Case Report

Nightmare-Induced Atypical Midventricular Tako-Tsubo Cardiomyopathy

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1. Introduction

Tako-Tsubo cardiomyopathy (TTC), also called broken heart syndrome or apical ballooning, is a reversible cardiomyopathy characterized by acute left ventricular (LV) segmental dysfunction, often precipitated by a psychophysical stressful event, whose clinical presentation mimics that of acute myocardial infarction [1]. TTC most commonly occurs in women, particularly in the postmenopausal period, and is estimated to represent about 1-2% of subjects presenting with troponin-positive acute coronary syndrome [2]. The classic pattern of TTC is characterized by akinesia of the mid-apical segments of LV walls, often with hyperkinesia of the basal segments. The resulting shape of LV cavity typically shows a round bottom and narrow neck, resembling that of a traditional Japanese octopus trap called “Tako-Tsubo.” Atypical clinical and echocardiographic patterns of TTC in which the diagnosis may be difficult to make, often leading to misdiagnosis, have recently been described [3]. Although several types of triggering event have been previously described, identification of the stressor may sometimes be challenging, as up to one-third of TTC patients present with no clear evidence of definite stressful event [4]. In this report, we describe a case of TTC with atypical clinical and echocardiographic features, which was triggered by an unusual stressor, that is, a recurrent nightmare.

2. Case Report

A 45-year-old woman was urgently brought to the Emergency Department because of persistent loss of consciousness. Her history was unremarkable, except for anxiety-depressive disorder. She had no cardiovascular risk factors except for current cigarette smoking, and she was not assuming any type of cardiovascular or noncardiovascular medication. Two hours earlier, she had phoned the Medical Emergency Team because of general discomfort and intense anxiety. She had been administered benzodiazepines, but the discomfort had worsened with progressive neurological deterioration. At admission, chest and heart examination was normal, blood pressure was 100/70 mmHg, and heart rate was 70 bpm. On neurological examination, there were spontaneous eye opening, absence of verbal response, and withdrawal from
painful stimuli (Glasgow coma scale 9), arm and leg extension, lock jaw, hypersalivation, and diffuse hyporeflexia. The electrocardiogram showed sinus rhythm, a nonsignificant (<1 mm) ST-elevation with ascending slope in right precordial limbs, with no evidence of mirror images, and a negative T wave in aVL (Figures 1(a) and 1(b)). Echocardiography showed normal left ventricular (LV) ejection fraction (55%) with akinesia of the middle segments of ventricular septum and both anterior and inferior walls (Figure 2, Movie 1 in Supplementary Material available online at http://dx.doi.org/10.1155/2015/292658). Blood examinations showed increased levels of troponin I (1.99 ng/mL, n.v. < 0.09 ng/mL) and D-dimer (4069 ng/mL, n.v. < 500 ng/mL) and leukocytosis (11.0 × 10³/µL). Arterial blood gas analysis showed mild compensated metabolic acidosis with hyperlactatemia (7.4 mmol/L, n.v. 0.3–0.8 mmol/L). During the stay in the Emergency Department, the patient developed convulsive seizures, which were treated with i.v. diazepam. An electroencephalogram and brain computer tomography showed normal findings. The patient was transferred to the Intensive Care Unit with the diagnosis of acute coronary syndrome. Treatment with aspirin, clopidogrel, fondaparinux, atorvastatin, and pantoprazol was started. Low-dosage metoprolol was started on day 2 but was discontinued the day after because of low blood pressure. Because the patient showed preserved LV ejection fraction, no symptoms or signs of heart failure, and a tendency toward hypotension, we also did not start ACE-inhibitors, angiotensin receptor blockers, or diuretics.

Few hours later, progressive recovery of consciousness was observed, with retrograde amnesia and a transient phase of behaviour abnormalities with histrionic issues. The day after, following full recovery of consciousness, the patient referred that during the previous five days she had experienced a recurrent nightmare with sudden awakenings characterized by severe anxiety, prolonged tachycardia, and sweating. Although she remembered the frightening experience associated with the nightmare, she was not able to remember its contents. An angiography showed normal coronary arteries (Figure 3). The electrocardiogram showed development of negative T waves in V1-V2. Blood examinations showed increased NT-proBNP plasma concentration (2514 pg/mL, n.v. < 125 pg/mL), a borderline level of C-reactive protein (0.52 mg/dL, n.v. < 0.50 mg/dL), and progressive normalization of troponin I. Patient's general conditions rapidly improved. Atypical midventricular TTC was diagnosed. She was discharged on aspirin 100 mg od, with the advice of continuing it indefinitely. On day 10, echocardiography showed improvement of LV systolic function with residual hypokinesia of the middle segments of the septum.
natural disasters such as earthquakes, tempests, and floods, car accidents, strenuous physical efforts, exacerbation of a chronic medical illness, newly diagnosed diseases, surgical interventions, hospital stay in an intensive care unit, seizure, migraine, asthmatic crisis, carbon monoxide poisoning, use of or withdrawal from illicit drugs, and suicide attempt [19–22].

To our knowledge, this is the first report of midventricular TTC triggered by a recurrent nightmare. Several atypical issues should be pointed out in this report: (1) the unusual type of stressor; (2) the clinical presentation, characterized by persistent loss of consciousness, neurological deterioration despite normality of electroencephalogram and brain computer tomography; and absence of typical symptoms of TTC; and (3) the early clinical evolution, which might suggest the typical pattern of an hysterical crisis, characterized by an aura followed by epileptoid, histrionic, and recovery phases. Although the loss of consciousness followed by retrograde amnesia cannot allow excluding the fact that our patient also experienced chest pain or dyspnea, it is interesting to observe that TTC triggered by emotional stressors, rather than physical, often tends to present without typical symptoms [23]. Another interesting issue is that seizures have been reported as causes of TTC [24]. In particular, it has been reported that patients with seizure-associated TTC tend to be younger, more frequently males, and often present with no chest pain but frequent cardiogenic shock and sudden hemodynamic deterioration [25]. Although most of these characteristics were not present in our patient, it cannot be excluded that the hysterical episode that occurred in our patient, and particularly the convulsive phase, may have played a role as an additional trigger.

**Conflict of Interests**

The authors declare that there is no conflict of interests in connection with this paper.

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