RESEARCH PAPER

Tachycardia-induced cardiomyopathy, a diagnosis forgotten in the real world: Review of case series.

Amar Alhamdi[1]

1. MRCP (UK), FRCP(Ed.), Cardiologist and Electrophysiologist, Sulaimanya, KRG, Iraq

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Abstract

Background: Left ventricular dysfunction induced by long-standing recurrent tachyarrhythmias, known as tachycardia-induced cardiomyopathy, is a reversible type of dilated cardiomyopathy and heart failure characterized by left ventricular failure that is usually recoverable once the tachyarrhythmia is well controlled by stabilizing the sinus rhythm or by heart rate control.

Patients and methods: Twenty four patients with heart failure due to dilated cardiomyopathy and tachyarrhythmia were included. The arrhythmia and heart failure were both treated accordingly. The New York Heart Association'sfunctional class, left ventricular ejection fraction assessment and chest X-ray were done periodically to assess improvement.

Results: Stabilization of the sinus rhythm was achieved in all patients. The NYHA class improved remarkably, from class III-IV to class I in all patients. The left ventricular ejection fraction rose remarkably from 20-30% to 45-57%. The cardiac size remarkably improved radiologically. The symptoms of recurrent long-standing palpitation preceding the symptoms of dyspnea in heart failure are the first implication that arrhythmia is inducing the heart failure this was noticed in 20 patients.

Conclusion: Tachycardia-induced cardiomyopathy is a reversible cause of heart failure. Controlling the arrhythmia recovers normal LV function.

Key words: tachycardia, cardiomyopathy

***Correspondence to,** Amar Alhamdi Cardiologist and Electrophysiologist, Sulaimanya, KRG, Iraq

✉ **amaralhamdi@yahoo.com**

Introduction

Cardiomyopathy is a primary heart muscle disorder with myriad causes and clinical presentations. The cardiomyopathies are often named according to the underlying etiology, like hypertensive, ischaemic, myocarditis and infiltrative cardiomyopathy[1-6]. The normal heart rate of 60 to 90 bpm allows normal left ventricular function [1]. A sustained heart rate increase in a tachyarrhythmia pattern may well induce a cardiomyopathy called tachycardia-induced cardiomyopathy (TIC).[1-6] It is a type of dilated cardiomyopathy (DCM) caused mainly by recurrent paroxysmal or incessant tachycardia or by disturbing the rhythm of various mechanisms.[1-6] It may manifest months or years after the onset of the arrhythmia.[3] Tachycardia - induced cardiomyopathy might well be rate

dependent, as faster tachycardia develops TIC earlier.[5] This entity was first reported in 1913 by Gossage and Braxton Hicks in a patient with atrial fibrillation who presented with heart failure (HF).[2] The association of tachyarrhythmias with heart failure is quite common, but what distinguish TIC are mainly the heart failure's full or partial resolution and the regaining of normal left ventricular function after a period of sinus rhythm stabilization or after reasonable control of the heart rate within the arrhythmia.[2-5] TIC can occur in a basically structurally normal heart where the arrhythmia purely precipitates the myocardial function suppression and the occurrence of heart failure. It may also occur in pathological heart diseases where the arrhythmia potentiates or worsens the left ventricular function impurely.[5] The most common arrhythmias causing TIC are atrial fibrillation (AF), atrial flutter (AFl) and atrial tachycardia (AT), whether paroxysmal, persistent or chronic permanent.[7,12] Incessant type of atrioventrcular tachycardia (AVRT) like permanent junctional reciprocating tachycardia (PJRT) is a well-known cause of heart failure and cardiomyopathy in young and childhood age groups.[9] Ventricular premature beats (VPCs) may induce DCM if they are frequent enough to impair LV function.[8,13] Other supraventricular tachycardias (SVT), including atrioventriculo nodal reentry tachycardia (AVNRT), rarely induces DCM and heart failure, possibly because they are almost paroxysmal.[6] Recognizing the arrhythmia as a cause of DCM and heart failure is clinically highly important for resolving the DCM and regaining normal LV function.[2,4,5,14] We are reporting 24 cases of TIC patients who regained full or partial normal LV function and resolution of the DCM after controlling the arrhythmia. Physicians need a high degree of suspicion to diagnose TIC and cannot forget tachycardia as an underlying etiology of DCM and HF.

