

Sinus surgery complicated by ventricular fibrillation in a young patient: Inverted (reverse) Takotsubo cardiomyopathy

Genç bir hastada sinüs cerrahisi sonrası gelişen ventrikül fibrilasyonu: Ters Takotsubo kardiyomyopatisi

Gültekin Günhan Demir, M.D., Gamze Babur Güler, M.D., Ekrem Güler, M.D.,
Hacı Murat Güneş, M.D., Filiz Kızılırmak, M.D.

Department of Cardiology, Medipol University Faculty of Medicine, İstanbul, Turkey

Summary– Takotsubo cardiomyopathy (TTC), also known as left ventricular apical ballooning syndrome or stress cardiomyopathy, is characterized by transient left ventricular systolic dysfunction and the absence of obstructive lesion in the epicardial coronary arteries. The most common presentation is acute substernal chest pain, although occasionally dyspnea and syncope, and rarely shock with ST-segment elevation and elevated cardiac biomarkers have been observed. Inverted (reverse) TTC is a rare pattern characterized hypokinesia of the basal and midventricular segments. Presently described was case of a 27-year-old woman with ventricular fibrillation following endoscopic nasal sinus surgery.

Takotsubo cardiomyopathy (TTC) is characterized by transient left ventricular systolic dysfunction in the apical and mid segments, with the absence of obstructive lesion in the epicardial coronary arteries.^[1] TTC predominantly occurs in post-menopausal women (90%) and is most frequently triggered by emotional or physical stress.^[2] Also known as left ventricular apical ballooning syndrome, inverted (or reverse) TTC is a less common pattern of stress cardiomyopathy, involving the basal segments of the left ventricle, and typically occurring in younger patients.^[3] Described in the present report is the case of a young female patient who presented with reverse TTC after undergoing endoscopic sinus surgery.

Abbreviations:

ECG Electrocardiogram
TTC Takotsubo cardiomyopathy

Özet– Takotsubo kardiyomyopatisi (TTK), apikal balonlaşma veya stres kardiyomyopatisi olarak da bilinmektedir ve epikardiyal koroner arterlerde kritik darlığın eşlik etmediği geçici sol ventrikül fonksiyonu bozukluğu ile kendini gösterir. En sık başvuru şekli akut substernal göğüs ağrısı, bazen ani nefes darlığı ve senkop ve nadiren de şok şeklinde olup tabloya ST-segment yükselmesi ve kardiyak biyobelirteçlerin yükselmesi eşlik eder. Ters TTK ise çok daha nadir görülür ve bazal ve/veya midventriküler segmentlerin hipokinezi ile karakterizedir. Bu yazıda, kronik sinüzit ve nazal polip nedeniyle nazal sinüs cerrahisi geçiren ve cerrahi sonrası ventrikül fibrilasyonu gelişen 27 yaşındaki bir kadın hasta sunuldu.

CASE REPORT

A 27-year-old woman with chronic sinusitis and nasal polyposis was admitted for endoscopic sinus surgery. She was otherwise healthy and had no risk factors for cardiovascular disease. Preoperative electrocardiogram (ECG) and blood tests were unremarkable. Endoscopic sinus surgery was performed under general anesthesia induced with propofol. During the emergence period (recovery from general anesthesia), the patient developed ventricular fibrillation, and was successfully resuscitated. ECG obtained after defibrillation revealed ST-segment elevation in DI and aVL leads, and ST depression in inferior and V3–6 leads (Figure 1). Echocardiography demonstrated hypokinesia in the basal inferior and posterior segments of the left ventricle, and estimated ejection fraction was

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Correspondence: Dr. Gültekin Günhan Demir. Birlik Mahallesi, Bahçeler Caddesi,
No: 5, 34220 Esenler, İstanbul, Turkey.

