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Takotsubo sendromu ile ilişkili uzun QT'nin tetiklediği ventrikül taşikardisi

Long QT-induced ventricular tachycardia associated with Takotsubo cardiomyopathy

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Özet– Takotsubo kardiyomiyopatisi, önemli koroner arter hastalığı olmayan hastalarda akut miyokart enfarktüsünü taklit eden elektrokardiyografik değişikliklerle beraber ventrikül apeksinin geçici balonlaşması ile karakterize bir sendromdur. Bu yazıda, uzun QT sendromunun tetiklediği ventrikül taşikardileri olan nadir bir Takotsubo sendromlu olgu sunuldu. Metoprolol tedavisinden sonra aynı gün içinde uzun QT aralığı normale döndü ve ventrikül taşikardileri sonlandı.

Summary– Takotsubo cardiomyopathy (TC) is a syndrome characterized by transient left ventricular apical ballooning associated with electrocardiographic changes and mimicking acute myocardial infarction in patients without significant coronary disease.. We report an unusual case of a patient who presented with TC associated with long-QT syndrome-induced ventricular tachycardia. QT interval prolongation was normalized and ventricular tachycardias were stopped within the same day with metoprolol treatment.

Abbreviations:

AV	Atrioventricular
EF	Ejection fraction
MI	Myocardial infarction
TS	Takotsubo syndrome
VT	Ventricular tachycardia

Takotsubo cardiomyopathy (TS) also known as broken heart syndrome is a syndrome characterized by transient left ventricular apical ballooning associated with changes on electrocardiograms in patients without significant coronary artery stenosis as detected on angiograms.[1] In 1 % of the cases suggestive of acute myocardial infarction (MI) TS has been detected.[2] Arrhytmic complications seen in MI are also observed in TS which creates difficulties in differential diagnosis.[3]

We have presented a patient with TS who consulted to us with long QT syndrome - induced episodes of ventricular tachycardia (VT).

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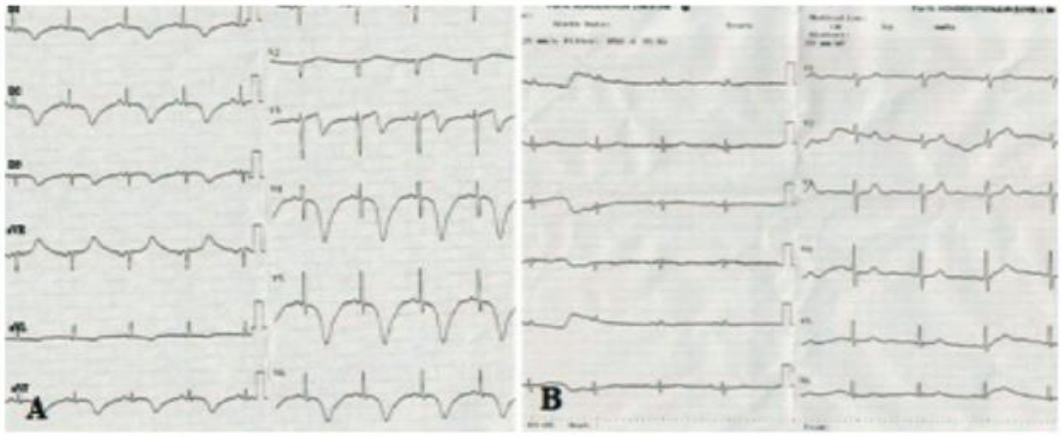

Figure 1. (A) Deep, and wide T wave inversion, QTc interval of 680 msecs on V3-6, D1-3, Avf leads of admission EKG (B) 24 hours after metoprolol treatment T waves normalized, and QTc interval dropped below 400 msecs