Patients and methods

Twenty four patients who were diagnosed as DCM with heart failure are recognized and included in this study; the DCM is associated with cardiac arrhythmia. The cases were seen from 2006 to 2017 in a single private clinic of the author. Full histories were taken, concentrating on which symptoms started first, for the total duration of the HF symptoms. If the palpitation symptoms preceded the shortness of breath (SOB), the patient was labeled as group A (GrA). If the SOB symptoms preceded the palpitation, the patient was enrolled in group B (GrB). If it was unclear which symptoms started first, they were included in group C (GrC). All the patients had gone through basic cardiac investigations, including 12-leads electrocardiogram (ECG), chest X-ray (CXR), an echocardiogram (ECHO) assessing the LV cavity's sizes in diastole and systole and calculating the left ventricular ejection fraction (LVEF). A Holter monitor was done in almost all the patients before treatment and within the course of therapy. All the tests were repeated periodically during the follow-up period, according to the patients' attendance at the follow-up. Eight patients had atrial flutter, 10 patients had atrial fibrillation, four patients had VPCs and two patients had PJRT. The treatment included therapies for HF and for arrhythmia. Sinus rhythm stabilization was

the main and only index of arrhythmia control. The arrhythmia therapy included either antiarrhythmic drug therapy (in 18 patients) or catheter radiofrequency ablation therapy (in 6 patients). According to the HF's clinical status, the treatment of HF included diuretics, an angiotensin-converting enzyme (ACE) inhibitor, and isosorbide and beta blockers. DC cardio version was used in 5 patients to revert their sinus rhythm during the early stages of the arrhythmia treatment. Assessment of the HF's improvement depended on a) regression in New York Heart Association (NYHA) functional class, b) improvement in LVEF and c) a reduction in cardiac size by CXR, expressed by a reduction in the cardiothoracic (C/T) ratio. Confounders implicated in the aetiology of tachycardia such as thyrotoxicosis and pheochromocytoma were excluded by appropriate tests. The DCM's improvement grade was based on the increase in LVEF, which is classified in as a full recovery of LVEF for more than 50% or a partial recovery for values from 45% to 49%. The total follow-up period ranged from three months to two years.

Results

Twenty-four patients were included in this study after fulfilling the selection criteria. Nine patients were males, and 15 were females. The ages ranged from 10 years to 55 years. DCM was diagnosedclinically by cardiologist, and provisionally structural pathological causes were excluded via clinical history, ECG, echocardiography findingsa specific tests. A diagnostic coronary angiography was done in 5 patients, which revealed normal coronary arteries. The arrhythmia symptoms preceding the HF symptoms were seen in 20 patients (GrA). The total palpitation symptoms' duration ranged from two months to three years. In two patients, the arrhythmia symptoms and the HF co- exist for the symptoms' total duration (GrC). In the remaining two patients, the HF symptoms of SOB preceded the arrhythmia symptoms (GrB), according to the patient's history. The NYHA functional class was in the level of II-IV at time of presentation and in NYHA I after variable times, ranging from seven days to three months, after stabilizing SR. The LVEF values rose remarkably, from an average of 20% to 32% to an average of 45% to 58% after the arrhythmia control (Figure-1). The cardiac size assessed by the C/T ratio in the CXR showed marked reduction after controlling the arrhythmia (Figure-2).

The recovery period for LV function after the arrhythmia control varied from one week to six months and correlated to the total duration of the arrhythmia and HF symptoms. Figures-3(A, B, C and D) show the remarkable improvement of LVEF and cardiac size after the atrial flutter reversion to sinus rhythm by catheter radiofrequency ablation therapy (CRFAT). Similar changes are seen in Figure 4 (A-D), showing a patient with atrial fibrillation, and Figure 5 (A-D), showing a patient with VPCs.

