardiac commodities, when rate control is difficult to achieve, and in TIC, while rate control is favored in asymptomatic/mildly symptomatic patients, as background therapy in all AF patients, or after failure of rhythm control [24-27].

As illustrated in our case, cardioversion (electrical or pharmacological) should always be considered as a method of acute rhythm control in symptomatic patients with or without hemodynamic instability. Several random clinical trials have shown an improvement in QOL standards and high success rates with insignificant recurrences after AF cardioversion [28-30].

Special emphasis has recently began being put on the efficacy of ablation therapy in AF with studies reporting more significant increases in EF in patients with AF and HF and less recurrence rates when compared to rate control medical therapy [12-14].

Moreover, some studies comparing rhythm control management methods even showed that catheter ablation reduces mortality, hospitalization, and recurrence rates in patients with AF and HF when compared to amiodarone [15,16].

Emerging evidence favoring the use of catheter ablation instead of standard medical rate and rhythm therapy in treating AF, especially in HF and TIC patients, combined with general wariness of physicians towards prescribing toxic and potentially dangerous antiarrhythmic drugs with relatively higher recurrence rates [31-33] pushes us to predict a broadening of catheter ablation indications currently being used and recommended.

The case we are discussing illustrates typical presentation symptoms, cardiac workup results, and long-term outcomes of TIC patients. This case reinforces existing literature surrounding TIC, and namely the premise that it, unlike ischemic and other non-ischemic causes of CM, is reversible if diagnosed promptly and managed correctly, and highlights the responsibility of physicians to keep this rare but well-studied diagnosis in their differential while assessing patients with tachyarrhythmias and Left Ventricular Systolic Dysfunction (LVSD) of unknown origin.

Conclusion

Despite being a rare cause of dilated cardiomyopathy, TIC should be always be included in the differential when assessing patients with a history of supraventricular or ventricular tachyarrhythmias and unanticipated LVSD as it is potentially reversible with guidelinecompliant heart failure medical therapy, anticoagulation in AF, and sinus rhythm restoration. TIC is a diagnosis of exclusion and full cardiac workup, including cardiac imaging, should be ordered to exclude cardiac muscle structural damage and necrosis. Finally, we would like to shine a light on recent data suggesting the superiority of ablation therapy over standard medical rate and rhythm therapy for AF in heart failure/TIC patients and non-HF patients in overall freedom from AF, better quality of life, and lower mortality and hospitalization rates.

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