

Spontaneous coronary artery dissection in a healthy adolescent following consumption of caffeinated “energy drinks”

Sağlıklı bir adolesanda kafeinli “enerji içeceği” tüketimi ardından gelişen spontan koroner arter diseksiyonu

Nihat Polat, M.D., İdris Ardiç, M.D.,# Murat Akkoyun, M.D.,# Ertan Vuruşkan, M.D.*

Department of Cardiology, Dicle University Faculty of Medicine, Diyarbakir;

#Department of Cardiology, Sutcu Imam University Faculty of Medicine, Kahramanmaraş;

*Department of Cardiology, Dr. Ersin Arslan State Hospital, Gaziantep

Summary– Spontaneous coronary artery dissection (SCAD) is a rare and uncommon case of sudden cardiac death and acute coronary syndrome. Herein, we present a 13-year-old boy with chest pain who was diagnosed with acute ST-segment elevation myocardial infarction associated with SCAD, possibly caused by the consumption of an energy drink, which has not been reported previously in the pediatric age group. On coronary angiography, the left anterior descending artery showed extensive dissection from the distal part of the vessel. Based on the morphology of the vessel with a dissection and TIMI flow grade III, it was decided to manage this patient conservatively with close follow-up. The aim of this report is to highlight the risks associated with the consumption of caffeinated energy drinks in children.

Spontaneous coronary artery dissection (SCAD) is a rare and uncommon cause of sudden cardiac death and acute coronary syndrome.^[1] SCAD remains an unclear etiopathological entity. The most common pathologies associated with SCAD are coronary atherosclerosis and vascular changes occurring during the peripartum period. SCAD may be associated with Marfan’s syndrome, Ehlers-Danlos disease, intensive exercise, cocaine abuse, and female hormonal treatments such as oral contraceptives, although in some cases no predictor could be identified.^[1] However, a large number of cases must be classified as idiopathic because no underlying condition can be detected.

Özet– Spontan koroner arter diseksiyonu (SKAD) ani kardiyak ölüm ve akut koroner sendrom ile kendini gösteren nadir bir durumdur. Burada, çocukluk yaş grubunda daha önce tanımlanmamış olan göğüs ağrısı ile başvuran SKAD ile ilişkili akut ST-segment yükselmeli miyokart enfarktüsü tanısı konan 13 yaşında bir erkek olgu sunuldu. Koroner anjiyografide sol ön inen arterde, damarın distal kısmında geniş diseksiyon izlendi. Diseksiyon yapısı ve damar morfolojisi ve distal TIMI III akımı dikkate alınarak, hastaya konservatif tedavi ile yakın takip kararı verildi. Bu yazının amacı, çocuklarda kafeinli enerji içecekleri tüketimi ile ilişkili riskleri vurgulamaktır.

Caffeine-containing beverage consumption in children and adolescents in the last 30 years continues to grow exponentially. In the

United States, adolescent caffeine intake averages 60 to 70 mg/day and ranges up to 800 mg/day.^[2] Most caffeine intake among youth comes from soda; however, energy drinks are becoming increasingly popular.

We report a case of ST-segment elevation myocardial infarction, in which SCAD may have been caused by the consumption of an energy drink.

Abbreviations:

ECG	Electrocardiogram
LAD	Left anterior descending
SCAD	Spontaneous coronary artery dissection
TIMI	Thrombolysis in myocardial infarction
TTE	Transthoracic echocardiography

Received: February 06, 2013 Accepted: May 02, 2013

Correspondence: Dr. İdris Ardiç. Sütçü İmam Üniversitesi Tıp Fakültesi, Kardiyoloji ABD, Kahramanmaraş.

Tel: +90 344 - 225 75 75 / 532 e-mail: idrisardic@yahoo.com

© 2013 Turkish Society of Cardiology



CASE REPORT

A previously healthy 13-year-old boy was admitted to our clinic after presenting with acute-onset, “crushing,” mid-sternal chest pain over a period of about two hours. He had no history of diabetes, hypertension, hyperlipidemia, or cigarette smoking. He had no family history of familial hypercholesterolemia, early coronary artery disease, or sudden death. He denied use of cocaine, amphetamines, hormones, steroids, alcohol, or other recreational drugs. The patient had ingested an energy drink for the first time the previous night. About 8 hours after the high-energy drink consumption, the patient’s chest pain started.