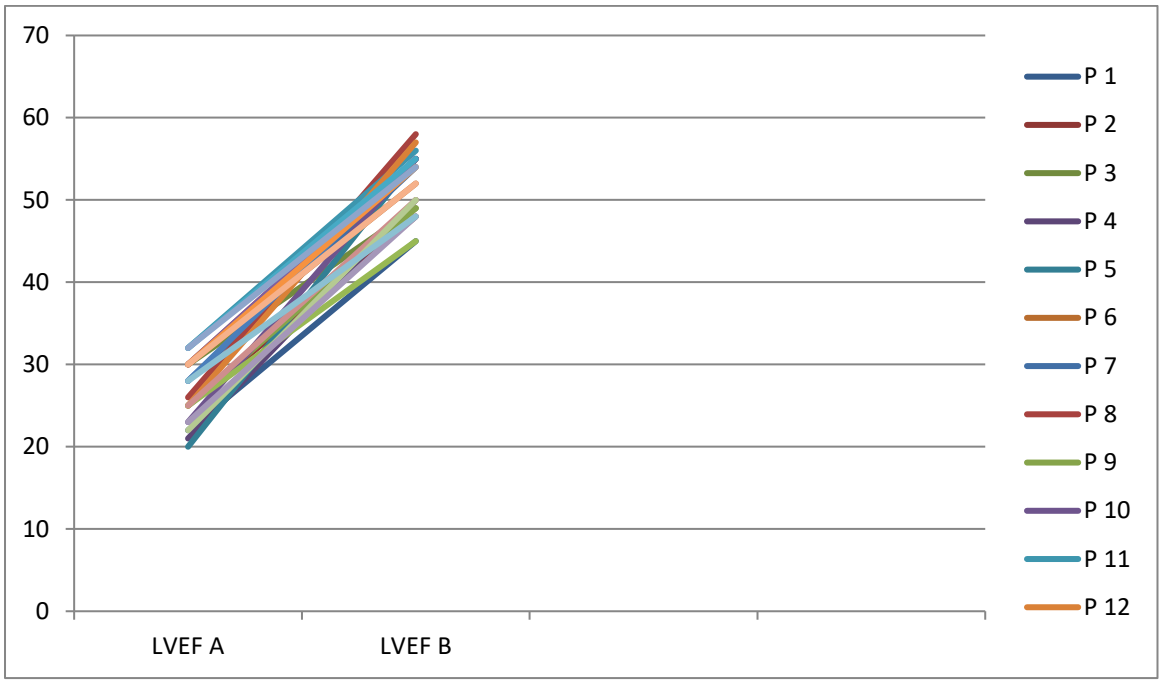

Figure 1 , The improvement in LVEF before (A) and after (B) arrhythmia control in the 24 patients.

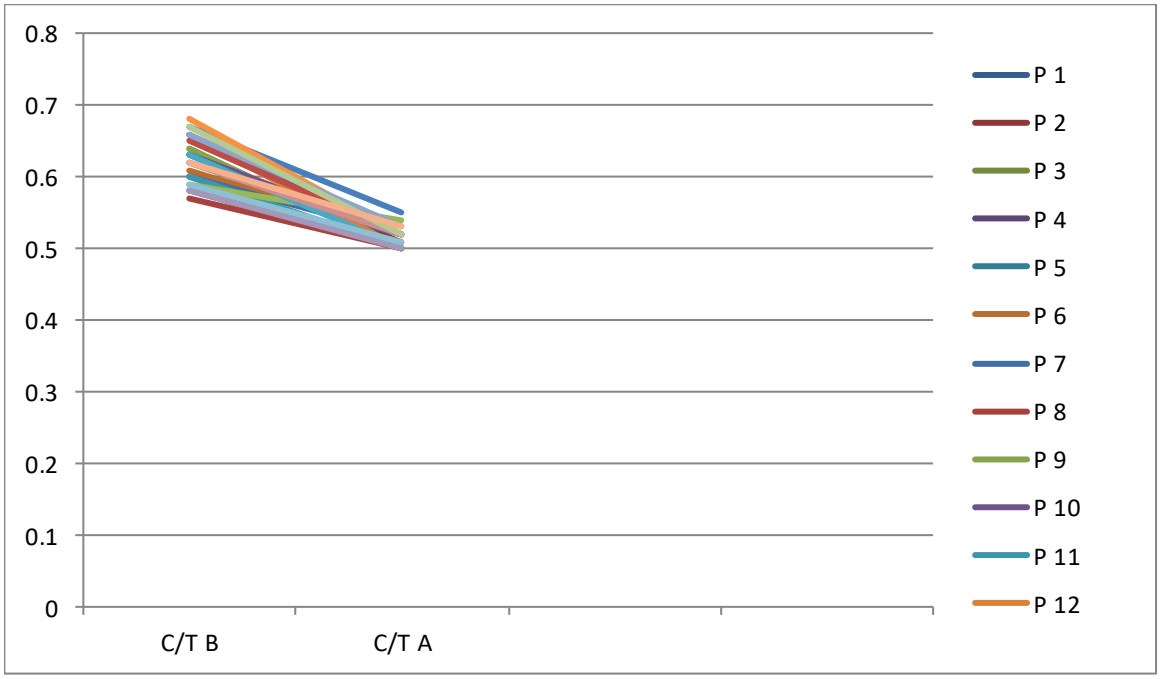

Figure 2, chest X ray cardiac size improvement assessed by cardiothoracic ratio (C/T ratio) before (C/T B) and after (C/T A) arrhythmia control.

