Tako-tsubo Cardiomyopathy Complicated by Recurrent Torsade de Pointes in a Patient with Anorexia Nervosa

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Abstract

We report the case of a 57-year-old woman with anorexia nervosa showing evidence of “tako-tsubo” cardiomyopathy complicated by several syncopes due to recurrent episodes of torsades de pointes. Prolongation of QT interval and QT dispersion have been reported both in the “tako-tsubo” cardiomyopathy and in anorexia nervosa. The QT prolongation and the QT dispersion has been linked as risk indicators for sudden cardiac death. The combination of “tako-tsubo” cardiomyopathy with a condition associated with the prolongation of QT and/or with an increase of QT dispersion, such as anorexia nervosa, makes the acute and subacute prognosis of this disease much more severe than usual.

Key words: tako-tsubo cardiomyopathy, anorexia nervosa, torsade de pointes, long QT syndrome, apical ballooning syndrome, QT dispersion


Introduction

“Tako-tsubo” cardiomyopathy (TTC), also known as “apical ballooning syndrome”, is a cardiac disease, usually triggered by emotional and/or physical stress, characterized by a transient left ventricular apical wall motion abnormality without epicardial coronary artery disease. The prognosis of this syndrome is generally good but the combination with clinical disorders associated with the prolongation of QT interval and/or with increase of QT dispersion (QTd) makes the acute and subacute prognosis much more severe than usual. Here, we present a case of a 57-year-old woman with anorexia nervosa (AN) showing evidence of TTC. The prolongation of QT and QTd, risk indicators for sudden death, have been reported both in TTC and in AN. For this reason, the present patient experienced several syncopes due to recurrent episodes of torsades de pointes.

Case Report

A 57-year-old woman was transported to the emergency department of our hospital because of severe chest pain at rest and syncope, which occurred 12 hours after a dispute with her relatives. She had a history of AN and, at that time, was not taking any medication. Her family history was negative for sudden death. On examination, the patient was malnourished, with a body max index (BMI) of 13 kg/m², heart rate 75 beats per minute (bpm), blood pressure 75/40 mmHg and oxygen saturation 90%.

Laboratory tests showed hypokalemia (3.4 mEq/L; nv = 3.5-5.5 mEq/L), hypomagnesemia (1.0 Eq/L; nv =1.2-2.3 Eq/L), hypocalcemia (7.9 mg/dL; nv =8.5-10.5 mg/dL), anemia (hemoglobin =8.9 g/dL) and elevated levels of aspartate aminotransferase (peak 70 UI/L; nv =15-46 UI/L), creatine kinase isoenzyme MB mass (peak 5.2 ng/mL; nv =0.0- 4.0 ng/mL) and troponin I (peak 0.13 ng/mL; nv =0.00-0.06 ng/mL).

A 12-lead electrocardiogram (ECG) revealed sinus rhythm at 75 bpm, T wave inversion in V1 to V6 and in II, III, aVR; prolonged QT interval [QT =0.58”, corrected QT interval (QTc) =0.65”], increased QTc dispersion (Qtdc) =105 msec, low voltage of R-wave in V6 (Fig. 1). Echocardiography showed depressed global systolic function with ejection fraction (EF) =43%, dyskinesia involving apical segments, hyperkinesia in mid-basal segments and slight reduction of left
ventricular mass index (LVMI) (16 g/m²) (Fig. 2).

The patient was admitted to the coronary intensive care unit (CICU) and experienced several syncopes due to recurrent episodes of torsades de pointes (TdP) (Fig. 3), that were often self-limiting and in some instances, sustained and treated by electrical shock. Therefore, a temporary transvenous pacemaker, programmed in VVI mode at a rate of 90 bpm, was placed in the right ventricle; therapy with in-
travenous (i.v.) magnesium sulphate, potassium chloride, calcium gluconate, aspirine and heparin was started. We performed emergency coronary angiography that did not show significant stenosis (Fig. 4). Despite therapy, the patient continued to have recurrent TdP for seven days (Fig. 3). During the hospitalization the patient received psychological and nutrition therapy.