Physical examination revealed a well-developed teen in moderate distress. His blood pressure was 120/70 mmHg, heart rate 80 beats/min, and respiratory rate 16 breaths/min. He had no jugular venous distention, and his lungs were clear to auscultation in all fields. Cardiac auscultation revealed an S4 gallop with a normal S1 and S2. The electrocardiogram (ECG) revealed sinus rhythm with 2- to 3-mm ST-segment elevations in leads II, III, aVF, and V3

through V5 (Fig. 1a). The transthoracic echocardiography (TTE) showed left ventricular ejection fraction estimated to 0.54 and moderate apical hypokinesis. He had been given aspirin, subcutaneous enoxaparin, sublingual nitroglycerin, enalapril, and metoprolol at presentation. After treatment, the patient’s chest pain was relieved. Initial laboratory studies, within 4 hours of the onset of his symptoms, were normal, with a white blood cell count of 7,260 cells/mm³, myoglobin level of 70 U/L (normal range 0-150 U/L), creatine kinase-MB fraction of 3.2 ng/ml (normal range: 0-5 ng/mL), and mildly elevated troponin I level of 0.65 ng/mL (normal range 0-0.06 ng/mL). The patient’s blood count, cholesterol, hypercoagulability panels, and kidney and thyroid function tests were normal. Chest X-ray was unremarkable, and found to be normal. The patient’s chest pain decreased after medical treatment, and thus no thrombolytic therapy was given. Four hours of recorded ECG revealed no dynamic changes with respect to the baseline ECG (Fig. 1b). The troponin-I value after 24 hours was 3.96 ng/mL. Dynamic T-wave changes were observed in ECG recording leads V3-V5 (Fig. 2). For these reasons, the

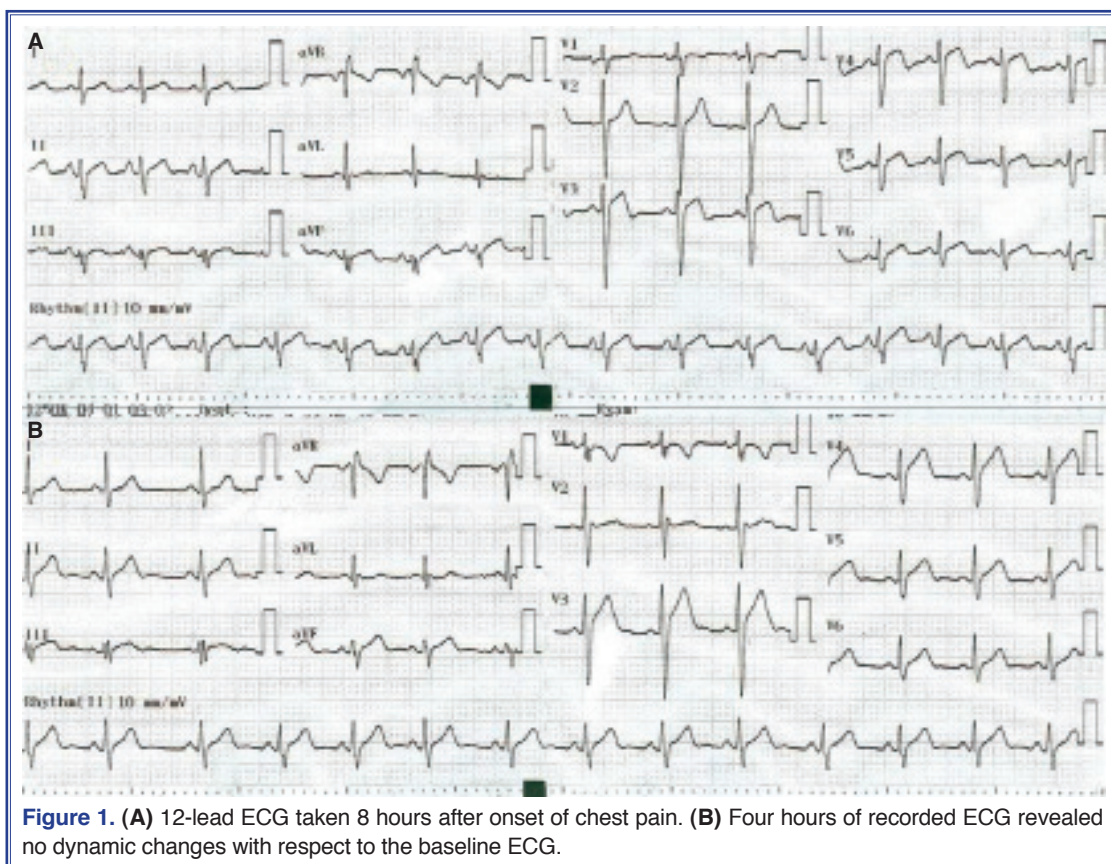


Figure 1. (A) 12-lead ECG taken 8 hours after onset of chest pain. (B) Four hours of recorded ECG revealed no dynamic changes with respect to the baseline ECG.