Figure 3A. The 12-leads ECG showing atrial flutter.

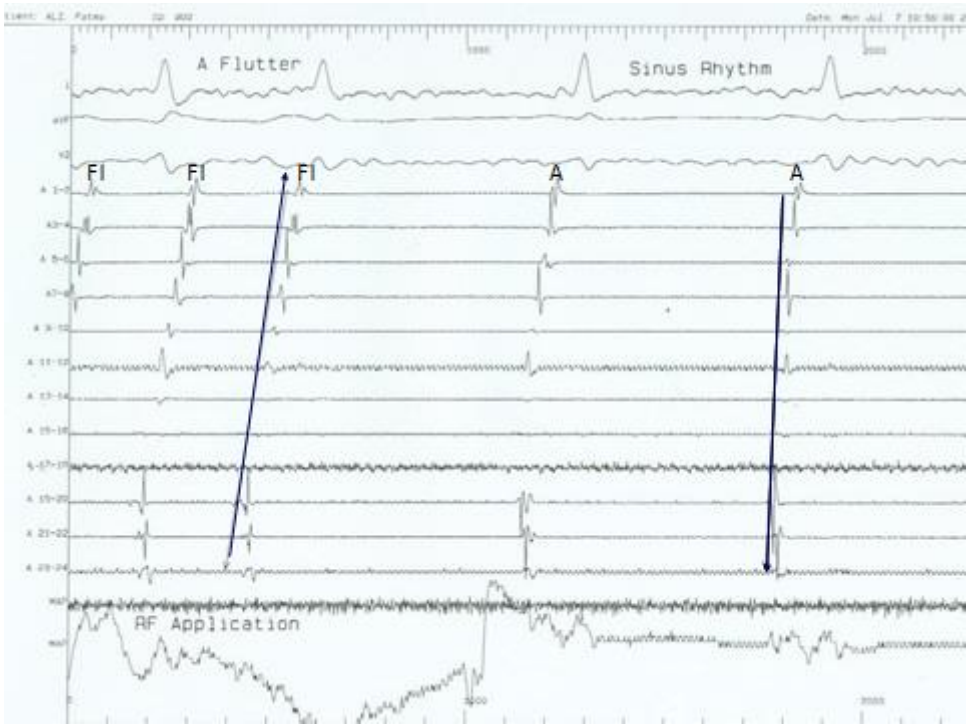

Figure 3B. The electrophysiology trace showing the reversion of atrial flutter in sinus rhythm with catheter ablation therapy.

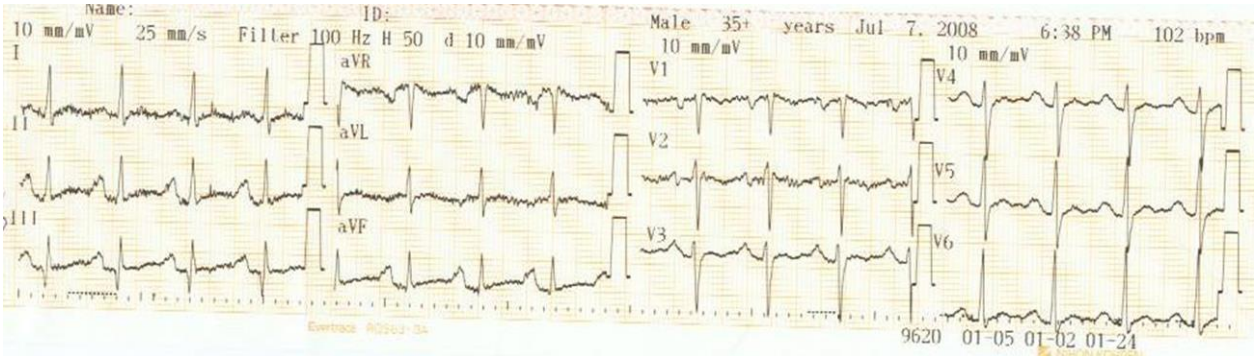

Figure 3C. The twelve leads ECG with sinus rhythm after catheter ablation therapy.

