Exercise-induced ventricular tachycardia associated with asymptomatic Brugada syndrome in a patient with urinary bladder stone

Mesane taşı bulunan, asemptomatik Brugada sendromlu bir hastada egzersizle ortaya çıkan ventrikül taşikardisi

Özcan Özeke, M.D., Kumral Ergün Çağlı, M.D.,[1] Dursun Aras, M.D.,[1] Erdoğan İlkay, M.D.

Department of Cardiology, MESA Hospital, Ankara;
[1]Department of Cardiology, Türkiye Yüksek İhtisas Heart-Education and Research Hospital, Ankara

It is well-known that autonomic nerve modulation has an important role in the occurrence of ventricular tachyarrhythmias in Brugada syndrome. A 59-year-old man underwent cardiac evaluation before surgery for urinary bladder stone. He had no cardiac complaints and the only coronary risk factor was heavy smoking. The electrocardiogram showed a saddleback type ST-segment elevation in leads V1-V2, and left axis deviation. During exercise stress test, ventricular tachycardia with a left bundle branch block pattern appeared, and the saddleback type ST-segment elevation in V2 changed into a coved-type ECG at the recovery phase. The ventricular tachycardia was hemodynamically stable and normalized without medication. An echocardiogram showed normal left and right ventricular functions, and subsequent coronary angiography revealed normal coronary arteries. Based on these findings, a diagnosis of asymptomatic Brugada syndrome was made. Considering this particular case, it can be speculated that bladder-cardiac reflex may stimulate the autonomic nervous system via the vagus nerve and unmask Brugada syndrome.

Key words: Autonomic nervous system; bundle-branch block/etiology; electrocardiography; heart conduction system; syndrome; tachycardia, ventricular/etiology; urinary bladder diseases.

Brugada sendromlu hastalarda ventrikül taşikardisinin oluşumunda otonom sinir sisteminin önemli rol oynadığı iyi bilinmektedir. Elli dokuz yaşında bir erkek hasta, mesane taşı nedeniyle yapılacak ameliyat öncesinde kardiyolojik açıdan değerlendirildi. Daha önce kardiyak sorunları olmayan ve koroner risk faktörü olarak sadece sigara içme öyküsü olan hastanın elektrokardiyogramında V1-V2 derivasyonlarında semer sırtı şeklinde ST-segment yükselmesi ve sol eksen deviyasyonu izlendi. Egzersiz stres testinde, sol dal bloku ile birlikte ventrikül taşikardisi ortaya çıktı ve dinlenme fazında V2'deki semer sırtı şeklindeki ST-segment yükselmesi çukur (coved) tipe dönüştü. Ventrikül taşikardisi hemodinamik olarak stabildi ve ilaç tedavisi olmaksızın normale döndü. Ekokardiyografide sağ ve sol ventrikül fonksiyonları, anjiyografide koroner arterler normal bulundu. Bu bulgular ışığında, tanı asemptomatik Brugada sendromu şeklinde kondu. Sunulan olgu göz önüne alındığında, mesane-kardiyak refleksin vagus siniri aracılığıyla otonom sinir sistemini uyardığı ve Brugada sendromunu açığa çıkardığı ileri sürülebilir.

Anahtar sözcükler: Otonomik sinir sistemi; dal bloku/etyoloji; elektrokardiyografi; kalp iletim sistemi; sendrom; taşikardi, ventriküler/etyoloji; mesane hastalığı.

Brugada syndrome is an inherited cardiac disease causing life-threatening ventricular tachyarrhythmias in patients with a structurally normal heart and a characteristic electrocardiographic (ECG) pattern of right bundle branch block and ST-segment elevation in the right precordial leads.[1] In recent years, an increasing number of reported cases have appeared in the literature with clinical, genetic, cellular, ionic, and molecular aspects of the syndrome. However, the mechanism responsible for the characteristic ECG changes and occurrence of ventricular arrhythmias is still unclear.[2,3]

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Correspondence: Dr. Özcan Özeke. Mesa Hastanesi, Kardiyoloji Kliniği, 06510 Ankara, Turkey.
Tel: +90 312 - 292 99 86 e-mail: ozcanozeke@gmail.com