Tel: +90 212 - 440 10 00 e-mail: ggdemir@gmail.com

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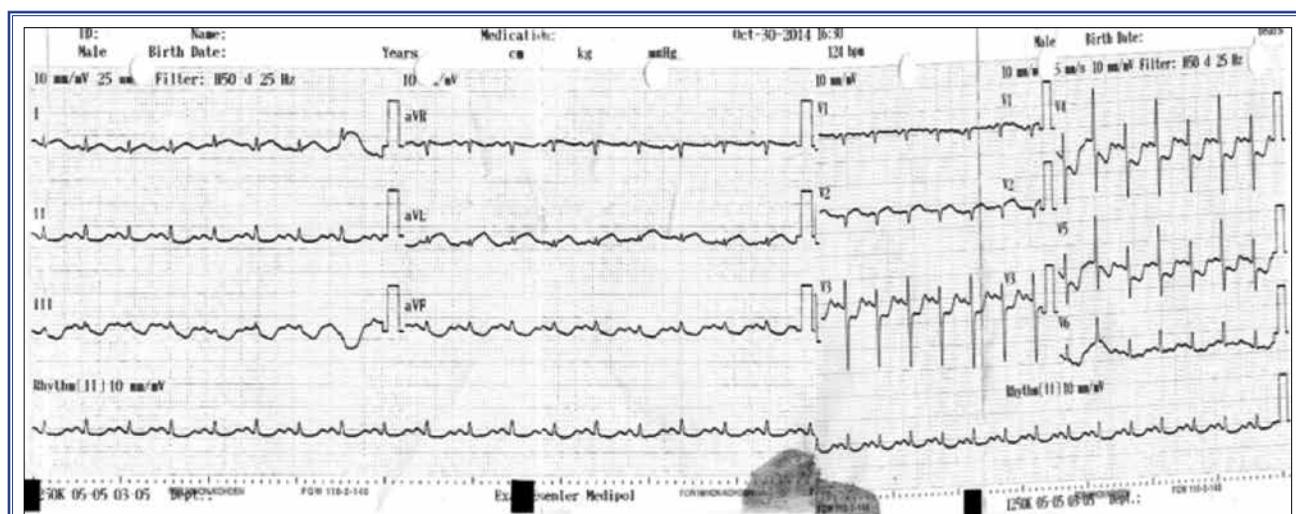


Figure 1. Electrocardiogram obtained immediately after defibrillation showing ST elevation in DI and aVL leads, and ST depression in inferior and V3–6 leads.



Figure 2. Transthoracic echocardiographic parasternal long-axis image showing hypokinesia in the basal septum and posterior segments (blue arrows). Hyperkinetic image in the remaining segments of the left ventricle.

40 percent (Figure 2). The patient was immediately transferred to the catheterization laboratory for coronary angiography, which revealed normal left coronary system and osteal plaque formation in the right coronary artery.

The patient received appropriate treatment in the coronary care unit and was extubated the following day. Biochemical blood tests, including that for magnesium, were normal. Troponin I levels were mildly elevated (peak level: 0.058 ug/L; upper limit: 0.023 ug/L). Serial ECG follow-up showed complete resolution of ST-segment elevation. Strain and strain rate imaging analysis obtained prior to discharge revealed ongoing recovery, with the exception of posterobasal

hypokinesia (Figure 3). Control echocardiographic examination showed improved left ventricular ejection fraction, confirmed with cardiac magnetic resonance imaging. No cardiac symptoms were observed during postoperative course, and control ECG was completely normal (Figure 4).

DISCUSSION

Sato et. al described the first case of TTC in 1990,^[4] and though it was initially considered to be a rare condition, recent retrospective data has demonstrated that cases of TTC represented approximately 2% of all suspected cases of acute coronary syndrome, owing to increased awareness among cardiologists. [4] Though over a quarter-century has passed since its first description, pathophysiological mechanisms of TTC have yet to be clarified. Several mechanisms have been proposed, including coronary microvascular dysfunction, coronary vasospasm, induction of myocardial stunning by catecholamine excess and enhanced sympathomimetic activity, myocardial microinfarction, and impaired cardiac fatty acid metabolism. Cardiotoxicity induced by catecholamine excess and coronary microvascular dysfunction are the most popular hypotheses thus far.^[5]

Though no universally accepted definition exists, that most commonly used in daily practice and research incorporates Mayo Clinic diagnostic criteria, which were modified in 2008, and are as follows:

