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IMAGES IN MEDICINE

Takotsubo Cardiomyopathy and Acquired Long-QT Syndrome

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KEY WORDS: *Takotsubo cardiomyoapthy; long QT syndrome; acute myocardial infarction*

ABSTRACT

A 55-year-old woman developed acute myocardial infarction while grieving for the death of her pet. The initial ECG showed T-wave inversion in leads I, aVL, V2, and V3, and a corrected QT (QTc) interval of 529 ms. Cardiac enzymes were mildly elevated. Cardiac catheterization revealed normal coronary arteries with akinesis of the anterior wall with ballooning during systole, findings compatible with an atypical form of Takotsubo cardiomyopathy. The patient responded to medical therapy. The second day, the QTc interval was reduced to 476 ms and then gradually normalized. Echocardiography at 7 days showed complete resolution of the anterior wall motion abnormalities.

Abbreviations
ECG = electrocardiogram
QTc = corrected QT interval

A 55-year-old woman presented to the emergency room complaining of chest pain. The patient reported that the chest pain developed after the death of her pet. Her past medical history was unremarkable. The initial 12-lead electrocardiogram (ECG) showed T-wave inversion in leads I, aVL, V2, and V3, and a corrected QT (QTc) interval of 529 ms (Fig. 1). Laboratory tests showed mild elevation of serum cardiac enzymes. The patient underwent cardiac catheterization which revealed normal coronary arteries free of any stenoses (Fig. 2). Left ventriculography demonstrated akinesis of the anterior myocardial wall with ballooning during systole (Fig. 3). The patient was treated as having non-ST-segment elevation myocardial infarction. The second day, the QTc interval was reduced to 476 ms (Fig. 1). Transthoracic echocardiography at 7 days later showed complete resolution of the anterior wall motion abnormalities. The QTc interval gradually normalized. These findings were considered as consistent with an atypical form of Takotsubo cardiomyopathy.

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Takotsubo cardiomyopathy is a clinical entity that mimics an acute coronary syndrome. It is characterized by chest pain, ECG changes, wall motion abnormalities and minimal myocardial enzymatic release in patients with normal or near-normal coronary arteries at coronary angiography.[1] A major stressful event (emotional or physical) usually precedes the onset of symptoms. Assessment of left ventricular function in patients with Takotsubo cardiomyopathy reveals apical ballooning and hypokinesia with preservation of basal contraction in the majority of cases.[1] In a minority of patients, like in our case, a different pattern with preserved apical contractile function

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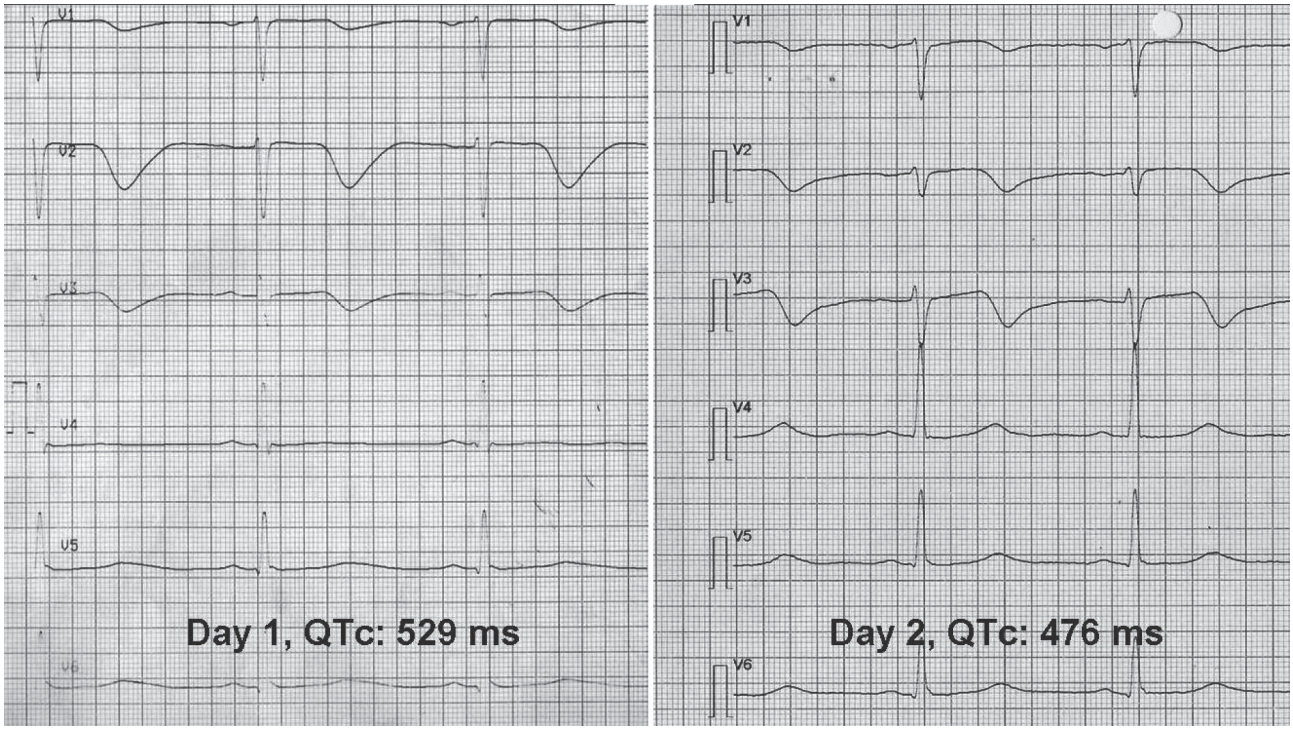

