

Ajmalin testi sırasında gelişen monomorfik ventrikül taşikardisi

Monomorphic ventricular tachycardia developed during the ajmaline challenge test

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Özet- Çarpıntı şikâyeti ile gittiği bir klinikte geniş QRS'li taşikardi saptanan 44 yaşında erkek hastanın amiodaron ile düzeldiği, daha sonra yapılan koroner anjiyografinin normal olduğu öğrenildi. Hastanın kliniğimize başvurduğu istirahat EKG'si Brugada tip 2 ile uyumlu olması nedeniyle ajmalin testi yapılması planlandı. İnfüzyonun dördüncü dakikasında tip 1 EKG bulgularının ortaya çıktığının görülmesi üzerine infüzyon sonlandırıldı. Yaklaşık 10-15 sn sonra hastada aniden 150 atım/dakika hızında monomorfik ventrikül taşikardisi gelişti. İzleme süresinde hastanın ritmi 3-4 dk içerisinde kendiliğinden sinüs ritmine döndü. Brugada sendromunda senkop veya ani kardiyak ölüme neden olan taşiaritmi sıklıkla polimorfik ventrikül taşikardisi veya ventrikül fibrilasyonudur. Bununla beraber olgumuzda olduğu gibi nadiren monomorfik taşikardi görülebilir. Bu yazıda, ajmalin testi sırasında çok nadir görülen monomorfik ventrikül taşikardili olgu sunuldu.

Summary- A 44-year-old male patient admitted with palpitations was diagnosed as tachycardia with wide QRS, but recovered after being treated with amiodarone. The patient's coronary angiography was normal. As the patient's resting ECG was compatible with Brugada type 2, an ajmaline challenge test was scheduled. The infusion procedure was stopped following an observation of type 1 ECG findings at the 4th minute of infusion. Approximately 10-15 seconds later, a monomorphic ventricular tachycardia with a rate of 150 bpm developed. During the follow-up period, the patient's heartbeat returned spontaneously to the sinus rhythm within 3-4 minutes. Polymorphic ventricular tachycardia or ventricular fibrillation tachyarrhythmias usually result in syncope or sudden cardiac death in cases with Brugada syndrome, while monomorphic tachycardia, as in our case, is rare. Here, we present a rare case of monomorphic ventricular tachycardia, which was observed during the ajmaline challenge test.

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Abbreviations:

| | |
|----|--------------------------|
| AV | Atrioventricular |
| VF | Ventricular fibrillation |
| VT | Ventricular tachycardia |

Brugada syndrome is an inherited heart disease progressing with arrhythmias characterized by a higher risk of sudden death. Brugada syndrome is responsible for 4 % of all-cause sudden deaths, and 20 % of sudden deaths in patients with normal cardiac functions.^[1,2] It occurs as a result of a mutation in sodium channel genes (SCN5A), and sudden death happens usually in association with polymorphic ventricular tachycardia or ventricular fibrillation.^[2,3]

Classical ECG finding (type 1) is ST-segment elevation in the right precordial derivations (V1-V3).^[2,3] However typical ECG findings can not be found always or they can be variable. Therefore ECG recordings should be obtained at different time points. Besides, sodium channel blockers used for the patients without typical ECG findings (concealed type, other than type 1; ie. type 2 or 3) can restore typical ECG tracings.

Ajmaline is a calcium channel blocker used to support the diagnosis in suspect Brugada syndrome patients without typical EKG findings. It is known that during application of ajmaline challenge test, though rarely, widening of QRS complex, atrioventricular (AV) block, polymorphic ventricular tachycardia (VT), ventricular fibrillation (VF) can occur. In this case report, an episode of monomorphic VT developed in a patient with the initial diagnosis of Brugada syndrome who had undergone ajmaline challenge test was presented.

