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Coexistence of Unstable Angina Pectoris and Wolff-Parkinson-White Syndrome Developed After Consumption of Energy Drink

Enerji İçeceği Tüketimi Sonrası Gelişen Unstabil Anjina Pectoris ve Wolff-Parkinson-White Sendromu Birlikteliği

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Abstract

Interest in energy drinks and their consumption are increasing in recent years. The underlying motivation for the users is mostly enjoyment or enhancement of performance and attentiveness. However; energy drinks also have unwanted side effects such as cardiac symptoms. A small amount of data is available about the cardiac problems caused by energy drinks. Wolff-Parkinson-White syndrome is a congenital heart disease, causing tachycardia and very rarely sudden death. In this case, we aimed to represent a young case diagnosed with unstable angina pectoris and intermittent Wolf-Parkinson-White syndrome, developed after consumption of energy drink.

Key Words: Angina, Energy Drink, Caffeine, Wolf-Parkinson-White Syndrome

Öz

Enerji içeceklerine ilgi her geçen gün artmakta ve enerji içeceği tüketimi de buna paralel olarak artmaktadır. Kullanıcılar çoğunlukla keyif almak, performans ve dikkati artırmak amacıyla enerji içeceklerini kullanmaktadır. Ancak enerji içecekleri istenmeyen kardiyolojik olaylara neden olabilirler. Literatürde enerji içeceklerinin yol açtığı kardiyolojik sorunlar az da olsa yayınlanmaktadır. Wolff-Parkinson-White sendromu taşikardilere neden olan, seyrek olarak ani ölümün görülebilebileceği doğumsal bir kalp hastalığıdır. Bu olguda enerji içeceği tüketilmesi sonrası unstabil anjina pektoris ve intermitant Wolf-Parkinson-White sendromu saptanan genç bir olguyu sunduk.

Anahtar Kelimeler: Anjina, Enerji içeceği, Kafein, Wolf-Parkinson-White Sendromu

Introduction

Consumption of energy drinks (ED) in the world and in our country is increasing every year. The companies rely on allegations that consumption of ED is enhancing physical and emotional stability and concentration and increase their market shares. However relation of ED with cardiovascular diseases has been started to be discussed almost on the day it was started to be consumed. It is used especially by young and adolescent people as a support to boost their physical and mental performance. Although ED contains many ingredients, the substance that was

heavily accused of causing an increase in the sympathomimetic activity in cardiac events was caffeine (1,2). In spite of the fact that; exact mechanism operating in the diseases developing from cardiovascular problems related to consumption of ED cannot be explained fully, various cases had been reported, including Acute Coronary syndrome, cardiovascular arrest, supra-ventricular and ventricular arrhythmia (3-6). Wolff-Parkinson-White (WPW) syndrome is identified by short PR interval and delta wave in electrocardiography (ECG) and may cause paroxysmal tachycardia including death in rare cases. In this study, we aimed to present a case of 34-year-old male patient, admitted to the emergency department because of

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tachycardia and chest pain after consumption of ED (Redbull®) containing high level of caffeine and on whom a percutaneous coronary intervention was performed with the diagnoses of unstable angina pectoris and intermittent WPW.

Case Report

Thirty four -year-old male patient admitted to the emergency department of our hospital with the complaint of tachycardia and feeling of a burning sensation in the chest. He was living abroad and was visiting Turkey for summer holiday. Two weeks before, he admitted to cardiology outpatient clinic with a complaint of atypical chest pain. Nothing suspicious was detected in the ECG, transthoracic echocardiography (TTE), and laboratory tests performed on the patient and no additional cardiac intervention was planned. Three hours after drinking two boxes of ED containing high amount of caffeine, tachycardia, shortness of breath, and chest pain has been started. These symptoms were continued, he admitted to the emergency department. Patient's past medical history was positive for only smoking and ED consumption. On initial evaluation, blood pressure was 90/60 mmHg, heart rate was 180 bpm, body temperature was 36.7 °C, respiratory rate was 18/minute, and peripheral pulses were clear. No additional sound or murmur was heard in cardiovascular examination. Systemic and neurological examinations revealed normal results. No pathology was detected as a result of biochemical analyses, hemogram, and thyroid function tests. In the patient's ECG with 12 derivations (Figure 1a), was consistent with wide QRS tachycardia. The patient was hemodynamically stable, 300 mg intravenous amiodarone infusion was administered. In a short time, sinus rhythm was achieved in ECG. However, a careful examination of ECG revealed small delta waves (Figure 1b) and so old ECGs were examined more carefully once again. Then