One month later, she was asymptomatic and was discharged with partial improvement in nutritional status (BMI =14.5 kg/m²). The ECG showed persistence of inversion of T waves, low R wave in V6, QT=0.57”, QTc=0.55”, QTcd=15 msec (Fig. 5). The laboratory tests returned to normal except for a mild anemia. The Holter ECG did not show sustained ventricular arrhythmias. Echocardiography demonstrated recovery of apical dyskinesia, and normal LV systolic function (EF 56%). The genetic analysis of KCNQ1, KCNH2, SCN5A, KCNE1, and KCNE2 failed to identify any known mutations associated with long QT syndrome. All her first-degree relatives showed an ECG with normal QT intervals. The ECG recorded 3 months after the onset of TTC had normal QT intervals (QT =0.37”, QTc =0.43”) (Fig. 6).

Discussion

The present case shows the clinical features of a TTC, a clinical entity disease usually triggered by intense emotional or physical stress, characterized by a transient left ventricular apical wall motion abnormality without epicardial coronary artery disease. In the TTC, also known as “left ventricular apical ballooning syndrome”, the electrocardiographic changes and the modest release of cardiac enzymes may mimick an acute myocardial infarction (1). Our patient was affected by AN, an eating disorder characterized by an abnormal loss of body weight, usually...
brought on by psychological disorders. The AN may be associated with cardiovascular complications (2) but, to our knowledge, an association between this disease and TTC has been reported in only one paper by Ohwada et al (3). However, in their case the precipitating event was hypoglycaemic coma while in our patient the blood glucose levels were normal.

The pathogenesis of the TTC is not clear but multivessel epicardial spasm, coronary microvascular dysfunction and a sudden catecholamine surge are the principal suggested mechanisms (4). In contrast, a vagal hyperactivity has been shown in AN (2). Instead, prolongation of QT interval and QT dispersion (QTd) have been reported both in the TTC (5) and in AN (6, 7). The QTd is the difference between the maximum and the minimum QT and QTc intervals in a 12-lead ECG and has been linked, such as QT prolongation, as a risk indicator for sudden cardiac death (8).

Various electrocardiographic abnormalities have been reported in AN, including bradycardia, reduction in QRS voltage, prolonged QT interval and QTd, alterations in ST segment (6). The cause of QT and QTcd prolongation in anorectic patients is still controversial and various mechanisms have been proposed (9). The lower left ventricular mass may lead to changes in homogeneity of cardiac ventricular repolarization. Cardiac hypotrophy may cause alterations of ion channels able to prolong the action potential. AN is often associated with hypokalemia, hypomagnesemia and hypocalemia and electrolyte abnormalities that may interfere with QT segment.

In the TTC the changes of QT are related to the transient myocardial damage. Moreover, according to some authors in certain circumstances, they may reflect an underlying genetic abnormality of repolarization (10, 11). In the present patient, the persistence of QT prolongation on 1-month ECG could suggest a primary repolarization abnormality. Abe et al (11) showed that the QT prolongation in 17 patients with a TTC returned to normal values between 97 and 191 days after the onset of symptoms. Nevertheless, we believe our patient was unlikely to have a genetic long QT syndrome. First, ECG recorded 3 months after TTC revealed normal QT intervals (Fig. 6). Furthermore, the genetic analysis failed to identify any known mutations. Eventually, the family history was negative for sudden death and the QT intervals of first-degree relatives were normal.

In our patient the QT prolongation and the increase of QTd caused by TTC was apparently amplified by AN and resulted in malignant and recurrent ventricular arrhythmias which persisted for a long time. Later, the slow reduction of QT interval and QTd was associated with a disappearance of arrhythmias.

Although the TTC generally has a good prognosis and ventricular arrhythmias are quite rare (5), the severe prolongation of the QT interval is to be considered as a predictor for the risk of sudden death (8). Sudden death is also a complication of AN, particularly if complicated by cardiac involvement (12). Thus, the combination of TTC with a condition associated with the prolongation of QT and/or with increase of QTd makes the acute and subacute prognosis of this disease much more severe than usually.
References


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