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Case Report

Complete His-ventricular block, atrial flutter and ventricular tachycardia as arrhythmogenic activities in a patient with takotsubo cardiomyopathy

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ABSTRACT

An association of atrial arrhythmias with takotsubo cardiomyopathy (TTC) has not been described previously. Here we report a 65-year-old male patient with TTC. The sudden appearance of atrioventricular block and subsequent bradycardia are believed to be key contributing factors for the development of TTC. Both ventricular tachyarrhythmia and various atrial arrhythmias, such as atrial flutter and atrial fibrillation, were observed during the initial management of the patient's TTC. We speculate that both the left ventricular contractile dysfunction and the arrhythmogenic activities may share a common underlying etiology in advanced heart failure patients with TTC.
<**Learning objective:** We describe a case of TTC complicated by ventricular tachycardia, atrial tachyarrhythmias, and an atrioventricular conduction disturbance and discuss the etiology of arrhythmogenic activities in TTC.>

Introduction

Takotsubo cardiomyopathy (TTC) is a specific disorder characterized by reversible, nonischemic, left-ventricular apical ballooning [1]. Although the precise etiology of TTC is unclear, various exogenous and endogenous stresses are suspected [1]. There are few case reports that describe the association between atrial arrhythmias and TCC. Here we report a case of heart failure with advanced TTC in which ventricular tachycardia (VT), complete His-ventricular (HV) block, and atrial flutter were associated with arrhythmogenic manifestations.

Case report

A 65-year-old male patient was transferred to our hospital because of the sudden appearance of dyspnea. He suffered from mild intellectual disability, but it did not interfere with routine daily life, and there was no emotional or physical stress. He was well before he experienced chest dyspnea. His symptoms worsened and his family physician recommended that he undergo further examination for acute heart failure. On admission, his chest radiograph showed both bilateral pulmonary congestion and cardiomegaly with a cardiothoracic ratio of 61%. Ambulatory monitor electrocardiogram showed torsades de pointes-type VT with a mean cycle length of 240 ms (Fig. 1). The patient's hemodynamics collapsed during VT, but VT was terminated by transient cardio-pulmonary resuscitation without electrical cardioversion. The electrocardiogram displayed a regular junctional rhythm with a heart rate of 45 beats/min, atrial flutter, and a prolongation of QT interval (QTc: 693 ms) with a giant negative

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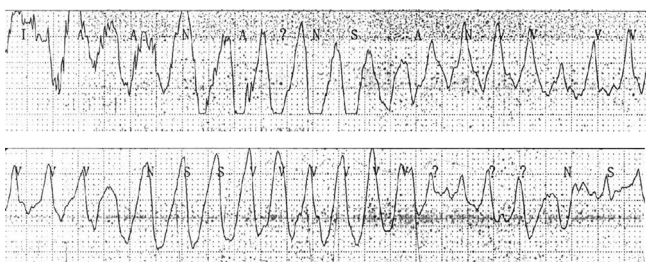

Fig. 1. Documentation of torsades de pointes-type ventricular tachycardia in an ambulatory monitor recording.

T wave in the precordial leads (Fig. 2). The laboratory data disclosed an elevated serum level of N-terminal pro-B-type natriuretic peptide (969.4 pg/mL). The serum levels of myocardial enzymes were within the normal range. The serum level of troponin I was also within the normal range (0.164 ng/mL). The ultrasound echocardiogram showed extensive akinesis of the left ventricular wall. Mechanical ventilation and temporary pacing were initiated to maintain appropriate hemodynamics. An emergent cardiac catheterization study was performed to exclude acute coronary syndrome. Coronary arteriography was normal, but left ventriculography showed systolic apical ballooning (Fig. 3). Based on these findings, the patient was diagnosed with TTC. Intravenous furosemide and carperitide were administered at the coronary care unit, and his systemic condition gradually improved.

An electrophysiological study performed 8 days after admission consistently confirmed both complete HV block and uncommon atrial flutter (Fig. 4). Post-pacing interval was identical to that of the tachycardia cycle lengths measured at the coronary sinus orifice, septal isthmus, and low lateral right atrium. The EnSite NavX™ (St. Jude Medical, St. Paul, MN, USA) electroanatomical mapping system showed clockwise rotation around the tricuspid annuls during atrial tachycardia. An implantable cardioverter–defibrillator (ICD) was implanted for backup pacing and for secondary prevention of VT after ablation of the cavotricuspid isthmus. The clinical course was uneventful; both the left ventricular dysfunction and the QT prolongation had returned to their normal range 6 months after discharge (Fig. 5). However, the atrioventricular block persisted, and paroxysmal atrial fibrillation was observed (Fig. 6). Aside from being consequent to advanced age, the precise cause of the HV block could not be determined, because the patient's previous history, laboratory data, and ultrasound echocardiogram did not indicate the presence of cardiac sarcoidosis, myocarditis, or non-ischemic myocardial disease, although an endocardial biopsy was not obtained.