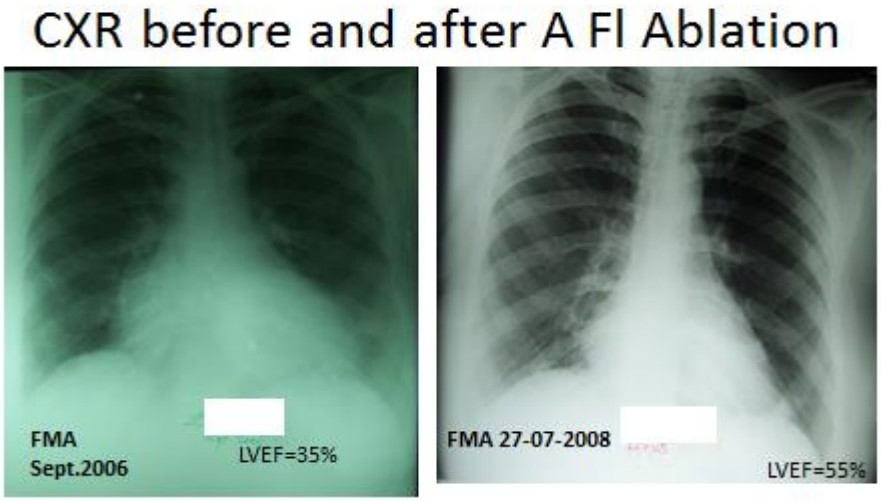

Figure 3D. Patient No 1.The reduction in cardiac size 10 months after stabilizing the sinus rhythm.

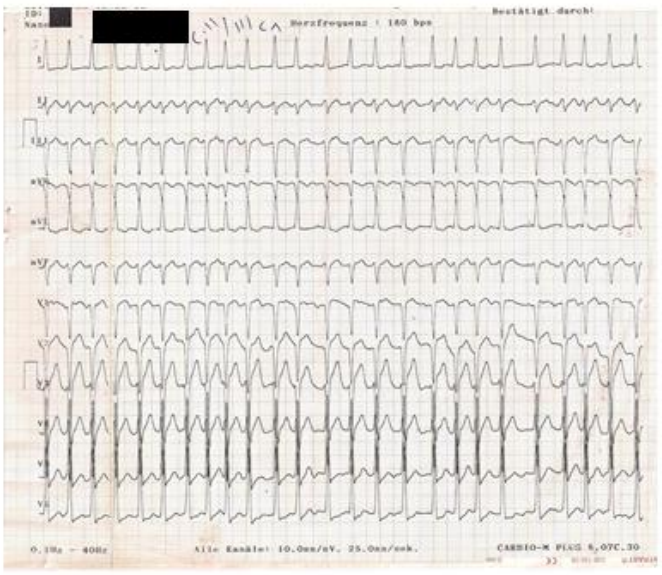

Figure 4A, ECG showing atrial fibrillation with very fast ventricular rate.