CASE PRESENTATION

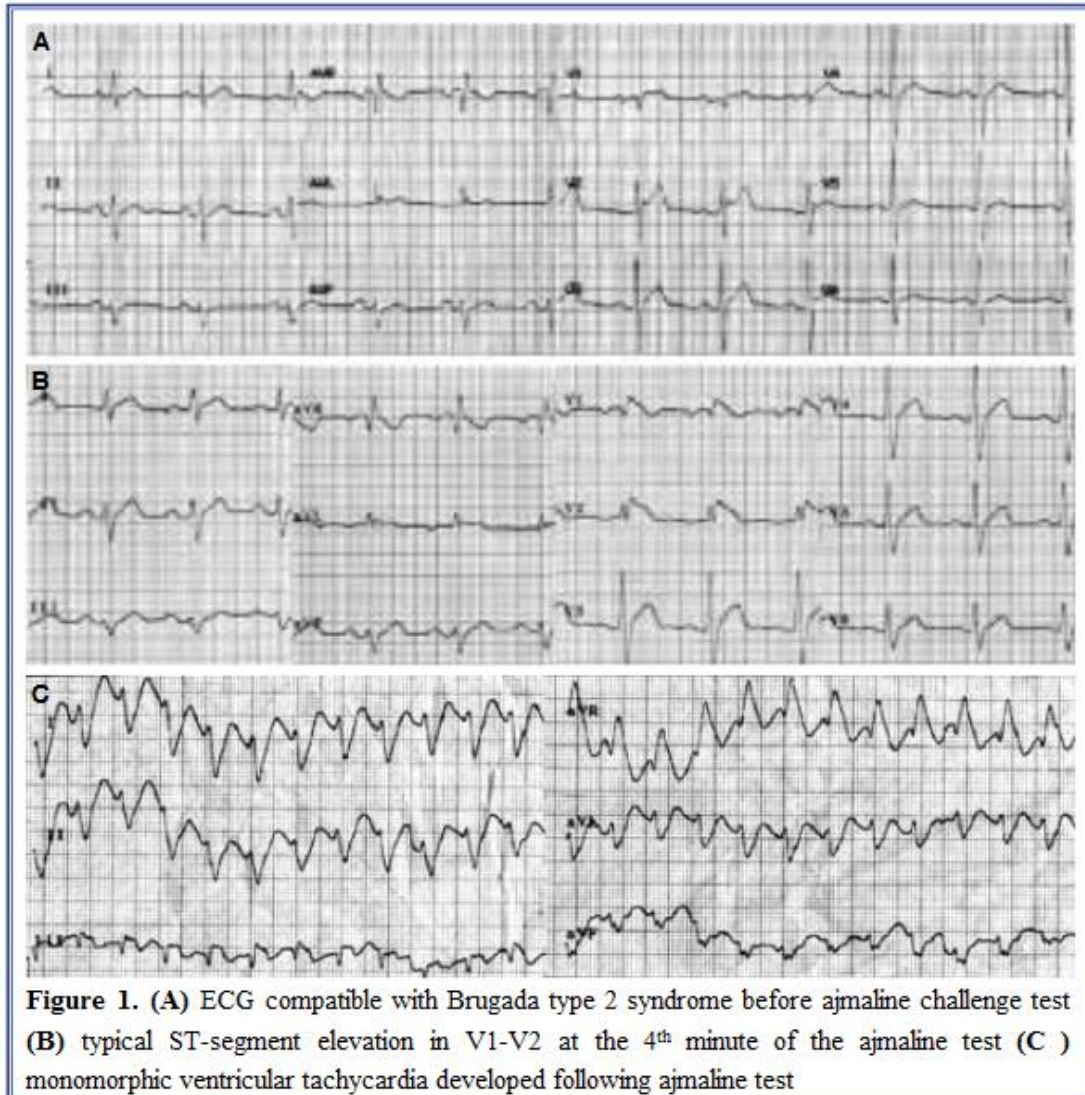
History of a 44-year-old male patient consulted to our clinic revealed

that he presented to another clinic one month ago with complaints of palpitation. His ECG demonstrated tachycardia with a large QRS complex which returned to sinus rhythm with amiodarone therapy, while his coronary arteries were found to be normal on angiograms obtained. His treatment was then initiated with daily oral doses of 400 mg amiodarone, then he was discharged from the hospital. It was learnt that nearly 10 years ago, he had been hospitalized for fainting episodes, and coronary artery angiograms obtain at that time was also unremarkable.

When the patient came to our clinic, his physical examination findings were normal, and biochemical test results were within physiological limits. On his echocardiogram, any pathological finding was not encountered. His resting ECG findings were compatible with Brugada type 2 syndrome (Figure 1a). His amiodarone therapy was discontinued, and electrophysiological study, and ajmaline challenge test were recommended. However, the patient did not consent to discontinue his amiodarone therapy. Therefore these procedures were performed without stopping amiodarone therapy. During electrophysiological study, despite extra triple stimuli delivered from both atrium, and ventricle, tachycardic heart beats could not be triggered. Subsequently, the patient was brought into coronary intensive care unit for ajmaline challenge test. He was connected to 12-lead ECG, and defibrillator for monitorization.. An intravenous route was opened, and IV ajmaline infusion at a dose of 1 mg/kg-bwt/5 min was started. Infusion was stopped upon detection of type 1 ECG findings at the 4th minute of the infusion (Figure 1b). Nearly 10-15 sec later suddenly, a monomorphic ventricular tachycardia at a rate of 150 bpm developed

(Şekil 1c). For the return to the sinus rhythm, induction of the ventricle of the patient whose blood pressure was 90/60 mm Hg, with stimuli delivered at a rate faster than the ventricular beat was

planned, and to that end he was transferred into the coronary angiography unit. However his heartbeat spontaneously returned to the sinus rhythm within 3-4 minutes.



However ECG tracings at sinus rhythm demonstrated persistence of ST-segment elevation in V1-V3. Close monitorization of the patient revealed after a while, decrease in ST-segment elevation down to baseline values. The patient, and his intimate were informed about his disease in detail, and implantation of ICD was recommended. The patient declined any intervention to be performed, and signed

treatment refusal form before his discharge from the hospital.

