Acute Tachycardia-Induced Cardiomyopathy: A Case Report

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Conflict of interest: None declared

Patient: Female, 88-year-old
Final Diagnosis: Acute tachycardia induced cardiomyopathy
Symptoms: Dyspnea
Medication: —
Clinical Procedure: Cardiac catheterization • cardiac MRI • cardioversion
Specialty: Cardiology

Objective: Unknown etiology

Background: Tachycardia from atrial fibrillation or flutter can lead to left ventricular systolic dysfunction. Some patients deteriorate quickly, and there is an acute drop in their left ventricular systolic function; however, they tend to normalize rapidly after treatment of the underlying arrhythmia. The aim of publishing the present case is to maintain awareness that tachycardia is one of the etiologies of acute systolic heart failure, which is potentially reversible by treatment when recognized.

Case Report: An 88-year-old woman with a history of hypertension and diabetes presented to the emergency department with shortness of breath and new-onset atrial fibrillation. The physical examination revealed jugular vein distention, an irregular heart rate of approximately 140 beats/min, bilateral basal lung crackles, and no murmurs. One week before this presentation, she underwent electrocardiography, which showed she was in sinus rhythm, and transthoracic echocardiography, which indicated an ejection fraction of 65%. After hospital admission, she was started on beta-blockers for heart rate control and diuretics for heart failure management. As her symptoms persisted, she underwent a transesophageal echocardiography-guided cardioversion, where her ejection fraction was 30%. A repeat transthoracic echocardiography 3 days after the cardioversion indicated the ejection fraction had normalized to 60%. She was followed up every month in the Outpatient Cardiology Clinic and has remained asymptomatic for 1 year to date.

Conclusions: Although most literature describes tachycardia-induced cardiomyopathy as a chronic process, it can be acute. Patients benefit from rhythm control, and with early diagnosis and appropriate management, the prognosis is good.

Keywords: Atrial Fibrillation • Heart Failure • Tachycardia

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/930732
Background

Tachycardia-induced cardiomyopathy (TICM) is a rare cause of dilated cardiomyopathy [1]. Once suspected, the challenge is to determine whether tachycardia is the primary cause of the cardiomyopathy or secondary to the cardiomyopathy [2]. TICM usually results from prolonged periods of tachycardia; its duration is an indicator that can determine tachycardia as the primary cause of TICM [2]. Chronic tachycardia can cause structural and cellular morphological changes in the heart that eventually lead to overt heart failure and systolic dysfunction, although the precise mechanisms involved are poorly understood [1-5]. Some patients seen in our daily practice exhibit atrial fibrillation then quickly deteriorate, and their left ventricular functions drop rapidly. This quickly reverts following treatment of the underlying arrhythmia. We describe a case of acute and rapidly reversible TICM.

Case Report

An 88-year-old woman with a history of hypertension, hyperlipidemia, and diabetes presented at the hospital reporting progressive shortness of breath for the past 3 days and irregular palpitations. Initially, the shortness of breath was exertional and now it had progressed to occurring when she was at rest. She reported orthopnea and paroxysmal nocturnal dyspnea without any chest pain.

A physical examination revealed jugular vein distention, an irregular heart rate of approximately 140 beats/min, bilateral basal lung crackles, and no murmurs. Electrocardiography indicated atrial fibrillation with a rapid ventricular response and a heart rate of approximately 140 beats/min (Figure 1). Her charts indicated that she had been in sinus rhythm 1 week before the current presentation, as determined by electrocardiography during a physical examination performed at the clinic at that visit.

Laboratory tests were unremarkable, including negative troponin levels. Her brain natriuretic peptide level was 1500 pg/mL. She was started on heart rate controlling medications with beta-blockers and diuretics for heart failure management. However, as her symptoms persisted, a transesophageal echocardiography-guided cardioversion was scheduled. During that procedure, her ejection fraction (EF) was 30% with preserved left ventricular internal dimensions, global hypokinesia, no specific segmental wall motion abnormality, no right ventricular dysfunction, and no new valvular regurgitation. However, she had a normal EF of 65% on the transthoracic echocardiography performed 1 week prior to presentation at the hospital.