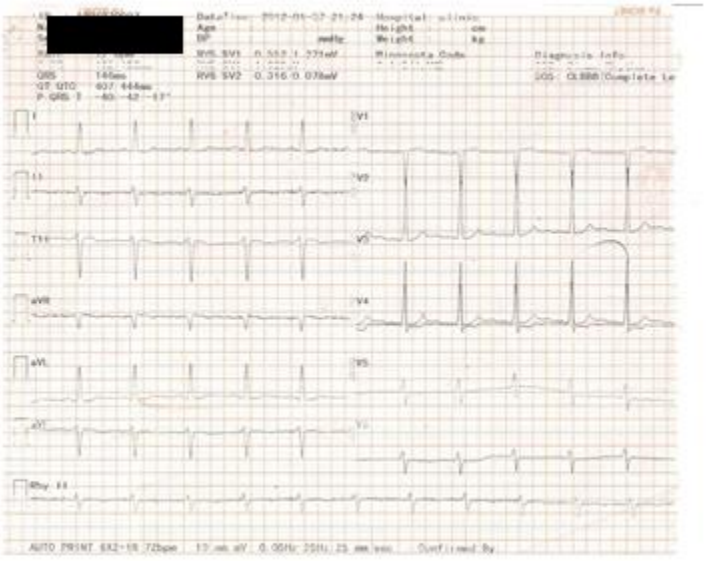

Figure 4B. ECG showing sinus rhythm

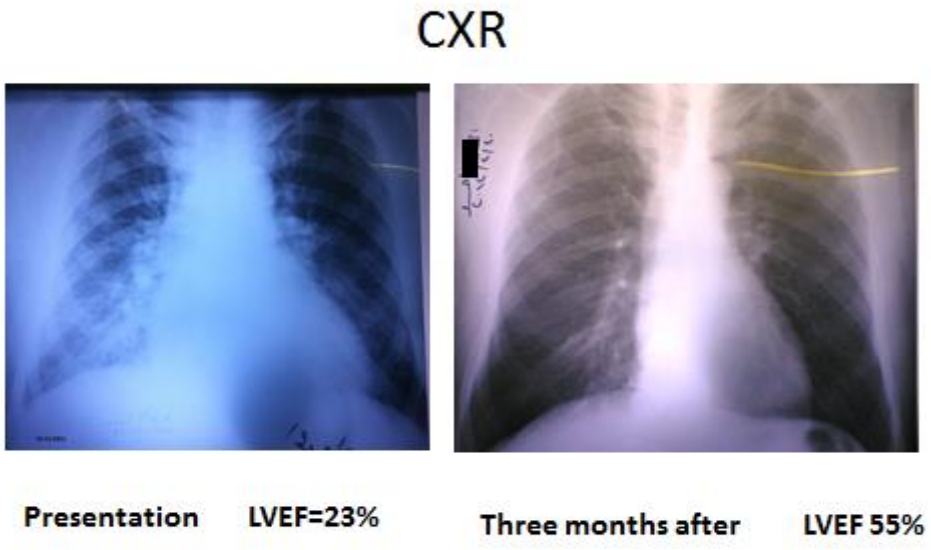

Figure 4C. Patient No. 2, chest X-ray showing improvement in cardiac size after arrhythmia control.

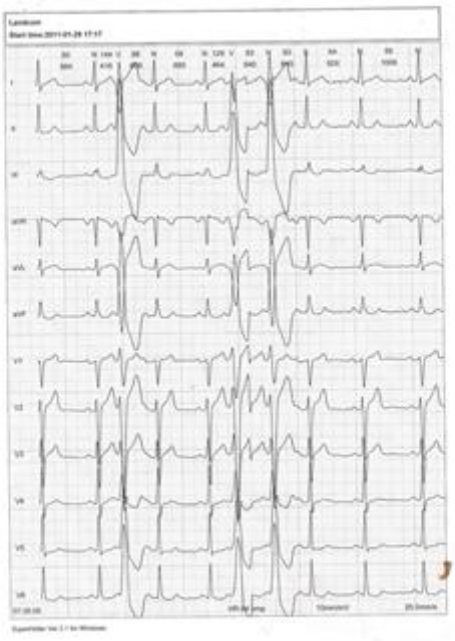

Figure 5A. Holter showing premature ventricular complexes.

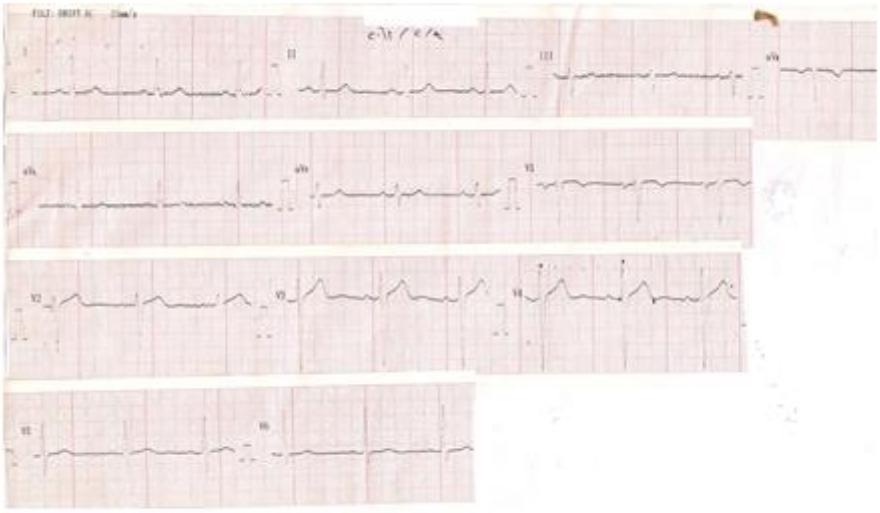

Figure 5B. ECG showing abolition of the VPCs.