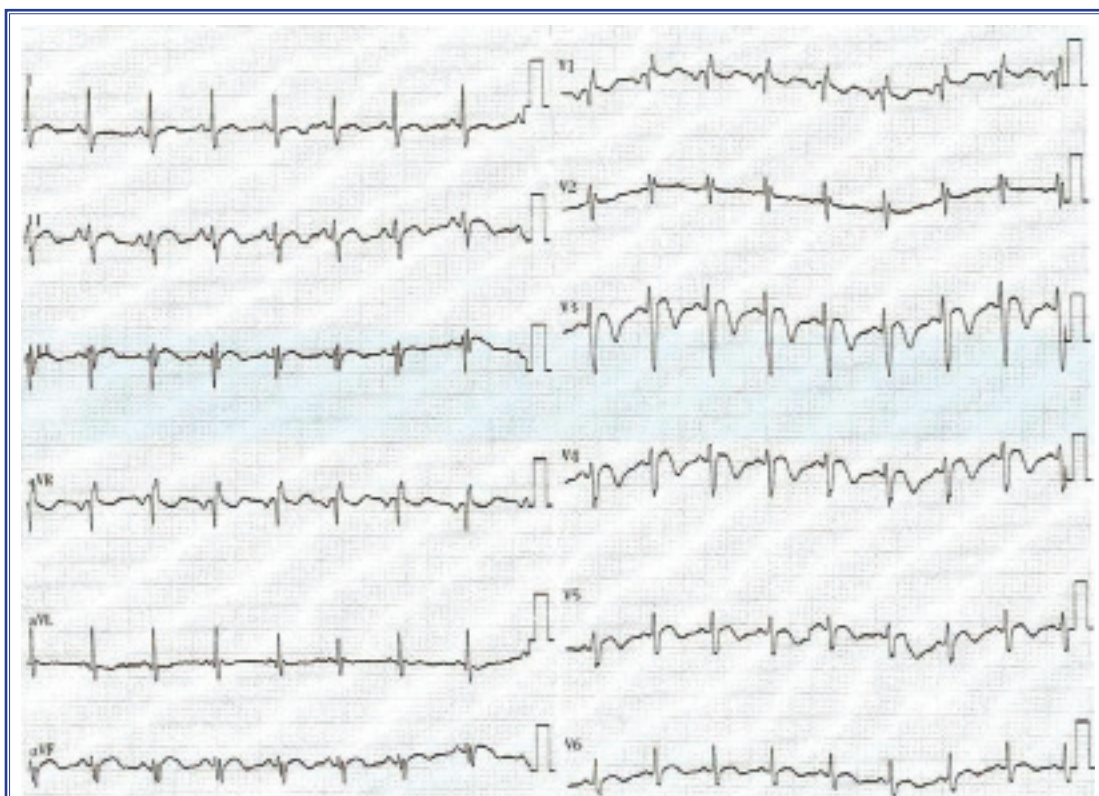


Figure 2. 12-lead ECG 24 hours after onset of chest pain showed dynamic T-wave changes in leads V3-V5.