- 1) Transient hypokinesia, akinesia, or dyskinesia

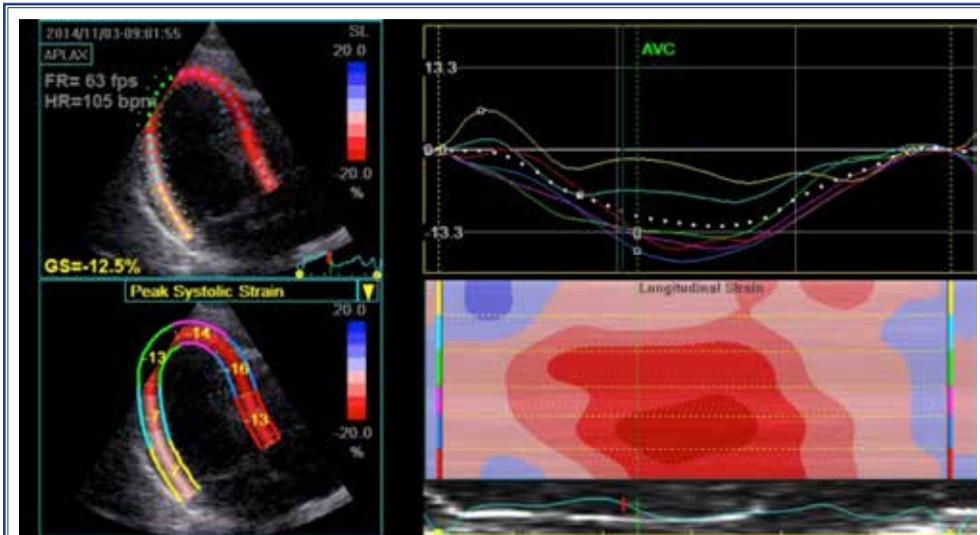


Figure 3. Measurement of left ventricle strain and strain rate, demonstrating ongoing left ventricular recovery, with the exception of posterobasal hypokinesia.