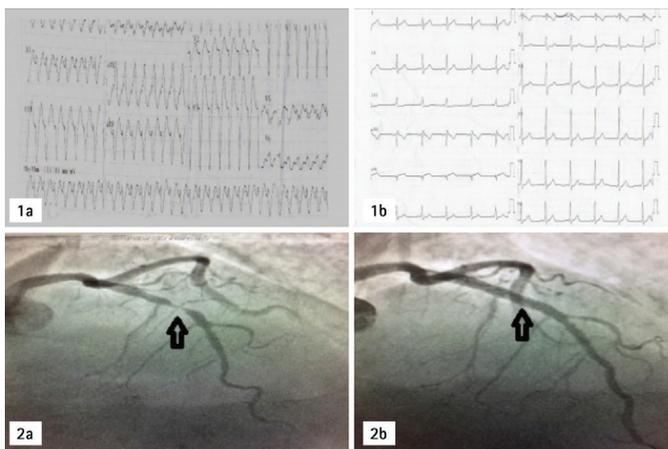


Figure 1, 2: 1a) Observance of tachycardia with expanded QRS in the patient's electrocardiography taken with 12 derivations 1b) emergence of delta waves, 2a) in coronary angiography, 95% obstruction was detected in left front artery body, 2b) final view taken after stent implantation

no delta waves were determined in previous ECGs. Thus, it was decided that these symptoms may be caused by intermittent WPW syndrome manifesting after consumption of ED. But the patient was still suffering chest pain, although normal sinus rhythm was achieved in ECG. The patient was complaining about a chest pain felt as a pressure and on TTE an apical hypokinesia was detected. Consequently, the patient was hospitalized in coronary intensive care unit, and coronary angiography was planned for the diagnosis of unstable angina pectoris. 95% obstruction was detected in the left anterior descending (LAD) artery body in the coronary angiography (Figure 2a). 200 µg intracoronary nitroglycerin was administered several times and coronary vasospasm was eliminated. After repeating the image from different angles, the lesion was finally decided to be serious. Through a direct stent method, one 3.0x16 mm sirolimus eluting coronary stent (Cre8 coronary stent system, CID/Alvimedica, Saluggia, Italy) was implanted (Figure 2b). In the follow-up, no chest pain and tachycardia was detected. But intermittent WPW syndrome was sometimes monitored in later ECGs. It was concluded that reason for tachycardia with wide QRS complex was intermittent WPW syndrome triggered by ED and an electrophysiological study (EPS) was performed on the patient before he was discharged from the hospital. Upon a detailed examination of ECG, it was thought that accessory path was the left posterolateral pathway. As a result of coronary sinus cannulation, it was proven that problem was related to posterolateral accessory path. On this zone, radiofrequency ablation was performed with a degree of 50 w/60 by using retrograde aortic method. On the 3rd second of ablation, continue atrium and ventricular wave records were started. After applying ablation for 60 seconds on this zone, the patient was kept on the stretcher for 30 minutes. As no delta wave occurred again, the procedure was successfully completed (Figure 3). The patient was discharged with a prescription of acetylsalicylic acid 100 mg/day, atorvastatin 40 mg/day and ticagrelor 90 mg twice daily. He was advised not to consume any ED. As a result of follow-up treatment for 3 months, rhythm Holter analysis of 72 hours was repeated for 2 times with no diagnosis of preexcitation syndrome.

Discussion

ED contain caffeine, taurine, vitamin B, sugar, and artificial sweeteners. These drinks contain caffeine in different levels from 80 mg up to 150 mg provided by various brands. Negative cardiac effects of ED were initially stated to be related to caffeine. Due to its inherent characteristic, increasing caffeine levels in the drink makes it more enjoyable to consume. Consuming only one box of ED daily does not mean caffeine overdose. However, if two or more boxes are consumed, caffeine overdose will be the natural result (1). Since most of the packing

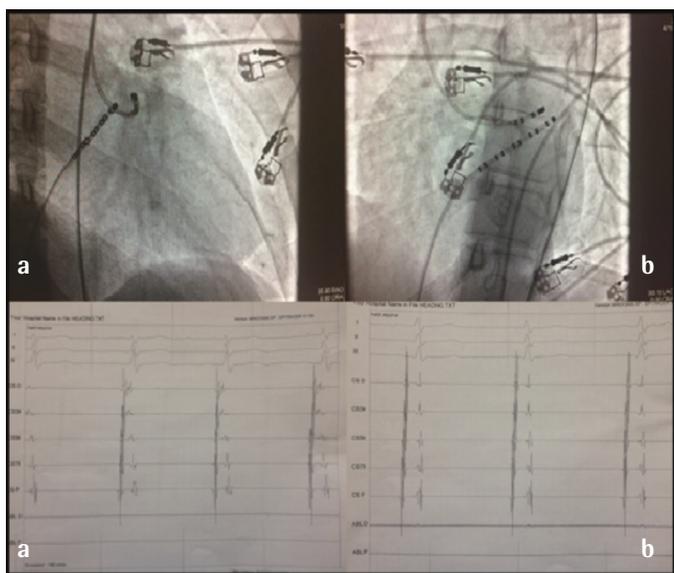


Figure 3: Elimination of right anterior oblique (a) and left anterior oblique (b) images respectively and of continuum AV record during electrophysiological study performed on the patient.