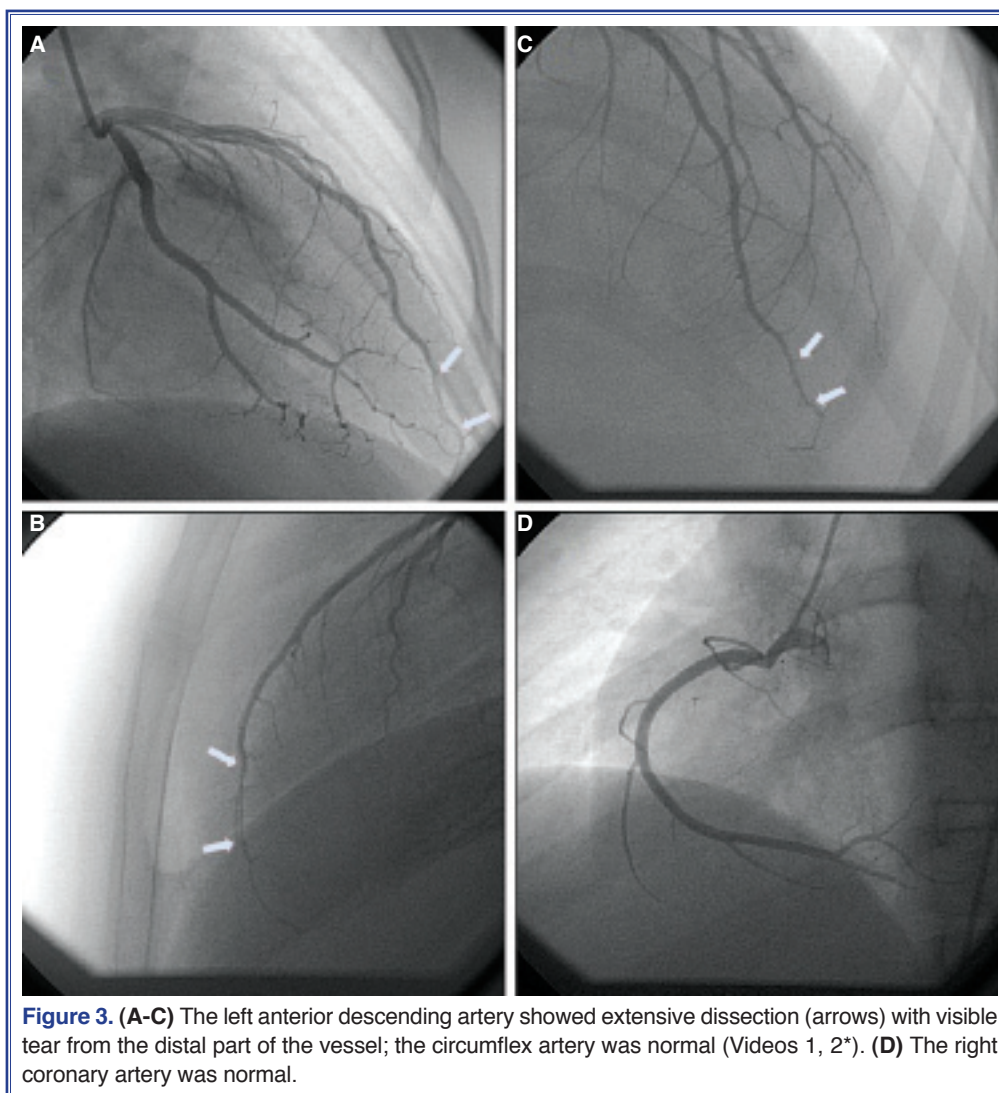
patient was transferred to a tertiary referral center for coronary angiography. The left anterior descending (LAD) artery showed extensive dissection with a visible tear from the distal part of the vessel. The TIMI (thrombolysis in myocardial infarction) flow grade was III (Fig. 3, Videos 1, 2*). The right coronary artery and the circumflex artery were normal. Based on the morphology of the vessel with a dissection and TIMI III flow grade, it was decided to manage this patient conservatively with close follow-up. We continued low-molecular-weight heparin, antiplatelet therapy, and enalapril. At the follow-up examination one month later, the patient had no chest pain. Acetylsalicylic acid and enalapril treatment was continued. Follow-up TTE revealed normal left ventricular function, with resolution of his apical hypokinesis.

DISCUSSION

A possible role of the consumption of caffeinated energy drinks in triggering SCAD events is described in this case. SCAD is a rare and uncommon cause of sudden cardiac death and acute coronary syndrome.^[1] However, a large number of cases must be classified

as idiopathic because no underlying condition can be detected. The female to male ratio is 2:1, and the dissection is diagnosed most frequently in the left coronary artery.^[3]

Coronary artery dissection is characterized by a separation of the layers of the artery wall. This results in a false lumen or an intramural hematoma in the area of the media. The clinical presentation of SCAD depends on the extent and severity of the dissection, and ranges from unstable angina to sudden cardiac death. Recognition of the dissection can be quite difficult and may require multiple angiographic views or intravascular ultrasound to confirm the diagnosis. We decided not to perform intravascular ultrasound because the dissection line in the LAD artery was clearly visible on angiography. There is no specific guideline to treat SCAD. If the vessel is open and the flow normalized at the time of angiography, it is defensible to treat the dissection conservatively. Good angiographic and clinical outcomes have been described with medical treatment only.^[4] Therefore, we decided to manage this case conservatively with close follow-up.



