



## Case Report

## An unexpected cause of chronic cough in a young woman: Tachycardia-induced cardiomyopathy

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## ABSTRACT

Heart failure typically manifests with exertional dyspnea or limb edema. Old age, underlying heart disease, and coronary risk factors are known risk factors for heart failure. Thus, congestive heart failure is rarely considered as a differential diagnosis of chronic cough in a previously healthy young person. However, tachycardia-induced cardiomyopathy (TIC) can result in such a situation. A case of a previously healthy 27-year-old woman with TIC is described herein. On the first examination, the patient presented with left ventricular (LV) dysfunction caused by persistent tachyarrhythmia. Cardiomyopathy subsided with only heart rate control; therefore, the patient was diagnosed as having TIC. TIC can develop irrespective of age and underlying heart disease. The long-term outcome may be good, and LV systolic function recovers in most patients following resolution of arrhythmia. Physicians need to consider TIC as a differential diagnosis in patients of any age who present with heart failure, because prompt treatment generally leads to resolution of symptoms and ventricular function.

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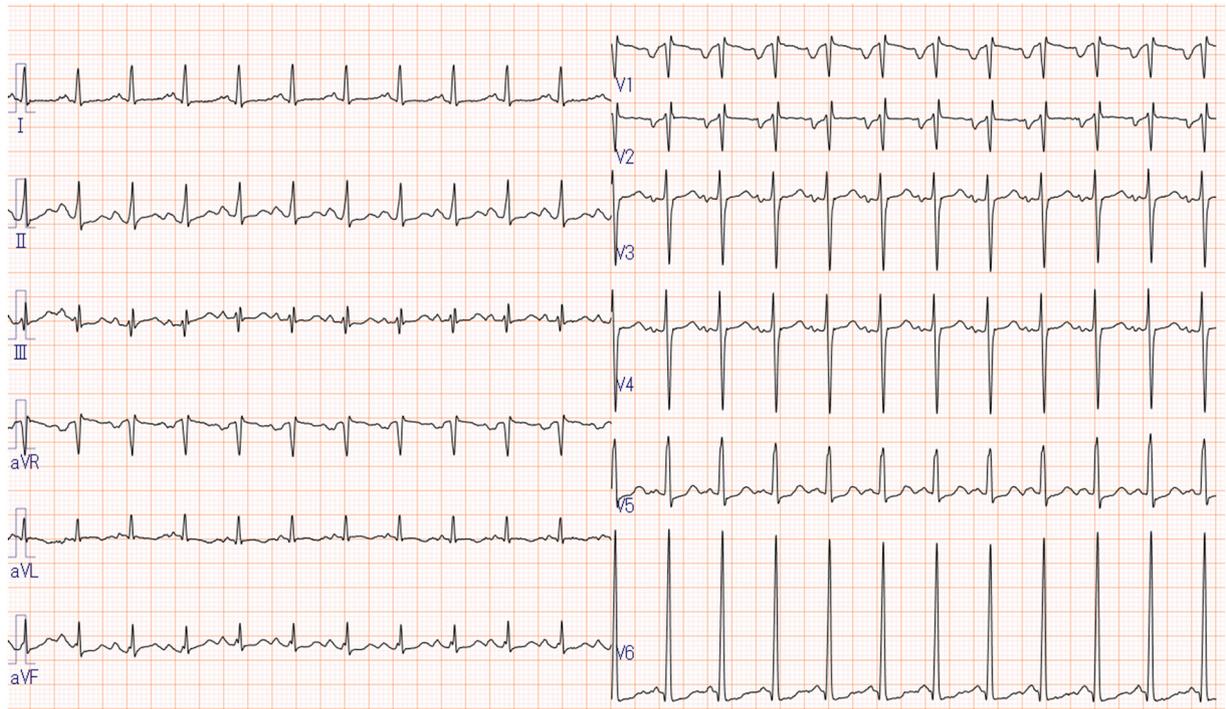
On the first examination, a previously healthy 27-year-old woman presented with a 2-month history of cough and progressive exertional dyspnea. Seven days before this visit, she also experienced general fatigue and anorexia, followed by fever and lower-limb edema. These symptoms gradually became exacerbated so she finally visited us for medical advice. She had no significant underlying diseases and was not pregnant. On arrival, the vital signs were as follows: temperature of 38.0 °C, heart rate of 135 beats per minute, respiratory rate of 32 breaths per minute, saturation of percutaneous oxygen of 97% on ambient air, and blood pressure of 167/114 mm Hg. The physical examination revealed jugular vein engorgement, crackles in the right lower chest, gallop sounds, and bilateral lower-extremity edema. There was no thyromegaly or proptosis. Laboratory examinations showed an elevated white blood cell count of 10,900/ $\mu$ L and B-type natriuretic peptide level of 1308.3 pg/mL (normal range: <18.4 pg/mL) with a normal hemoglobin of 11.6 g/dL (reference range: 11.6–14.8), hemoglobin A1c (HbA1c) of 5.8% (reference range: <6.4%), and creatinine kinase level of 99 U/L (reference range: 41–153). The result of the thyroid function test was normal. Electrocardiography showed sinus tachycardia with