contains 2 servings, caffeine amount consumed can reach to 300 mg. And if it is consumed with alcohol and stimulants, the problem gets more complex. Caffeine absorption is completed 1–2 hours after it is taken in drinks. Caffeine increases calcium levels in the cell. These effects result in positive inotropic and chronotropic effects, an increase in sensitivity of the myofilaments to calcium, and potential susceptibility to cardiac arrhythmias. They increase flow rate of the heart and oxygen consumption (1,2). Like in addictive drugs, caffeine cause release of excessive amounts of dopamine, serotonin, noradrenalin and adrenalin and also prolonged effects. In an invasive electrophysiology study performed on humans, the researchers correlated shortening of refracting period of right atrium, atrio-ventricular node and right ventricular by caffeine to the catecholamine discharge (1). In short, a high dose of caffeine may increase work burden of the heart and arterial tension levels and cause supraventricular/ventricular arrhythmias and cardiac incidents (3,4,5). It is also stated that certain people may be more sensitive and genetically more susceptible to arrhythmias than others. Coronary vasospasm (3), ST elevation myocardial infarction (5), aortic aneurysm dissection (6), cardiac arrest (4), cardiomyopathy, prolonged QT interval incidents are reported in connection with consumption of ED (7). The probable mechanism involved in the development of myocardial infarction after ED consumption is endothelial dysfunction and platelet aggression caused by arachidonic acid (7,8,9). Especially after consumption of two or more boxes of ED, a male patient aged seventeen suffered from coronary artery spasm and another patient suffered from cardiac arrest (3). Berger and Alford (4) reported a young patient who suffered anteroseptal myocardial infarction after consuming seven or eight boxes of ED. In a similar case,

a 24 year old patient who suffered ST segment elevation after drinking 20 boxes of ED was reported dead after entering into ventricular fibrillation during a primary percutaneous coronary attempt (5). There are case presentations mentioning about secondary ventricular arrhythmias and sudden cardiac deaths caused by consumption of ED. In a case reported by Nagajothi et al. (10) a 23 years old male applied to the hospital with tension in the chest developing after consumption of ED, and ECG revealed tachycardia with QRS complex in 219 bpm ventricular speed. Since carotid sinus massage and valsalva maneuver was unsuccessful, tachycardia was resolved by administration of adenosine. In another case reported by Goldfarb et al. (11) after a cardioversion applied in the emergency service due to ventricular fibrillation, the patient told that he smoked marijuana and consumed ED rich in caffeine but no pathology was detected in the cardiac magnetic resonance imaging, coronary angiography and EPS. Rutledge et al. (12) reported a young adult male who was brought to the emergency service because of ventricular fibrillation after consuming ED with alcohol. Following the resuscitation, ECG revealed type 1 Brugada (12). In our case, although the patient was not diagnosed for cardiac pathology in his examination made in the outpatient clinic two weeks ago, angina and tachycardia attacks started after consumption of ED with high caffeine level. In the emergency department, tachycardia with a wide QRS complex was detected and then resolved with antiarrhythmic drug. Although initially ventricular tachycardia was suspected due to a severe lesion in the patient's LAD artery and wide QRS tachycardia, an examination of ECG revealed supraventricular tachycardia. Even long RP tachycardia was detected and a concern was disclosed about potential concealed transmitted accessory path and resulting antidromic atrioventricular reentrant tachycardia or aberrant transmitted atrial tachycardia. As a matter of fact, observance of manifested WPW symptoms in sinus rhythm after amiodarone infusion supported our preliminary diagnosis of antidromic transmitted tachycardia. Social position and habits of the patient forced us to finally decide to perform accessory path ablation on the patient and thus WPW ablation was made. Consumption of ED with high caffeine levels caused unstable angina pectoris and then resulted first in antidromic transmitted tachycardia and later WPW manifest in the sinus rhythm ECG. It also facilitated antegrade and retrograde transmission in accessory path. As distinct with the cases presented in the literature, we came to conclude that ED consumption triggered WPW syndrome causing both unstable angina and antidromic tachycardia.

With an increase in the consumption of ED in the society, such cases may be seen more frequently. ED consumption seems to increase as sufficient data could not be gathered yet about the negative effects of ED on human health although consumption levels increased recently. Therefore, comprehensive experimental and clinical studies on ED consumption could

serve as a proper tool to remedy for lack of enough information about this subject. In this process, it is the duty of the physicians to warn people about potential side effects of ED and the risks arising from its consumption and mention the deadly diseases that could be triggered by ED.

Ethics

Informed Consent: It was taken.

Authorship Contributions

Surgical and Medical Practices: V.D., Y.Ç., Y.T., Concept: V.D., Y.Ç., Ş.H., H.E., Design: V.D., Y.Ç., Ş.H., Data Collection or Processing: V.D., Y.Ç., Analysis or Interpretation: Y.T., V.D., H.E., Literature Search: V.D., Y.Ç., Ş.H., Writing: V.D., Y.Ç., H.E.

Conflict of Interest: No conflict of interest was declared by the authors.

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