A full cardiac workup (including a coronary angiogram and magnetic resonance imaging) was performed because of the acute left ventricular systolic dysfunction; it depicted normal coronary arteries. Cardiac magnetic resonance imaging was performed to rule out acute myocarditis, and it was negative for evidence of myocarditis, infiltrative pathology, and cardiac scarring. Transthoracic echocardiography was repeated 3

![Figure 1. Twelve-lead surface electrocardiogram showing atrial fibrillation with a rapid ventricular response, estimated rate approximately 140 beats/minute, and no evidence of bundle branch block or ST-segment changes.](Image)
days later, and it indicated normalization of the EF to 60% after the heart rate was controlled and sinus rhythm was restored. Strain analysis was not performed as it does not have additional value in the diagnosis or management. The reasons for her development of acute left ventricular dysfunction with atrial fibrillation of such short duration remain unresolved. She was followed closely every month at the Outpatient Cardiology Clinic and has remained asymptomatic on beta-blockers and apixaban for 1 year to date.

The patient gave informed verbal consent for the publication of this case report, and all study material has been de-identified. As no clinical trial was carried out and the patient received standard care management, approval from the Institutional Review Board was not deemed necessary.

**Discussion**

TICM is a relatively rare cause of dilated cardiomyopathy [1]. It results from prolonged periods of increased heart rate [2]. It is well known that chronic tachycardia can cause structural and cellular morphological changes in the heart, although the precise mechanisms of these remain poorly understood [1-5]. The diagnosis of TICM is usually made after the successful trialing of therapy to reduce the ventricular rate and restore sinus rhythm together with the exclusion of other causes of left ventricular dysfunction [6]. The treatment for TICM is the same as that for heart failure with a reduced EF, with a particular focus on heart rate control with or without sinus rhythm restoration [6].

Heart rate vs rhythm control for atrial fibrillation remains a controversy. Our current approach is to lean towards rhythm control when rate control cannot be achieved via standard medications, and the patient remains symptomatic despite the heart rate control. Recently, rhythm control has been adopted more frequently because of its increased benefit and association with a decrease in adverse cardiovascular events, according to the EAST-AFNET 4 trial [7].

As seen with the current patient, some patients cannot tolerate atrial fibrillation and rapidly develop systolic left ventricular dysfunction. This has been reported infrequently in the literature, and research is needed to accurately determine the types of patients that are likely to develop acute TICM. The patient in the present case developed TICM in <1 week, and her EF quickly recovered after achieving heart rate control and sinus rhythm restoration. In order to determine that tachycardia was the primary cause of her cardiomyopathy rather than cardiomyopathy having caused secondary tachycardia, other etiologies, including acute coronary syndrome and acute myocarditis, had to be ruled out via coronary angiography and cardiac magnetic resonance imaging, respectively. The duration of the tachycardia was short, given the evidence of sinus rhythm and normal systolic function only 1 week earlier. The rapid improvement of the left ventricular systolic functions ultimately confirmed that tachycardia was the primary cause of the acute left ventricular systolic dysfunction.

This case report expands the scope of the EAST-AFNET4 trial results and is concordant with the fact that rhythm control in early atrial fibrillation is associated with improved cardiovascular outcomes. With regards to the present case, it is uncertain whether heart rate control would have yielded the same results; the decision to pursue cardioversion was made because she was symptomatic. Whether these patients have acute stress cardiomyopathy derived from tachycardia rather than acute TICM requires further investigation; this would be difficult to prove, and regardless of the outcome, the treatment would be the same.

**Conclusions**

Despite most of the literature describing TICM as a chronic process, it can be acute. These patients benefit from rhythm control, and with early diagnosis and appropriate treatment, the prognosis is usually good.

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**Department and Institution Where Work Was Done**

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**Conflicts of Interest**

None.
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