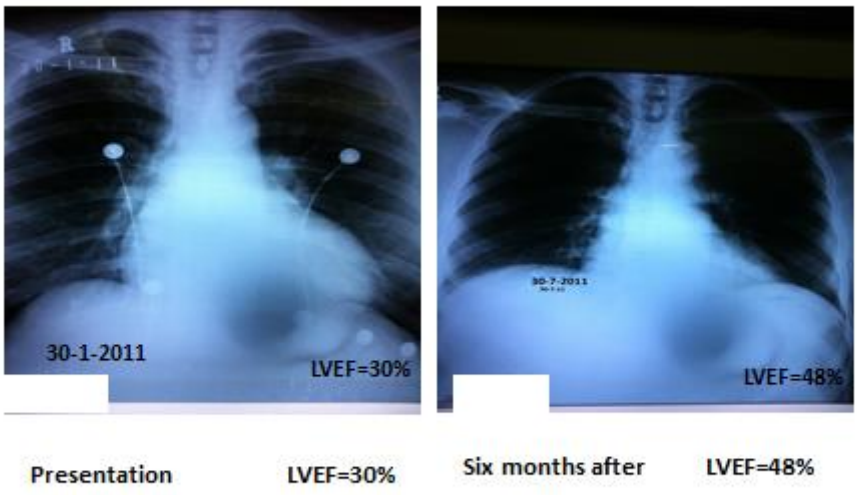

Figure 5C. Patient no. 3.CXR before and after control of VPCs.

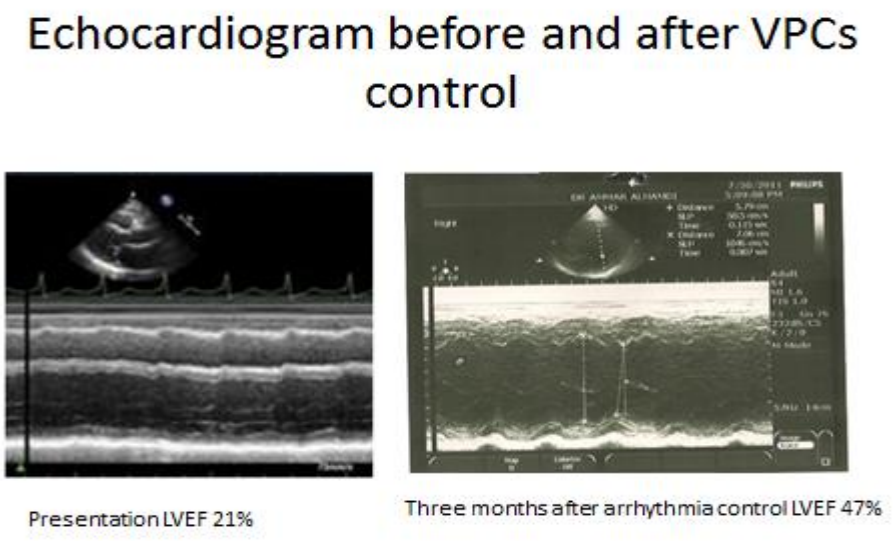

Figure 5D. Patient No. 4, echocardiogram before and after VPC control.

Discussion

Heart failure is a major health problem all over the world, and its prevalence is increasing, emphasizing the value of diagnosing potentially reversible etiologies.[1-6,14] Dilated cardiomyopathy is one major cause of heart failure in the young and pediatric age group, and it impacts the long-term survival of HF patients.[2,3,4] In spite of all the progress in treating HF due to DCM, the mortality is still significantly high.[2,14] Tachycardia-induced cardiomyopathy (TIC) is one resolvable etiology of heart failure and DCM.[1-6,14] Accordingly, it is highly significant to recognize and not misdiagnose TIC. Identifying this entity and treating it accordingly via arrhythmia control will resolve the DCM and improve LV function.[1-6,14] Distinguishing the symptoms of palpitation preceding the SOB in the history of the patient with DCM is the first key to clinical suspicion that the tachycardia is inducing the DCM and HF, rather than associating arrhythmia with the heart failure. This was seen in the 20 GrA patients in this series. Still, the feeling of palpitation within or after the SOB does not exclude arrhythmia as a precipitating factor of the HF and the DCM, as in GrB & C. The prognosis of the HF and DCM in all three groups looks excellent after controlling the arrhythmia. Rate control might be good enough to help resolve the DCM in TIC patients, but rhythm control by stabilizing the sinus rhythm is far more effective for resolving the DCM. [2,3,5] In this study, we relied on sinus rhythm stabilization as an end point in the arrhythmia control. Since the first recognition of TIC in 1913, a huge number of case reports and studies on animals and humans has shown the effectiveness of DCM induction and LVEF reduction in animals paced artificially for a long time or in humans developing