“Energy drinks” are beverages that contain caffeine, taurine, vitamins, herbal supplements, and sugar or sweeteners, and they are marketed to improve energy, weight loss, stamina, athletic performance, and concentration.^[5] Energy drinks contain three-to-four-fold the caffeine as a typical soda and promise to boost performance and enhance metabolism.^[5] Caffeine is a well-known and commonly used neurostimulant. The mechanism of action is thought to be direct adenosine receptor stimulation, in addition to the effects on monoamine neurotransmitters.^[6] Moreover, caffeine is a cyclic adenosine monophosphate (cAMP) phosphodiesterase inhibitor and can also cause the release of intracellular calcium stores. Documented adverse cardiovascular effects include tachycardia, extrasystoles, increased stroke volume, and possibly other arrhythmias. Caffeine may also

enhance the inotropic effect of β -adrenergic agents. Caffeine has been shown to directly stimulate cardiac function while dilating blood vessels, and appears to have only mild effects on blood pressure.^[6] Caffeine is known to trigger a heart rhythm disorder. In some cases, the caffeine is thought to cause serious adverse effects like myocardial infarction and cardiac arrest.^[7] The use of caffeine can cause increased sympathetic nerve activity and raised blood pressure in humans.^[8]

The endogenous circadian rhythm affects many physiological and biochemical parameters. This is associated with rises in plasma catecholamines and cortisol and increased platelet aggregability in the morning.^[9] The energy drink consumed by our patient contained 80 mg of caffeine (equivalent to one cup of espresso) per can. The drink also contains high doses

of taurine (an amino acid) and glucuronolactone (a glucose metabolite), neither of which is considered to have significant toxicity, although there is a paucity of data. Chest pain in our case, in accordance with the circadian rhythm, began around 7 a.m. This indicates that the caffeine in energy drinks has increased circadian rhythm impact on physiological and biochemical parameters and may have triggered the coronary artery dissection. It is impossible to draw conclusions on this issue with a single case report; more studies about the effects of caffeine on the vessel wall are needed.

In conclusion, energy drinks may be one of the factors leading to SCAD. Energy drinks, especially in children, can lead to serious adverse events, as seen in the presented case.

Conflict-of-interest issues regarding the authorship or article: None declared.

***Supplementary video files associated with this article can be found in the online version of the journal.**

REFERENCES

- Butler R, Webster MW, Davies G, Kerr A, Bass N, Armstrong G, et al. Spontaneous dissection of native coronary arteries. *Heart* 2005;91:223-4. [CrossRef](#)
- Pollak CP, Bright D. Caffeine consumption and weekly sleep patterns in US seventh-, eighth-, and ninth-graders. *Pediatrics* 2003;111:42-6. [CrossRef](#)
- Verma PK, Sandhu MS, Mittal BR, Aggarwal N, Kumar A, Mayank M, et al. Large spontaneous coronary artery dissections-a study of three cases, literature review, and possible therapeutic strategies. *Angiology* 2004;55:309-18. [CrossRef](#)
- Maeder M, Ammann P, Angehrn W, Rickli H. Idiopathic spontaneous coronary artery dissection: incidence, diagnosis and treatment. *Int J Cardiol* 2005;101:363-9. [CrossRef](#)
- Seifert SM, Schaechter JL, Hershorin ER, Lipshultz SE. Health effects of energy drinks on children, adolescents, and young adults. *Pediatrics* 2011;127:511-28. [CrossRef](#)
- Samenuk D, Link MS, Homoud MK, Contreras R, Theoharides TC, Wang PJ, et al. Adverse cardiovascular events temporally associated with ma huang, an herbal source of ephedrine. *Mayo Clin Proc* 2002;77:12-6. [CrossRef](#)
- Berger AJ, Alford K. Cardiac arrest in a young man following excess consumption of caffeinated "energy drinks". *Med J Aust* 2009;190:41-3.
- Corti R, Binggeli C, Sudano I, Spieker L, Hänseler E, Ruschitzka F, et al. Coffee acutely increases sympathetic nerve activity and blood pressure independently of caffeine content: role of habitual versus nonhabitual drinking. *Circulation* 2002;106:2935-40. [CrossRef](#)
- Muller JE, Abela GS, Nesto RW, Tofler GH. Triggers, acute risk factors and vulnerable plaques: the lexicon of a new frontier. *J Am Coll Cardiol* 1994;23:809-13. [CrossRef](#)

Key words: Beverages/adverse effects; caffeine/adverse effects; coronary artery dissection, spontaneous; energy drinks; heart arrest/chemically induced.

Anahtar sözcükler: İçecekler/yan etki; kafein/yan etki; koroner arter diseksiyonu, spontan; enerji içeceği; kalp durması/kimyasal yolla oluşan.