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Case Report

Coronary Artery Thrombosis Associated with Energy Drink Consumption

Abstract

Energy drinks have become readily available and are being utilized globally. However, the potential side effects of their use are not well understood. Most consumers use these beverages with the goal of increasing energy. However, unintended ramifications of energy drinks may include increased sympathetic drive, endothelial dysfunction, and mitigation of coronary blood flow. A few reports have brought to light the rare phenomenon of myocardial infarction occurring secondary to energy drink consumption. We present this case of a young healthy man who suffered a myocardial infarction requiring emergent coronary intervention to contribute to the limited literature available on this topic. The increasing popularity and use of energy drinks necessitates further investigation into their unintended cardiac complications.

Background

Worldwide, consumption of energy drinks (EDs) has become exceedingly popular and beverage sales have evolved into a multi-billion dollar industry [1]. Regular consumption of EDs has been reported as high as 68% in adolescents and 30% in adults [1]. These flavored beverages contain large quantities of artificial caffeine, sugar, taurine, B vitamins, herbal supplements, guarana (natural source of caffeine), glucuronanone and ginseng. The safety of ED consumption is being questioned due to associations with cardiovascular complications, including: elevated arterial pressure, QTc interval prolongation, arrhythmias, coronary artery spasm, coronary artery thrombosis, Takotsubo cardiomyopathy, myocardial infarction, aortic dissection, and sudden cardiac death [2-4]. Rising heart rate, elevated systemic blood pressure, endothelial dysfunction, increased platelet aggregation, and increased blood viscosity are all proposed mechanisms of ED's potential side effects [2,5]. This report describes a case of a healthy young man with no past medical history presenting with an ST-Segment Elevation Myocardial Infarction (STEMI) secondary to thrombosis of the right coronary artery after consuming numerous energy drinks.

Case Presentation

A 28 year old man with no past medical history had been drinking an average of four to five Red Bull energy drinks per evening for several weeks. Shortly after a routine exercise session at the gym, he was feeling well and without any symptoms of fatigue or cramping, when suddenly he experienced acute crushing retrosternal chest pressure and shortness of breath. He contacted emergency medical services and was taken to the nearest emergency room. On arrival, the electrocardiogram (ECG) showed sinus rhythm with hyper-acute T waves in leads II, III, aVF, and ST-segment depression in leads I, aVL, V1, and V2 (Figure 1a). Cardiac enzymes revealed a mildly elevated creatine kinase and normal troponin levels. His urine drug screen was negative for cocaine or amphetamines. He received aspirin 325 mg, clopidogrel 600 mg, atorvastatin 80 mg, and was stated on intravenous unfractionated heparin therapy. A transthoracic echocardiogram demonstrated basal inferior and inferior septal hypokinesis, with a preserved left ventricular ejection fraction of 60%. A repeat ECG showed 3 mm ST-segment elevations with tombstone morphology in leads II, III, aVF and ST segment depression in I and aVL (Figure 1b). The patient underwent emergent left heart catheterization with selective coronary angiography. The left coronary system was normal. However, a complete occlusion of the mid right coronary artery (RCA) was observed with TIMI 0 flow in the mid and distal portions (Figure 2a). Aspiration thrombectomy was performed with removal of a substantial amount of thrombus (Figure 3). Residual 20–30% stenosis was noted in the mid RCA with the aid of intravascular ultrasonography. Post intervention angiography showed good distal runoff with TIMI 3 flow (Figure 2b). Neither angioplasty nor stenting were performed due to the quality of flow, patient's age, and paucity of comorbidities.

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The patient was continued on dual antiplatelet therapy and monitored in the coronary care unit. His creatinine kinase levels peaked at 3,940 U/L, troponin T at 5.27 ng/mL and troponin I at greater than 40 ng/mL. Repeat echocardiogram was performed after catheterization and remained unchanged. The remainder of his lab work was unremarkable. The patient did not develop any further chest pain throughout the hospitalization and was discharged home in stable condition on dual antiplatelet therapy. He has remained free of chest pain for over six months after discharge and continues to abstain from energy drink consumption.