Discussion

Many patients with TTC suffer from emotional and physical stress [1]. The clinical course of TTC is basically benign if a precise diagnosis is made and appropriate conservative treatment is initiated [1]. The prognosis of TTC depends primarily on the generation of ventricular tachyarrhythmias and/or contractile dysfunction [1]. Kurisu et al. [2] reported two cases of TTC initiated by advanced atrioventricular block and subsequent bradycardia. In the current case, an HV block-based bradycardia emerged initially, and was then followed by the appearance of bradycardia-oriented TTC.

It is interesting to note that three types of critical arrhythmia coexisted in a single TTC patient. VT probably developed as a result of advanced QT prolongation, a common electrical anomaly in TTC, and cardiopulmonary resuscitation was required for VT termination. The TTC patient's atrial flutter is rare, but could occur sometimes. The rate of its association with TTC was 1.9% as Pant and colleagues [3] suggested in their recent review of TCC. Lastly, the patient exhibited a complete HV block leading to subsequent

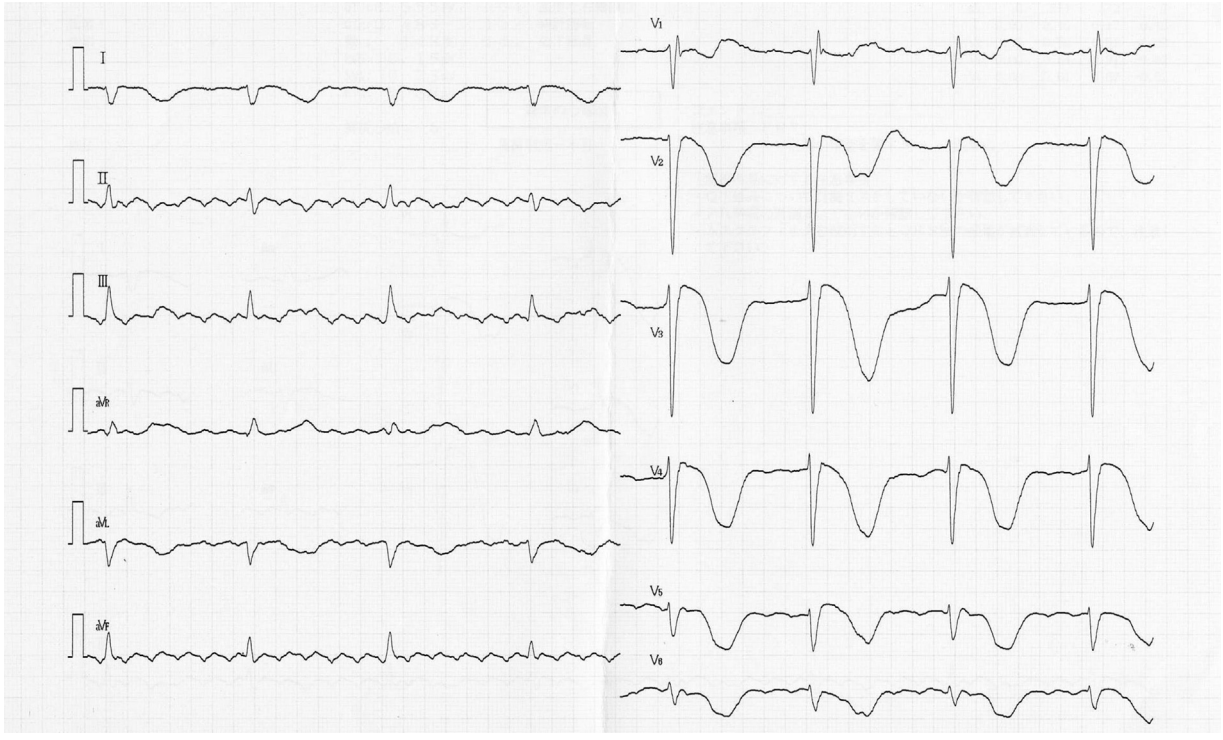

Fig. 2. Twelve-lead electrocardiogram on admission. Serrated negative P′ wave in leads II, III, and aVF with a junctional regular rhythm of 45 beats per minute is demonstrated. Note the giant negative T wave on long QT interval (QTc; 693 ms) in precordial leads.