Proximal left anterior descending coronary artery thrombosis and acute anterior myocardial infarction due to energy drink

Enerji içeceğine bağlı proksimal sol ön inen arter trombozu ve ön yüz myokard enfarktüsü

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Abstract
Energy drinks (EDs) are commonly consumed by youth. They are often used as a source of energy in order to enhance physical and mental performance. Case reports, observational studies, and meta-analyses have been reported in order to determine the effects of EDs on cardiovascular changes. The detrimental effects of EDs are cardiac arrhythmias, myocardial infarction, prolonged QT interval, aortic dissection, and death. A case with proximal left anterior descending coronary artery thrombosis and anterior myocardial infarction is reported in this paper.

Keywords: Energy drinks, coronary thrombosis, myocardial infarction.

Case Report
A 25-year-old previously healthy man presented to the emergency department with 2 hours history of chest pain on New Year’s Eve night. His symptom started 3 hours after drinking mixed 3 bottles of energy drink and alcohol. He denied cigarettes, cocaine, and any other illicit drug use. Blood pressure was 120/80 mmHg, pulse 110 bpm, respiratory rate 18 breaths per minute, temperature 37.2°C, and oxygen saturation 98% on room air. Physical examination revealed tenderness over the cardiac region and diaphoresis. Electrocardiogram showed anterior ST elevation myocardial infarction. Coronary angiography revealed proximal left anterior descending coronary artery thrombosis. A diagnosis of acute anterior myocardial infarction was made. The patient was treated with aspirin, heparin, and thrombolysis. He was discharged on warfarin and statins.

other drug uses. There was no family history of premature coronary artery disease.

At admission, his vital signs were blood pressure 115/58 mm Hg, pulse 85, temperature 36 °C, 99% oxygen saturation, and breathing rate was 21 respirations per minute. Physical examination was unremarkable. The electrocardiogram showed marked ST elevation V1-V6 [Fig. 1]. A bedside echocardiogram demonstrated anterior mid-apical segments, apical and interventricular septum hypokinesis, and normal aorta and left ventricular ejection fraction was 40%.

Figure 1. The 12 derivation ECG showed marked ST elevation V1 through V6.

An emergent coronary angiogram demonstrated a thrombus resulting in approximately 80% of stenosis at the proximal left anterior descending [LAD] artery [Fig. 2 a, b, c]. No atherosclerotic lesions, dissection or coronary malformations were identified.

Figure 2. a: Thrombus in proximal LAD.

Figure 2. b: 3,5x28 mm DES was implanted.

Figure 2. c: TIMI 3 flow was achieved.

During coronary angiography, intracoronary tirofiban bolus was administrated and IV infusion was started and drug-eluting stent was successfully implanted at the proximal LAD.

He remained in the hospital for two days after stent placement and experienced no further chest pain. Further laboratory tests, including a lipid profile and coagulation panel, were within normal limits. The patient was discharged to home with a dual antiplatelet therapy.

Discussion

Energy drink consumption is increasing and has become very popular, especially among young people [8]. These are often used to improve weight loss, athletic performance, energy level, concentration, and decrease the aftereffects of alcohol [1]. Almost all EDs have
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the same basic stimulants. Caffeine is one of the main stimulants in EDs. The concentration varies in different products. Mechanisms of action are dose-dependent. Serious toxicities such as seizure and cardiac arrhythmias, seen with caffeine plasma concentrations of 15 mg/L or higher, have caused poisoning or, rarely, death; otherwise, concentrations of 3-6 mg/kg are considered safe. Caffeine concentrations of 80-100 mg/L are considered lethal [9]. Other components like guarana, taurine, theophylline, ginkgo biloba, ginseng, vitamins, L-carnitine etc. are also present in variable amounts [1]. It is difficult to know which component is responsible for the effect of platelet aggregation and endothelial dysfunction.

Energy drink consumption acutely increases platelet aggregation and decreases endothelial function in healthy young adults [3, 10]. Endothelial function acutely becomes worse after drinking energy beverage [11]. Pommerening et al. [3] reported that EDs are associated with increased platelet aggregation via the arachidonic acid pathway within 1 h of consumption. Because this is the same pathway inhibited by aspirin therapy, energy drink consumption may lead to an increased risk for thromboembolic disease and adverse cardiovascular events. EDs may have contributed to triggering platelet hyper-aggregation and endothelial dysfunction, thus promoting the development of coronary thrombosis. In our case, alcohol intake with three different energy drinks at the same time may have triggered thrombus formation and myocardial infarction.

Svatikova et al. [12] performed a randomized, placebo-controlled, crossover trial of 25 healthy volunteers who consumed 16 oz of a commercially available energy drink or placebo (matched in taste and color but without caffeine or stimulants) and studied effects on blood pressure, heart rate, and serum norepinephrine levels. Despite no differences in heart rate between energy drink use and placebo, there was a significant increase in systolic, diastolic, and mean blood pressure with energy drink use as compared to placebo. This was associated with a significant increase in norepinephrine levels after energy drink as compared to placebo. These findings suggest that adrenergic stimulation with EDs may predispose patients to cardiovascular risk and may be particularly concerning in patients with unrecognized cardiac disorders.

Previous case reports had linked EDs with sudden cardiac death, coronary vasospasm, thrombosis and dissection, prolonged QT interval, myocardial ischemia and infarction, supraventricular and ventricular arrhythmias, including ventricular fibrillation, cardiomyopathy, and aortic dissection [12]. We report a case of anterior STEMI with coronary thrombus and obstruction due to temporally drinking commercially available caffeinated EDs. This is the fourth case with coronary thrombus and myocardial infarction in the literature.

The effects of EDs should be better investigated because of their increasing consumption by the public and their potentially lethal effects. Clinicians should be aware of the potential cardiovascular adverse events of EDs in young persons who present with acute cardiovascular problems.

Conflict of Interest: I have no conflicts of interest.

References