Discussion

The reported toxicities associated with EDs have some physicians raising questions regarding their safety. EDs differ greatly from traditional soft drinks, coffee, and tea in that every ED exceeds the official FDA caffeine concentration limit of 71 mg / 12 fl oz [2]. Case reports have established rare instances of EDs being linked with STEMIs. At least four reports of coronary artery thrombosis triggering STEMIs in the setting of ED consumption have been described in the literature.

The first case of coronary artery thrombosis associated with EDs was published in 2012. It described a 24 year old healthy male that presented to the emergency room with severe retrosternal chest pain, palpitations, nausea and vomiting after consuming three drinks of vodka mixed with an ED [6]. His ECG demonstrated ST-segment elevation in the anterolateral leads and bedside echocardiogram showed apical hypokinesis. Coronary angiography demonstrated a large thrombus occupying most of the length and 70% of the diameter of the left main coronary artery, extending to the ostium of the left circumflex coronary artery with almost 90%

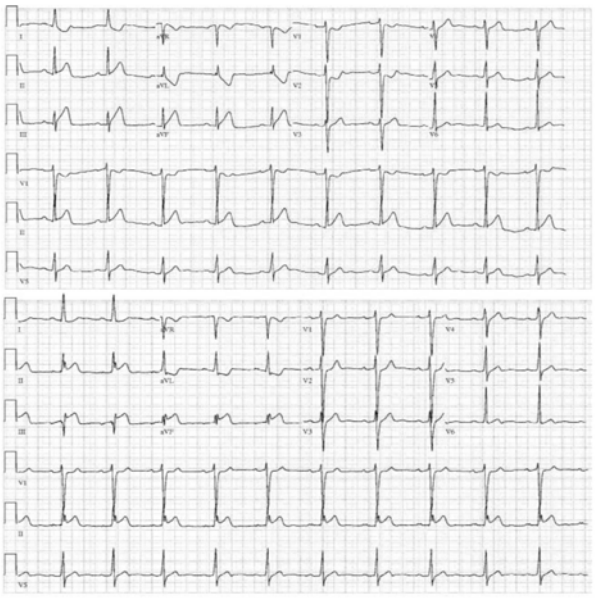

Figure 1: (1a) Initial electrocardiogram with peaked T waves and convex ST-segment elevation in leads II, III, aVF, as well as ST-segment depression in leads I and aVL.
(1b) Repeat ECG with 3 mm ST-segment elevation with 'tombstone' appearance in leads II, III, aVF and ST segment depression in I and aVL.

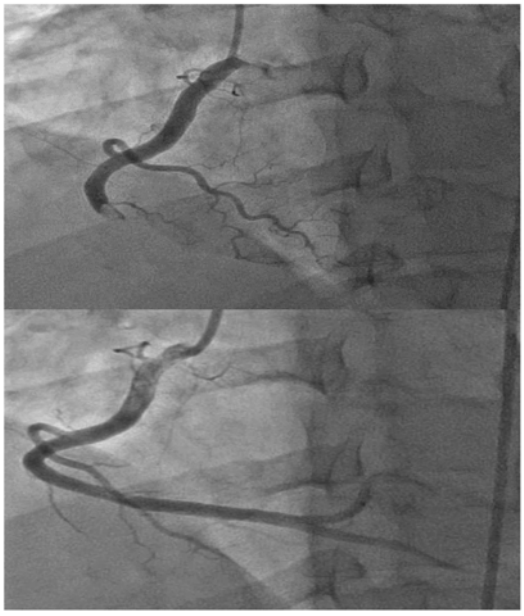

Figure 2: (2a) Coronary angiography revealed complete occlusion of the mid right coronary artery.
(2b) Coronary angiography after thrombectomy revealed TIMI 3 flow distally

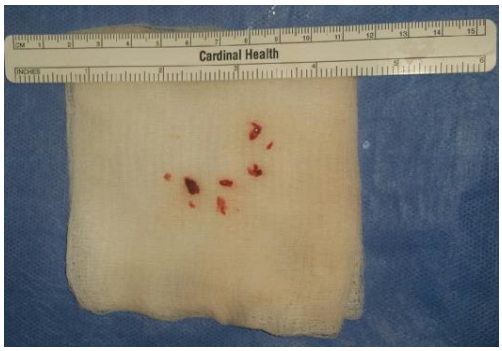

Figure 3: Post aspiration thrombectomy showing a substantial amount of clot removed.

obstruction. A second thrombus occluding the distal LAD was also noted. This patient was placed on an intra-aortic balloon pump and underwent emergent coronary bypass graft surgery.

