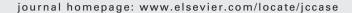


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Case Report

Recurrent takotsubo cardiomyopathy with variant forms of left ventricular dysfunction

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KEYWORDS

Apical ballooning; Heart failure; Invert; Stress; Takotsubo Summary A 78-year-old man presented to our emergency department with dyspnea. The patient was diagnosed as having pneumonia from the chest X-ray which depicted mass-like opacity in the left lower lobe. On the 5th hospital day, electrocardiography showed giant negative T waves in pericardial leads and echocardiography demonstrated left ventricular apical akinesis and basal hyperkinesis. Accordingly, the patient was retrospectively diagnosed as having typical takotsubo cardiomyopathy. Two years later, the patient was admitted again to our hospital with pneumonia. On the 2nd hospital day, echocardiography showed left ventricular basal and mid-ventricular akinesis combined with normal apical wall motion. Ventricular wall motion was normalized within two months. The patient was finally diagnosed as having inverted takotsubo cardiomyopathy. Here, we report the patient who had recurrent takotsubo cardiomyopathy with variant forms of left ventricular dysfunction caused by repeated physical stress in two years.

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Introduction

Takotsubo cardiomyopathy is recognized as reversible ventricular dysfunction and characterized by transient left ventricular (LV) apical ballooning and electrocardiographic (ECG) changes that mimic acute myocardial infarction in the absence of obstructive coronary artery disease [1]. Various types of transient ventricular dysfunction have been

reported [1]. The mechanism of takotsubo cardiomyopathy is still a matter of debate, which varies according to the individual. Recurrent takotsubo cardiomyopathy is rare; only a handful of case reports have described the typical and atypical ventricular contractile patterns in the same patients [2]. Here, we report our encounter with recurrent takotsubo cardiomyopathy with variant forms of LV dysfunction which was caused by repeated physical stress.

Case report

A 78-year-old man presented to our emergency department with dyspnea. On admission, the pulse was 120 beats/min,

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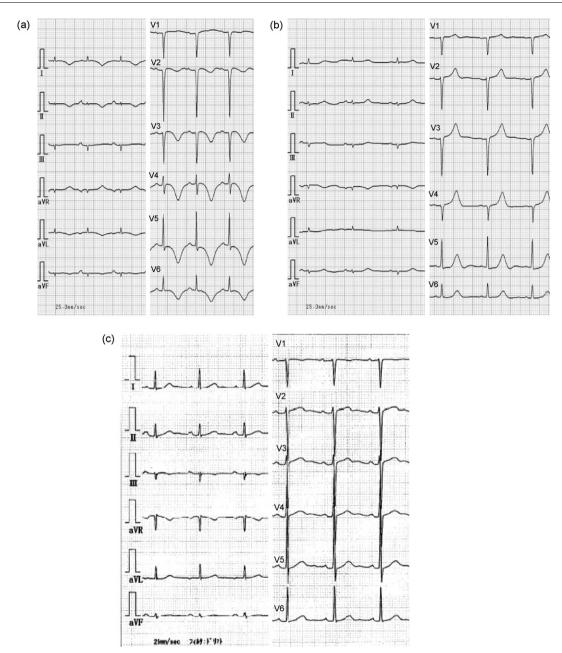


Figure 1 12-lead electrocardiogram. (a) On the 5th hospital day on the first admission, sinus rhythm (91 beats/min) with inverted T waves in V2-6 and abnormal Q in III, aVF, V1-3 was observed. (b) On the 1st day of the 2nd admission, sinus rhythm (64 beats/min) with abnormal Q wave formations in V1-4 and low voltage in limb leads was observed. (c) Before 1st admission, sinus rhythm (72 beats/min) with high voltage in V5, 6.

the blood pressure was 114/69 mmHg, and the body temperature was 37.0 °C. The laboratory data showed leukocytes of 6200×10^9 /L, hemoglobin of $10.9\,\mathrm{g/dL}$, platelets of 234×10^9 /L, creatine kinase of $133\,\mathrm{U/L}$, and C-reactive protein of 21.3 mg/dL. Chest X-ray showed mass-like opacity in the left lower lobe; the patient was diagnosed as having pneumonia and immediately treated with antibiotic therapy. On the 5th hospital day, 12-lead ECG showed sinus rhythm (91 beats/min) with inverted T waves in leads V2 through V6 and abnormal Q formations in leads V1 through V3 (Fig. 1a). Echocardiography depicted LV akinesis except in

the basal region; however, no pericardial effusion, LV hypertrophy, or thrombus was found (Fig. 2a). Multi-detector computed tomography showed no significant obstruction in the coronary arteries. Accordingly, the patient was diagnosed as having takotsubo cardiomyopathy. On the 21st hospital day, ¹²³I-metaiodobenzylguanidine (MIBG) myocardial scintigraphy showed decreased uptake in the apical area (Fig. 3a). The defect area was accentuated in the delayed image; the accelerated washout of ¹²³I-MIBG was observed in the apical area. The heart to mediastinum (H/M) ratio remarkably decreased both in the early (1.29) and delayed

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