FIGURE 1. ECG on admission (day 1) demonstrating T-wave inversion in leads V2, and V3, and a corrected QT (QTc) interval of 529 ms (left panel). On the second day, the QTc interval was reduced to 476 ms (right panel). ECG = electrocardiogram.

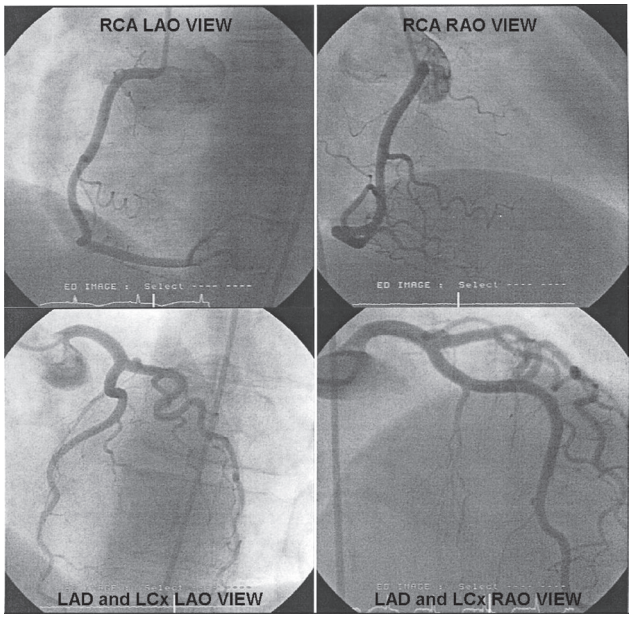

FIGURE 2. Coronary angiography showing normal coronary arteries. RCA: right coronary artery; LCx: left circumflex artery; LAD: left anterior descending artery; RAO: right anterior oblique; LAO: left anterior oblique.

and impaired midventricular contractility has been observed.[2,3]

The relationship between Takotsubo cardiomyopathy and QTc interval prolongation that predisposes to ventricular arrhythmias in about 1-1.5% of patients has been well documented.[4,5] Seth et al reported 12 cases of Takotsubo cardiomyopathy with an acquired long QT syndrome with a mean QTc interval of 478 ms.[6] Accumulating data have emerged providing evidence of an increased risk of polymorphic ventricular tachycardia in the form of torsade de pointes in patients with Takotsubo cardiomyopathy.[5,7,8] In a

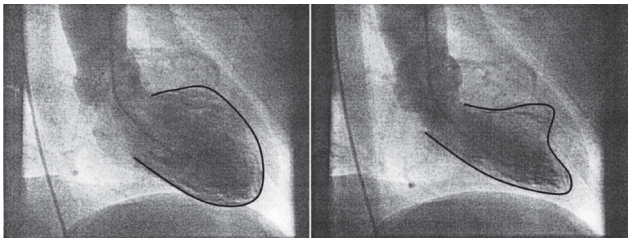

FIGURE 3. Left ventriculography with diastolic (left panel) and systolic (right panel) frames showing akinesia of the anterior wall with ballooning during systole (right panel).

systematic review, the mean QTc at presentation was 595 ms, the mean maximal QTc recorded was 706 ms, and the mean post-Takotsubo cardiomyopathy QTc was 481 ms in patients who developed torsade de pointes.[5] In another case series of Takotsubo cardiomyopathy, the presenting and maximal QTc intervals in the cases with torsades de pointes was significantly longer than those without torsades de pointes.[9] The presence of QTc interval prolongation at times other than during Takotsubo cardiomyopathy in a significant number of cases suggests that a reduced repolarization reserve may be the underlying mechanism in this acquired form of long QT syndrome.[10] Furthermore, a hyperadrenergic state (stimulation of the left stellate ganglion) has been shown to prolong the QT interval,[11] while catecholamines may induce early afterdepolarizations and torsade de pointes.[12]

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