DISCUSSION

Diagnosis of Brugada syndrome requires the presence of type 1 ECG findings. Typical ECG findings are right bundle branch block in leads V1-V3 together with coved type ST-segment elevation more than 2 mm.^[1,2] Together with these typical

ECG tracings, any one of the characteristic clinical findings establish the diagnosis of Brugada syndrome. In patients with suspect Brugada syndrome without typical ECG findings (type 2 and type 3) ajmaline challenge test can be applied to support the diagnosis.

Majority of the patients with suspect diagnosis of Brugada syndrome are asymptomatic. Tachyarrhythmic conditions as polymorphic VT or VF frequently cause syncope or sudden cardiac death.^[1,2] However, rarely monomorphic VT is seen in patients with Brugada syndrome. In an electrophysiological study performed in a patient diagnosed as Brugada syndrome, development of VF following extra stimulation applied on the right ventricular outflow tract, and emergence of monomorphic VT induced by isoprotorenol administered after defibrillation were reported.^[4] Still in another patient with Brugada syndrome, authors reported fatal electrical storm during the febrile state of the patient caused by monomorphic VT.^[5]

Although ajmaline challenge test is recognized as a safe test, development of polymorphic VT, and rarely monomorphic VT, and VF has been reported during ajmaline test.^[6-10] In a study by Veltmann et al performed on 667 patients, ventricular tachycardia related to ajmaline test was seen in only 2 (0.3 %) patients. Transient polymorphic VT was observed in one of these patients, while the other patient experienced sudden – onset VF.^[6] Still in another study carried on 158 patients, polymorphic VT was seen in only 2 (1.3 %) patients.^[7] In a case presentation published in the year 2000, following ajmaline challenge test, development of firstly transient, then sustained monomorphic VT was reported^[8] In another study on ajmaline challenge

test, development of transient monomorphic VT (triple extrasystoles in one patient, and in the other patient priorly triple, then quadruple extrasystoles) was reported.^[9] During an electrophysiological study performed on a patient by Karaca ve et al.^[11] the investigators had priorly observed monomorphic VT, however after administration of propafenone, the patient developed polymorphic VT. In our case, we noted development of monomorphic VT starting immediately after ajmaline test and lasting for nearly 3 or 4 minutes. A case with long-lasting monomorphic VT, without any preceding polymorphic VT episode has not been cited in the literature so far.

We think that our study has a few striking limitations related to our case. Priorly, only limb- lead ECGs were obtained during wide QRS complex tachycardic episodes. Unfortunately, at the 4th minute of the ajmaline challenge test, following a typical ST-segment elevation, tachycardias were observed just before termination of ECG recordings. At that time, chest electrodes of the patient were removed. With the onset of tachycardias, in consideration of potential requirement of the electrical cardioconversion, chest electrodes were not placed again, and ECG tracings were recorded using limb leads. Therefore 12-lead ECG recordings of our case could not be obtained. However when especially aVR, D1, and aVL tracings are examined (Figure 1) alteration in the electrical axis of the heart during tachycardic episodes can be seen. This finding, confirms that wide QRS complex tachycardia observed in our case was of monomorphic VT type. The second limitation of ours, is that we can not exactly determine whether this monomorphic VT is of idiopathic type, related to the impact of ajmaline in a patient who was receiving amiodarone or a

rare case of Brugada syndrome. Failure to trigger arrhythmia by electrophysiological means might mostly rule out supraventricular arrhythmias induced by reentrant tachycardias. However, some types of idiopathic VTs can not be induced by extra stimuli. Therefore we can not say definitely that these tachycardic episodes do not represent idiopathic VT. On the other hand type 1 ECG tracings elicited after ajmaline challenge test in a patient who had previously suffered from episodes of syncope reinforce our diagnosis of Brugada syndrome.

It is recognized that in Brugada syndrome, a voltage gradient develops between epicardium, and endocardium as a result of premature inactivation of Na⁺ channels secondary to gene mutation. Experiments performed on dogs have demonstrated that administration of class 1 antiarrhythmics decreases duration of epicardial action potential. These reentry loops developed in response to myocardial voltage gradient can lead to polymorphic VT or VF.^[1,2] It is not possible for us to explain precisely the mechanism involved in the development of monomorphic VT instead of polymorphic VT during ajmaline challenge test. However, a possible mechanism that might explain monomorphic VT involves a probable alteration in the electrical pathways in the myocardium caused by these two antiarrhythmic drugs when ajmaline challenge test was performed on the patient who had previously used amiodarone. Besides, another explanatory mechanism is that amiodarone prescribed to our patient might “organize” reentry loops leading to monomorphic VT.

In conclusion, as is seen in our case with monomorphic VT, it should not be forgotten that during ajmaline challenge test ventricular tachyarrhythmias or AV blocks might develop. Therefore, it will

be appropriate to perform ajmaline challenge test in the electrophysiology laboratory with a defibrillator, and facilities to perform transient pacing or overdriving pace in case of need.

Conflict of Interest: none declared

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Anahtar sözcükler: Ajmalin / yönetim ve dozaj / yan etkiler; Brugada sendromu; elektrokardiyografi; taşikardi, ventrikül / kimyasal nedenlerle oluşmuş.

Key words: Ajmaline / management and dosage / adverse effects; Brugada syndrome; electrocardiography; tachycardia, ventricle / chemically induced.