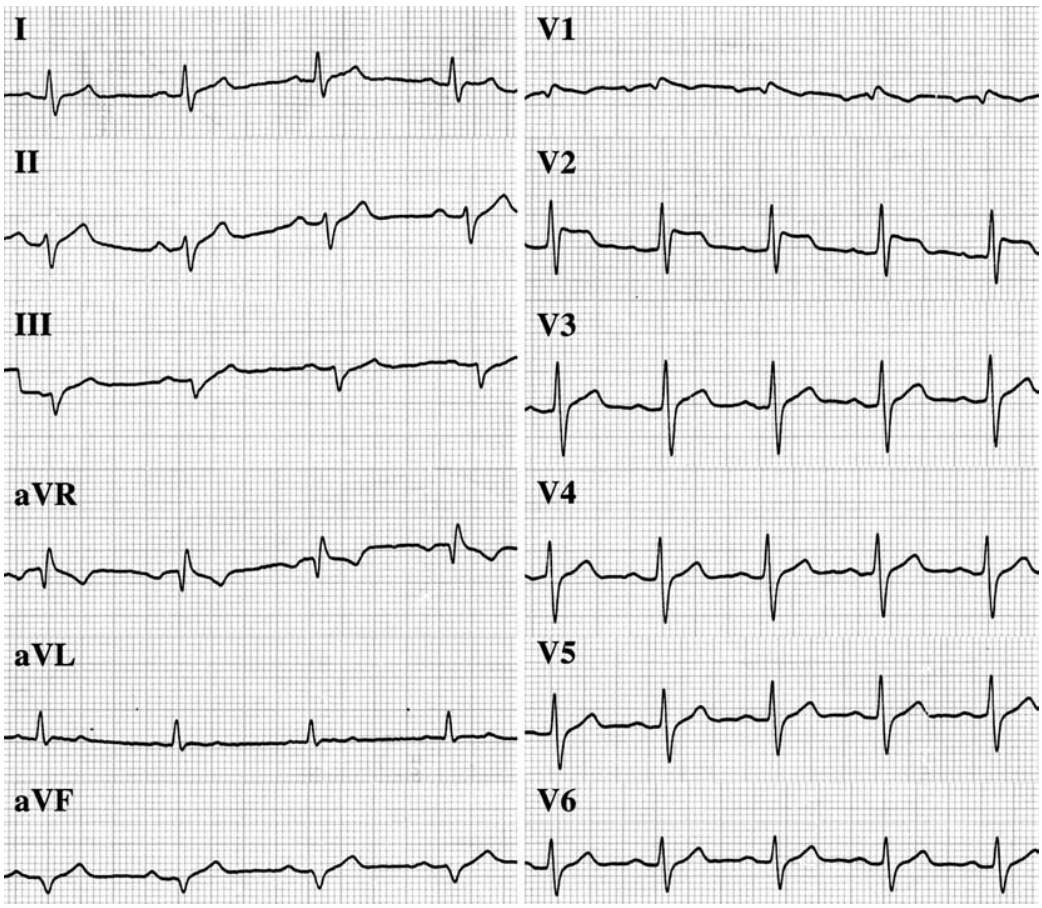

Figure 1. The electrocardiogram shows incomplete right bundle branch block with saddleback type ST-segment (J-point) elevation in leads V1-V2 and left axis deviation.

CASE REPORT

A 59-year-old man was referred to our clinic for a cardiac evaluation before surgery for urinary bladder stone by the urology department. He had virtually no cardiac complaint or any medication before. His family history was unremarkable for any cardiovascular pathology, and the only coronary risk factor was heavy smoking (30 packs/year). On physical examination, his heart rate was 85 beats/min, blood pressure was 130/80 mmHg, and body temperature was 36.1 °C. Cardiopulmonary findings were normal. Hematological and biochemical tests were within normal limits. The electrocardiogram showed a saddleback type ST-segment elevation in leads V1-V2, and left axis deviation (Fig. 1). During exercise stress test (EST) with the Bruce protocol, ventricular tachycardia with a left bundle branch block pattern appeared, suggesting a limited origin in the right ventricle, and the saddleback type ST-segment elevation in V2 changed into a coved-type ECG at the recovery phase (Fig. 2). The ventricular tachycardia was hemodynamically stable and normalized without medication. An echocardiogram showed normal left and right ventricular functions, and subsequent coronary angiography revealed normal coronary arteries. Based on the dynamic ECG changes (conversion from saddleback to coved-type ECG) and exercise-induced ventricular tachycardia showing a left bundle branch block pattern in a structurally normal heart, a diagnosis of asymptomatic Brugada syndrome was made. Since neither the patient nor any of his family members had experienced any arrhythmic symptom or sudden cardiac death, we did not perform an electrophysiologic study and medical follow-up was decided. After the discovery of asymptomatic Brugada syndrome, the patient refused the urologic operation and remained asymptomatic during follow-up without any medication.