The first case associating a STEMI with ED consumption was reported in Australia in 2009 [7]. A 28 year old man suffered a cardiac arrest after consuming 7 to 8 canisters of EDs throughout a day of motocross racing [7]. Shortly after completing a race he collapsed and after approximately 20 minutes of cardiopulmonary resuscitation, paramedics arrived and the patient's initial cardiac rhythm was ventricular fibrillation. He was defibrillated twice and normal sinus rhythm was restored. Initially at the hospital, his ECG demonstrated anteroseptal ST-segment elevation with reciprocal inferior ST-segment depression. He received thrombolytic therapy with tenecteplase, loaded with aspirin and clopidogrel, and initiated on an IV heparin infusion before being transferred to a tertiary care center for cardiac catheterization. His peak troponin I was 12.2 mmol/L. Coronary angiogram was normal, and his STEMI and cardiac arrest were attributed to an intense

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vasospasm. The physicians addressing this case associated the event with the high levels of caffeine and taurine in his blood secondary to increased ED consumption.

Two additional cases of STEMI secondary to coronary artery thrombosis in association with ED consumption have been reported [8]. A 32 year-old- man that presented with chest pain after consuming 5 to 6 EDs had ECG findings consistent with STEMI and elevated cardiac enzymes. Coronary angiography revealed two thrombi, the first obstructing 90% of the left main coronary artery, and the second completely occluding the proximal left anterior descending artery [8]. Another case involved a 26 year-old-man who presented with chest pain after consuming 8 to 10 EDs and had cardiac enzyme elevations and ECG changes consistent with a STEMI. Coronary angiography revealed a completely occluded left main coronary artery.

In our case, other etiologies of STEMI were ruled out via past medical history, social history, family history, urine drug screen, and patient age. There has not been a definitive study proving energy drink induced STEMI. However, as evidenced by the previously published reports, this case is consistent with the phenomenon of energy drink induced STEMI. Although a definitive causal relationship cannot yet be established, several prospective studies performed on the cardiovascular and hematological effects of EDs and their components may elucidate a mechanism for this phenomenon.

The patient in our case developed chest pain shortly after exercising. One possible explanation may be acute endothelial dysfunction. In healthy subjects who consume caffeine and then exercise afterwards, myocardial blood flow may be significantly reduced [9]. In two studies using positron emission tomography (PET) as the measuring tool, exercise induced myocardial blood flow response was significantly decreased by 14% ($P < .05$) and 22% ($P < .01$) after caffeine consumption [9]. Furthermore, studies that utilized flow mediated dilation of the brachial artery as a surrogate for coronary artery endothelial function, demonstrated that during dynamic exercise, caffeine attenuated the increase in forearm blood flow by as much as 53% [9-13]. The underlying mechanism for these findings may be caffeine's attenuation of the usual adenosine and sympathetic mediated increased vasodilatation that occur to match the increase in cardiac work during exercise [9].

Interestingly, a study was conducted on 50 healthy subjects with a median age of 22 years. The study group consumed 250 mL of a sugar-free energy drink and the control group consumed 250 mL of carbonated water [5]. Platelet aggregation, endothelial function and mean arterial pressure were tested before and one hour after consumption of the respective beverages. In the ED group, there was a significant increase of approximately 13% in platelet aggregation by adenosine diphosphate-induced optical aggregometry [5]. Endothelial function assessed by peripheral artery flow mediated dilation was acutely and significantly decreased after ED consumption and mean arterial pressure was significantly increased [5].

The increased prevalence of ED consumption in recent years, has seen a rising incidence of emergency room visits and serious cardiovascular events [2,4]. There is growing evidence suggesting that these beverages are not innocuous performance enhancers and may harbour significant risks to healthy young people [2,14]. More research in the field is vital to developing a better understanding of their impact on risk for cardiovascular events.

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