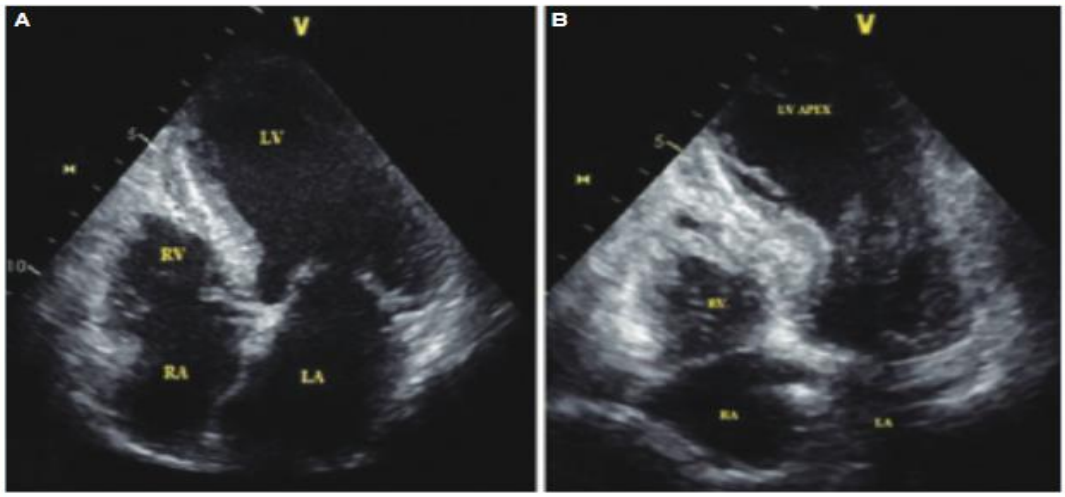

Figure 2. (A) Diastolic phase on electrocardiogram (B) In systolic phase thinning, aneurysmatic configuration of apical segment of the left ventricle is observed.

CASE PRESENTATION

A 67 –year-old female patient consulted to our emergency service with complaints of chest pain, dizziness, and episodes of palpitation starting nearly 12 hours previously. Her history revealed that she had lost her sister one month ago, and thereafter she was in severe mental depression, and complaining of sleep disorders. Besides, her eating habits were deranged, and she lost nearly 7-8 kg since then. She was also hypertensive. Her physical examination was unremarkable. On EKG, symmetrical, giant inverted T waves were detected on V3-6, D1, -3, and aVF derivations, and duration of QTc was 680 msec.(Figure 1a) During tracking of her EKG tracings on monitor screen, transient episodes of VT were observed. Her biochemical parameters were as follows: troponin I 3.8 ng/ml; NT-proBNP, 3200 pg/ml; calcium 8.9 mg/dl; magnesium 2.5 mg/dl; potassium 4.4 mg/dl; WBC, 8500/mm^3; sedimentation, 28 mm/h, and CRP, 12 mg/dl.. The patient was hospitalized in the intensive care unit During bedside echocardiographic examination, left ventricular ejection fraction (LVEF) was calculated as 40 % using modified

Simpson method. Apical segment was aneurysmatic, otherwise mobility of heart walls was within normal limits. (Figures 2a, and b). During coronary angiography performed to achieve early revascularization, a significant coronary artery stenosis was not detected (Figure 3a, and b), while apical aneurym was seen on ventriculograms (Figures 3c, and d). Emotional complaints of the patient, and typical echocardiographic, and ventriculographic findings suggested probable diagnosis of TS. Cardiac magnetic resonance (MR) imaging revealed apical wall thinning, and aneurysmatic formations (Figures 4a-d). During monitorization of the patient in the intensive care unit, frequently recurring transient episodes of VT were oıbserved. Upon occurrence of a single sustained episode of VT which impaired normal hemodynamic state of the patient, DC cardioversion was performed using a 100 Joules, and return to a sinus rhythm was achieved. As a maintenance therapy of the patient metoprolol (100 mg/q.i.d), acetylsalicylic acid (100 mg/ q.i.d), ramipril (10 mg/ q.i.d) were given. During psychiatric evaluation diagnosis of major depression was made, and sertraline (100 mg/ q.i.d), alprazolam (0.5 mg/ q.i.d) therapy was initiated. She hadn't suffered from any recurrent episodes of ventricular tachycardia during her follow-ups. Twenty-four hours after administration of metoprolol, despite a QTc interval of 390 msec, signs suggestive of apical aneurysma were observed on echograms (Figure 1b). Echocardiographic examination performed three weeks later demonstrated that apical segments were within normal limits, and LVEF was 60 percent (Video 1*) while on EKG, QTc interval was 400 msecs.