DISCUSSION

Exercise-induced monomorphic ventricular tachycardia originating from the right ventricular outflow tract without evidence for structural heart disease can be

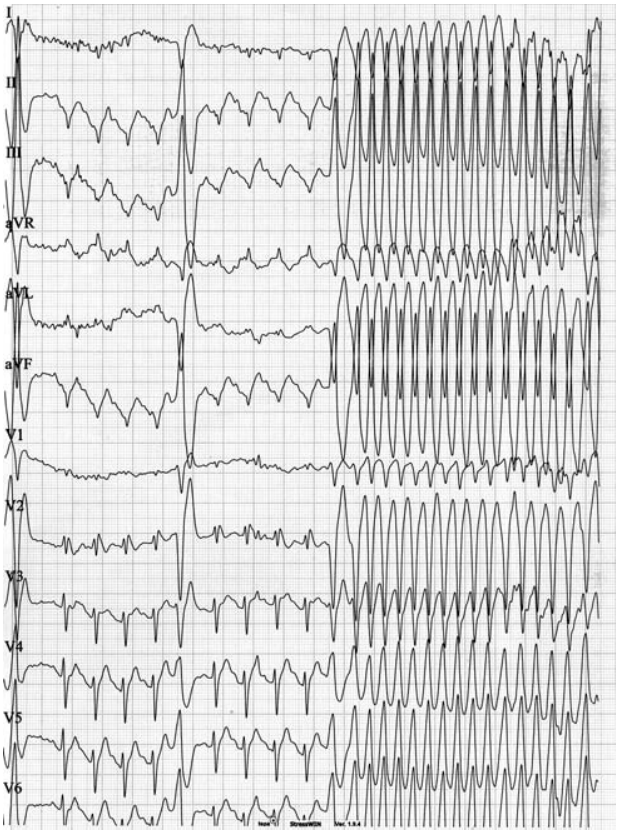

Figure 2. The electrocardiogram of exercise-induced ventricular tachycardia. The QRS morphology of ventricular tachycardia shows left bundle branch block pattern and coved-type ST-segment elevation in V2.

idiopathic or can be due to structural abnormalities such as arrhythmogenic right ventricular dysplasia; however, in the presented case, there was no electrocardiographic and echocardiographic evidence for arrhythmogenic right ventricular dysplasia.

The genesis of ventricular arrhythmias in Brugada syndrome is still under investigation. Unbalanced autonomic nervous activity appears to have an important role in inducing Brugada-type ECG signs, as suggested by the nocturnal occurrence of associated tachyarrhythmias, suggesting parasympathetic dominance to be a triggering factor.[4] Reduced adrenergic activity with subsequent dominance of the parasympathetic tone has been proposed as a mechanism of autonomic imbalance.[5-8] It is well-known that cardiovascular and urinary systems are under control of autonomic nervous system. There are indications that inputs from the arterial baroreceptors and urinary bladder converge on the same autonomic efferent pathways.[9] Distension of the urinary bladder has been shown to cause a reflex response for an increase in sympathetic activities, resulting in reflex increases in heart rate and arterial blood pressure, which in turn lead to increases in the maximal rate of rise of left ventricular pressure (dP/dt_{max}) and coronary blood flow.[10-12] It has been reported that the Brugada-type ST shift is induced by acute cholecystitis or augmented by gastric distension after a large meal, and it has been postulated that the existence of a biliary/gastric-cardiac reflex and gastric or gallbladder traction may stimulate the vagus nerve and induce electrocardiographic changes.[13,14] In addition, distension of other viscera such as the urinary bladder, stomach, or descending colon has been shown to primarily cause reflex coronary vasoconstriction, which can precipitate myocardial ischemia, similar to that occurring with distension of the gallbladder.[15] Ventricular tachycardia due to distention of the urinary bladder has also been reported.[16] Clinically, an interaction between the heart and urinary bladder is well-known in micturition syncope. The functional relation between bladder distension and sympathetic vasoconstrictor activity probably plays a role in clinical conditions such as autonomic dysreflexia and micturition syncope.[17-19]

In the presented case, there was no conclusive proof of the etiology and the coexistence of the Brugada-type electrocardiographic pattern and urolithiasis might be coincidental; however, a causal association could not be ruled out on the basis of generalized autonomic dysfunction (possibly an abnormal dominant parasympathetic state). Clinically, autonomic system may be affected by excessive distension of the bladder by bladder stone causing complete obstruction of the flow. The mechanism of this occurrence could also be related to the existence of a bladder-cardiac reflex whereby traction on the urinary bladder may stimulate the autonomic nervous system via the vagus nerve and induce electrocardiographic changes.

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