tachyarrhythmias of different modalities for variable intervals and rates and in humans with long-standing right ventricular apical pacing for variable degrees of heart block.[1-6,11] In spite of these many studies clearly identifying TIC as the most common etiology of reversible DCM and HF, we still face many missed cases of DCM that managed as idiopathic and the associated arrhythmia is either missed or considered as an associate and not as the underlying major etiology of DCM and HF. The ventricular dysfunction recovery in TIC patients following SR restoration varies depending on the arrhythmia's rate and duration.[1-6] Many studies show that the greatest recovery of LV function is seen one month after SR stabilization and then continues improving, reaching complete resolution after up to one year.[2-8] These findings are comparable to our series. Those recovered TIC patients with recurrent arrhythmia had a rapidly declining LVEF, heart failure and a high incidence of sudden cardiac death.[2,15] The pathogenesis of TIC has been hypothesized as resulting from a) an alteration in the cellular metabolism in the setting of a mitochondrial injury with increased Krebs cycle activity, b) increased level of oxidative stress that have been shown to accompany higher degrees of myocyte apoptosis in animal models of TIC, c) the downregulation of beta-adrenergic receptors and a resultant decreased sympathetic responsiveness in TIC and d) abnormal calcium handling that has been shown to play a role in TIC. [16] Recognizing that many of these changes are not specific for to TIC and are seen in other forms of heart failure is important. Changes seen early after initiating rapid pacing are more likely related to a fast heart rate in arrhythmias and cardiac pacing, whereas later changes are more likely due to a combination of the arrhythmia and the downstream of the HF syndrome.[2,16]

Conclusion

Tachycardia-induced cardiomyopathy is a reversible etiology of DCM and HF. TIC should be considered in patients with DCM of unknown etiology and in those with tachycardias. Abolishing the tachycardia with anti-arythmic drugs or **CRFAT** often results in clinical improvement and LV function recovery.

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اعتلال عضلة القلب الناجم عن عدم انتظام دقات القلب، وهو تشخيص منسي في العالم الحقيقي:

مراجعة لسلسلة من الحالات

الخلفية: اختلال وظيفي البطين الأيسر الناجم عن عدم انتظام ضربات القلب المتكررة منذ فترة طويلة ، والمعروف باسم اعتلال عضلة القلب الناجم عن عدم انتظام دقات القلب ، هو نوع قابل للعكس من اعتلال عضلة القلب المتوسعة وفشل القلب الذي يتميز بفشل البطين الأيسر الذي يمكن استرداده عادة بمجرد السيطرة على عدم انتظام ضربات القلب. أو عن طريق التحكم في معدل ضربات القلب.

المرضى والطرق: تم تضمين أربعة وعشرين مريضا يعانون من قصور في القلب بسبب اعتلال عضلة القلب المتوسعة وعدم انتظام ضربات القلب. تم علاج اضطراب نظم القلب وفشل القلب وفقًا لذلك. تم إجراء الفصل الوظيفي لجمعية نيويورك للقلب، وتقييم جزء طرد البطين الأيسر والأشعة السينية للصدر بشكل دوري لتقييم التحسن.

النتائج: تم تحقيق استقرار إيقاع الجيوب الأنفية في جميع المرضى. تحسنت فئة NYHA بشكل ملحوظ ، من الدرجة الثالثة إلى الرابعة إلى الدرجة الأولى في جميع المرضى. ارتفع جزء طرد البطين الأيسر بشكل ملحوظ من 20-30٪ إلى 45-57٪. تحسن حجم القلب بشكل ملحوظ إشعاعيًا. أعراض الخفقان المتكررة طويلة الأمد التي تسبق أعراض ضيق التنفس في قصور القلب هي أول دلالة على أن عدم انتظام ضربات القلب يحفز قصور القلب الذي لوحظ في 20 مريضًا.

الخلاصة: اعتلال عضلة القلب الناجم عن عدم انتظام دقات القلب هو سبب قابل للعكس لفشل القلب. السيطرة على عدم انتظام ضربات القلب يستعيد وظيفة LV العادية.

الكلمات المفتاحية: عدم انتظام دقات القلب، اعتلال عضلة القلب