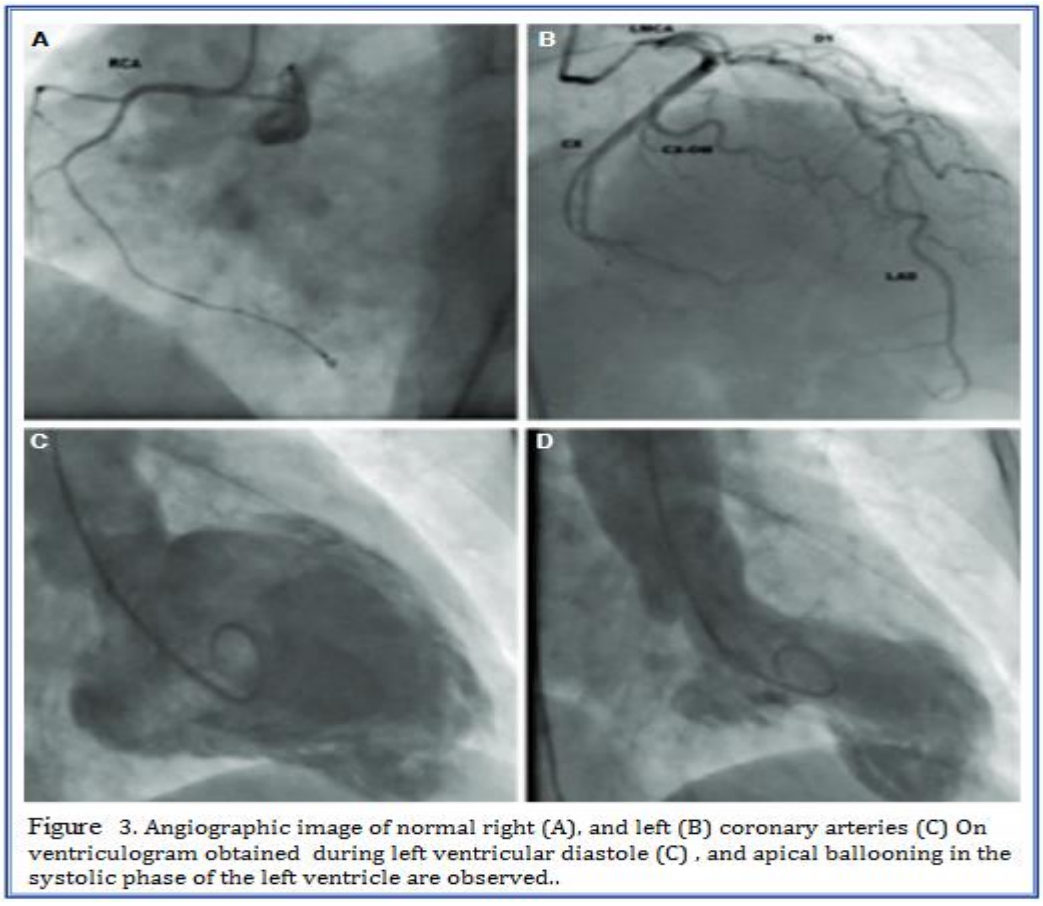

Figure 3. Angiographic image of normal right (A), and left (B) coronary arteries (C) On ventriculogram obtained during left ventricular diastole (C) , and apical ballooning in the systolic phase of the left ventricle are observed..

DISCUSSION

Takotsubo syndrome is usually seen in postmenauposal women. In its pathogenesis, stress factors, increased adrenergic activity, decreased estrogen levels, microvascular diseases, and insufficient fatty acid metabolism of the myocardium have been implicated. Besides higher serum cathecolamine levels were detected in these patients.[4] The patients usually mention of stressful physical or emotional events before the onset of symptoms. The disease rarely follow a clinically asymptomatic course. Our patient experienced an emotional depression after the death of her sister, and the patient was diagnosed as major depression at the time of her hospitalization. In patients with TS, symptoms as chest pain, dyspnea, and syncopes can be seen. Patients can present with symptoms of heart failure, supraventricular or ventricular arrhytmias. EKG findings may mimick MI. In anterior leads ST-segment elevation, T wave inversion, prolonged QT interval, and rarely abnormal Q waves can be seen.[4] Similarly, our patient was hospitalized with chest pain mimicking acute coronary syndrome. Besides, ischemic EKG signs, increased levels of cardiac enzymes, and prolonged QT interval on EKG. QTc was 680 msecs, and sharply steep symmetrical T wave inversion were found. During follow-ups, episodes of VT which we thought to be related to long QT were observed.