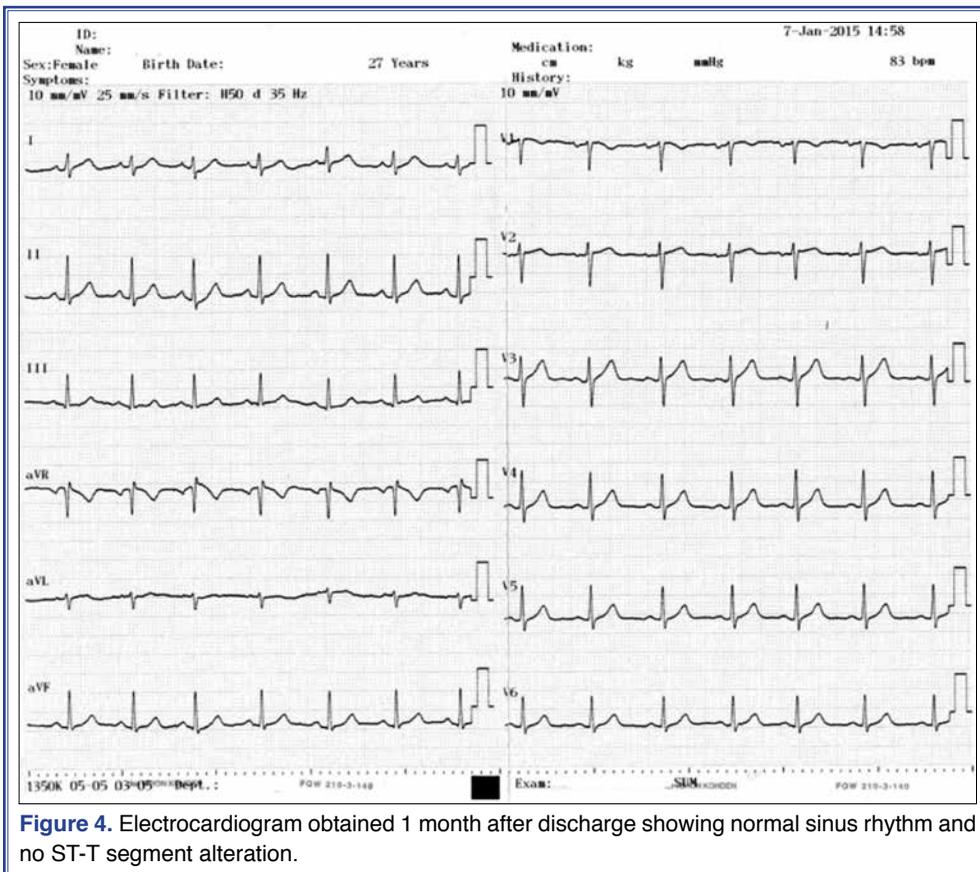


Figure 4. Electrocardiogram obtained 1 month after discharge showing normal sinus rhythm and no ST-T segment alteration.

of the left ventricular mid-segments with or without apical involvement. Regional wall motion abnormalities that extend beyond a single epicardial vascular distribution. A stress trigger is often, but not always present.

2) Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.

3) Newly occurring electrocardiographic abnormalities (ST-segment elevation and/or T-wave inver-

sion) or modest elevation in cardiac troponins. Absence of pheochromocytoma or myocarditis.^[6]

Regional wall motion abnormalities and electrocardiographic alterations (usually ST-segment elevation) that extend beyond a single coronary artery distribution, and documentation of normal or near-normal coronary arteries should be the key features in TTC recognition, as in the present case. Most cases of TTC described include ECG changes (usually ST-segment elevation), apical ballooning, and documentation of normal coronary arterial tree. Although a typically benign course has been recognized, TTC is also known to rarely cause life-threatening ventricular arrhythmias and sudden cardiac death. Syed et al. reported ventricular fibrillation prevalence of 1.8% (15 of 816 cases) in a review in which arrhythmia occurrence with TTC was investigated.^[7] This rate was reported as 4.2% (4 of 105 cases) elsewhere.^[8] Presentation of TTC with arrhythmia may challenge the clinician during differential diagnosis, as in the present case. Requisition of defibrillation may stun left ventricle functions and motions. However, echocardiographic images of the present patient were obtained after exposure to defibrillation, and it would have been impossible to discriminate the stunning of the left ventricle from the pathophysiologic process of TTC leading wall motion abnormality and ventricular fibrillation.

TTC has 4 subtypes: classical, reverse (or inverted), midventricular, and localized. Reverse TTC is characterized by transient systolic dysfunction of the basal segments of the left ventricle. Unlike the profile of classical TTC, which includes post-menopausal women, reverse TTC has been significantly associated with younger age and mental or physical stress, as opposed to catecholaminergic states.^[9] Reverse TTC has also been associated with diverse conditions and diseases, general anesthesia, and surgery.^[10] Reverse TTC can present with hemodynamic deterioration during any phase of anesthesia or surgery. Insufficient anesthesia and severe abrupt surgical pain stimulus are considered responsible for the catecholamine surge.^[11] In addition, reverse TTC can be triggered during recovery from general anesthesia, as in the present case.^[12,13]

TTC presents with chest pain, electrocardiographic and echocardiographic abnormalities, and increased cardiac biomarkers, mimicking acute

coronary syndrome and mandating prompt differentiation. Coronary angiography is usually essential for diagnostic confirmation, and most patients have angiographically normal epicardial coronary arteries or mild atherosclerosis, as the typical patient profile is a post-menopausal woman. Furthermore, it was recently suggested in a report by Napp et al. that TTC and coronary artery disease can coexist, and that detection of obstructive coronary artery disease should not exclude diagnosis of TTC in all cases.^[14] Similarly, regional wall motion abnormality beyond a single epicardial artery territory is a typical echocardiographic finding in patients with TTC. A similar debate regarding association of myocarditis and TTC is ongoing.^[15] Myocardial injury biomarkers may be normal or mildly elevated, different from the pattern observed in acute coronary syndromes.

Presently described was a case of reverse TTC following endoscopic sinus surgery in a young female patient. In spite of good prognosis and outcome, prompt recognition and treatment is of primary importance in this uncommon pattern of an uncommon disease.

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- Anahtar sözcükler:** Ters Takotsubo kardiyomiyopatisi; sinüs cerrahisi; ventriküler fibrilasyon.