left ventricular (LV) hypertrophy and no change in the ST-T segment (Fig. 1). The chest radiograph indicated cardiomegaly (Fig. 2A). The transthoracic echocardiogram (TTE) showed LV wall motion with diffuse severe hypokinesia (LV end-diastolic diameter [LVEDD] of 48 mm, LV end-systolic diameter [LVESD] of 45 mm) and LV ejection fraction (LVEF) of 0.20, moderate mitral regurgitation, and moderate tricuspid regurgitation. Based on a diagnosis of cardiac failure, furosemide, carperitide (a recombinant alpha-human atrial natriuretic peptide), nitroglycerin, and digoxin were initiated as treatment. Signs of cardiac failure gradually improved on hospital day 5, and inflammatory indices including body temperature normalized without specific treatment. We added bisoprolol fumarate to the treatment regimen on hospital day 12 after the patient's volume status had normalized. Cardiomegaly detected on the chest radiograph subsided (Fig. 2B), and TTE revealed normalization of the LV wall motion (no asynergy, LVEDD of 45 mm, LVEDs of 32 mm, and LVEF of 0.53) on hospital day 14. Results of the endomyocardial biopsy, which was intended to rule out dilated cardiomyopathy or myocarditis, revealed a failed myocardium without histological evidence of inflammation. Given that cardiomyopathy subsided with only heart rate control, a diagnosis of tachycardia-induced cardiomyopathy (TIC) was made. Re-examination of thyroid function revealed thyrotoxicosis (free tetraiodothyronine, 1.79 ng/dL [reference range: 0.75–1.75]; thyrotropic-stimulating hormone [TSH], <0.01  $\mu$ U/mL [reference range: 0.35–4.00]) with a high titer of the TSH receptor antibody, suggesting the onset of Graves' disease. Antithyroid therapy was initiated on hospital day 15, and she was discharged on the same day. One month later, the repeat TTE showed a normalized LVEF of 0.71.

*Abbreviations:* TIC, tachycardia-induced cardiomyopathy; LV, left ventricular; SpO<sub>2</sub>, saturation of percutaneous oxygen; TTE, transthoracic echocardiogram; LVEDd, left ventricular end-diastolic diameter; LVEDs, left ventricular end-systolic diameter; LVEF, left ventricular ejection fraction; T<sub>4</sub>, tetraiodothyronine; TSH, thyrotropic-stimulating hormone.

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**Fig. 1.** An electrocardiogram showing sinus tachycardia with left ventricular hypertrophy.

TIC develops over months to years, following persistent supraventricular or ventricular tachyarrhythmias. The manifestations of TIC include symptoms related to tachyarrhythmia, heart failure, or both. Myocardial dysfunction, characterized by ventricular systolic dysfunction and dilation, can be due to tachycardia alone (pure TIC) or due to the exacerbation of ventricular dysfunction in a patient with an underlying heart disease (impure TIC). Signs and symptoms of heart failure are generally reversible with normalization of heart rate in most patients, especially if the tachycardia is transient [1]. The prevalence of TIC is unknown. TIC may be rare and occur at any age. Generally, chronic cough and progressive dyspnea are rare symptoms of congestive heart failure and LV failure in a previously healthy young person [2,3]. However, TIC can result in such situations.

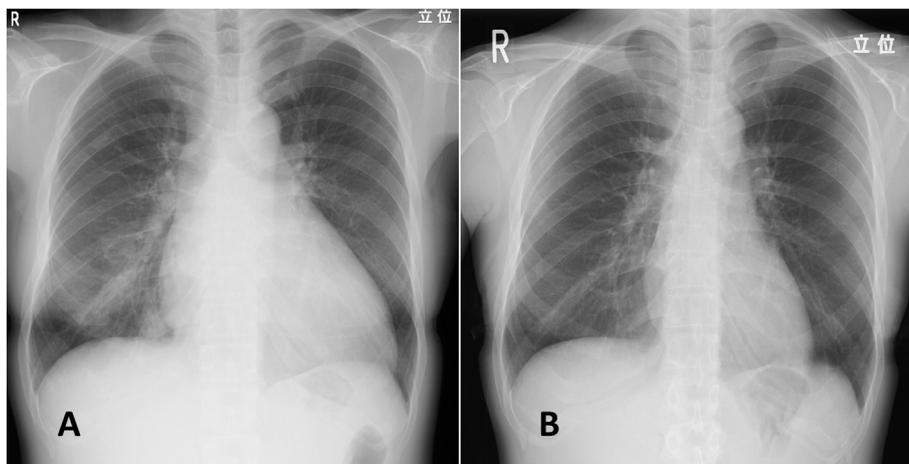
TIC is associated with most types of tachyarrhythmias and frequent ectopy. It is unclear why the initial result of the thyroid function test was normal in this patient—we suppose this case may be atypical on this point—but an association with sinus tachycardia, as seen in our

case, is extremely rare [4]. Approximately 6% of patients with thyrotoxicosis develop symptoms of heart failure, and only 1% develop TIC with a reduced LV systolic function [5]. Most patients recover their LV systolic function after tachycardia is controlled and a euthyroid state is achieved [5]. Therefore, TIC is an important etiology of heart failure that is curable. The long-term outcome may be good following the resolution of arrhythmia. However, caution is needed because sudden cardiac death has been reported even after the signs and symptoms have subsided [6].

TIC should be suspected in any patient of any age presenting with heart failure, even when the symptoms are not outstanding, because early recognition and prompt treatment result in the resolution of symptoms and ventricular function.

#### Declarations of interest

None to declare.



**Fig. 2.** (A) A chest radiograph showing cardiomegaly. (B) A chest radiograph showing improvement in cardiomegaly.

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