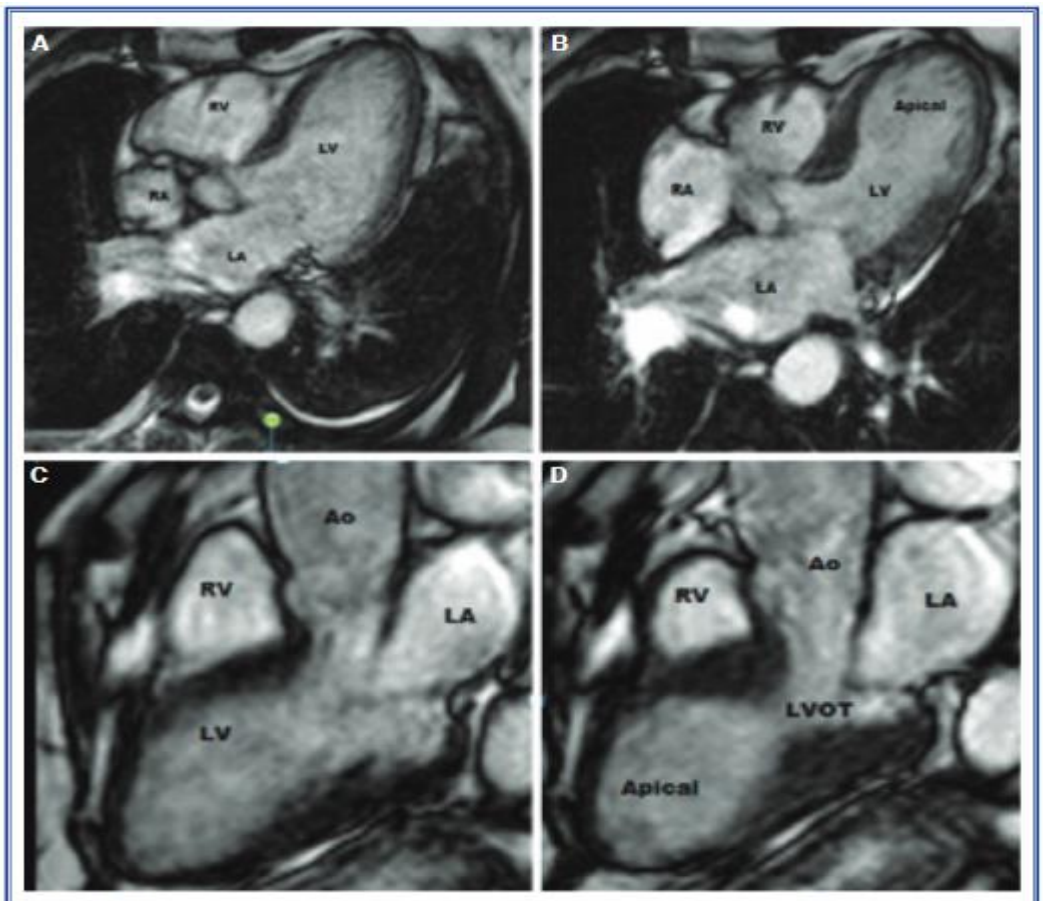

Figure 4. (A) On cardiac magnetic resonance images, left ventricular diastolic phase on horizontal axis, (B) left ventricular systolic phase, (C) left ventricular diastolic phase on sagittal axis and, (D) left ventricular systolic phase

In a patient diagnosed as takatsubo syndrome by Furushima et al. [5] duration of QTc was measured as 740 msecs. In their electrophysiological studies, prolonged action potential extending from basal part of the ventricle towards the aneurysmatic apical wall was detected, and the authors asserted that pathophysiology of deeper, and wider giant T wave inversion had stemmed from this prolonged action potential. Besides, long-QT syndrome has been reported to originate from wide T waves. Behr et al.[6] reported that prolonged QT interval, and apical aneurysm are generally transient in nature, and apical configuration returns to normal dimensions within nearly 3-6 months. The authors also warned that TS patients with a QTc interval longer than 500 msecs have a serious risk of developing torsades de pointes. In our patient QTc interval was 680 msecs, and transient VTs were found. However during follow-ups torsades de pointes did not develop. Syed et al. [3] from Mayo clinic performed a meta-analysis in 816 patients with diagnosis of TS, and detected development of ventricular fibrillation and/or VT (1-1.5 %), atrial fibrillation (4.7 %), sinus node dysfunction (1.3 %), atrioventricular (AV) node dysfunction (Mobitz type 1.,2., and 3. degree AV block) . Still in this meta-analysis, the authors asserted supportive data about beneficial effects of beta-blocker and/or mexiletine in the control of tachyarrhytmias. In our case, within a short time as 24 hours after initiation of metoprolol therapy, apical aneurysms persisted as detected on echocardiograms while duration of QTc decreased below 400 msecs, and episodes of VT terminated which supports the suggestions of Syed et al. However it is conceivable that normalization of QTc interval might stem from reversible characteristics of the disease. Further prospective studies are needed on this subject.

As an outcome of our study, we think that TC might have an arrhytmogenic potential, and in the etiology of long QT-induced VT, TC should be kept in mind. Though a clear-cut consensus has not been reached on the treatment of these patients, beta-blocker treatment can be beneficial in the management of arrhytmias. We have arrived at a conclusion in that further studies should be performed on this subject.

* Video file of the article is available online web address of the journal

Conflict of interest: None declared

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