

Circulation

JOURNAL OF THE AMERICAN HEART ASSOCIATION



Left Main Coronary Artery Acute Thrombosis Related to Energy Drink Intake

Alexandre M. Benjo, Andrés M. Pineda, Francisco O. Nascimento, Carlos Zamora,
Gervasio A. Lamas and Esteban Escolar

Circulation 2012, 125:1447-1448

doi: 10.1161/CIRCULATIONAHA.111.086017

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX
72514

Copyright © 2012 American Heart Association. All rights reserved. Print ISSN: 0009-7322. Online
ISSN: 1524-4539

The online version of this article, along with updated information and services, is
located on the World Wide Web at:

<http://circ.ahajournals.org/content/125/11/1447>

Subscriptions: Information about subscribing to *Circulation* is online at
<http://circ.ahajournals.org/subscriptions/>

Permissions: Permissions & Rights Desk, Lippincott Williams & Wilkins, a division of Wolters
Kluwer Health, 351 West Camden Street, Baltimore, MD 21202-2436. Phone: 410-528-4050. Fax:
410-528-8550. E-mail:
journalpermissions@lww.com

Reprints: Information about reprints can be found online at
<http://www.lww.com/reprints>

Left Main Coronary Artery Acute Thrombosis Related to Energy Drink Intake

Alexandre M. Benjo, MD, PhD*; Andrés M. Pineda, MD*; Francisco O. Nascimento, MD; Carlos Zamora, MD; Gervasio A. Lamas, MD; Esteban Escolar, MD

A 24-year-old previously healthy black man presented the emergency department with a 10-hour history of nausea, multiple episodes of emesis, palpitations, and severe retro-sternal chest pain, described as constant pressure. His symptoms started 1 or 2 hours after he had 3 drinks of vodka mixed with an energy drink at a local party. He used marijuana in the week before but denied cocaine or other recreational drug use. Two of his friends who shared the drinks had similar symptoms but without chest pain. There was no family history of premature coronary artery disease. The patient smokes no more than 5 cigarettes weekly.

At admission, his vital signs were blood pressure 138/94 mm Hg, pulse 63 bpm, breathing rate 18 respirations per minute, temperature 36°C, and 99% oxygen saturation on room air. Physical examination was unremarkable. The initial ECG showed normal sinus rhythm with a subtle J-point elevation in leads II, III, aVF, and V₂ through V₆ with a concave shape. However, a repeat ECG rapidly evolved to marked ST elevation and a convex shape in the lateral leads, with a decrease in R-wave progression (Figure 1). The initial troponin I test was negative. A bedside echocardiogram demonstrated apical hypokinesis and normal ascending aorta.

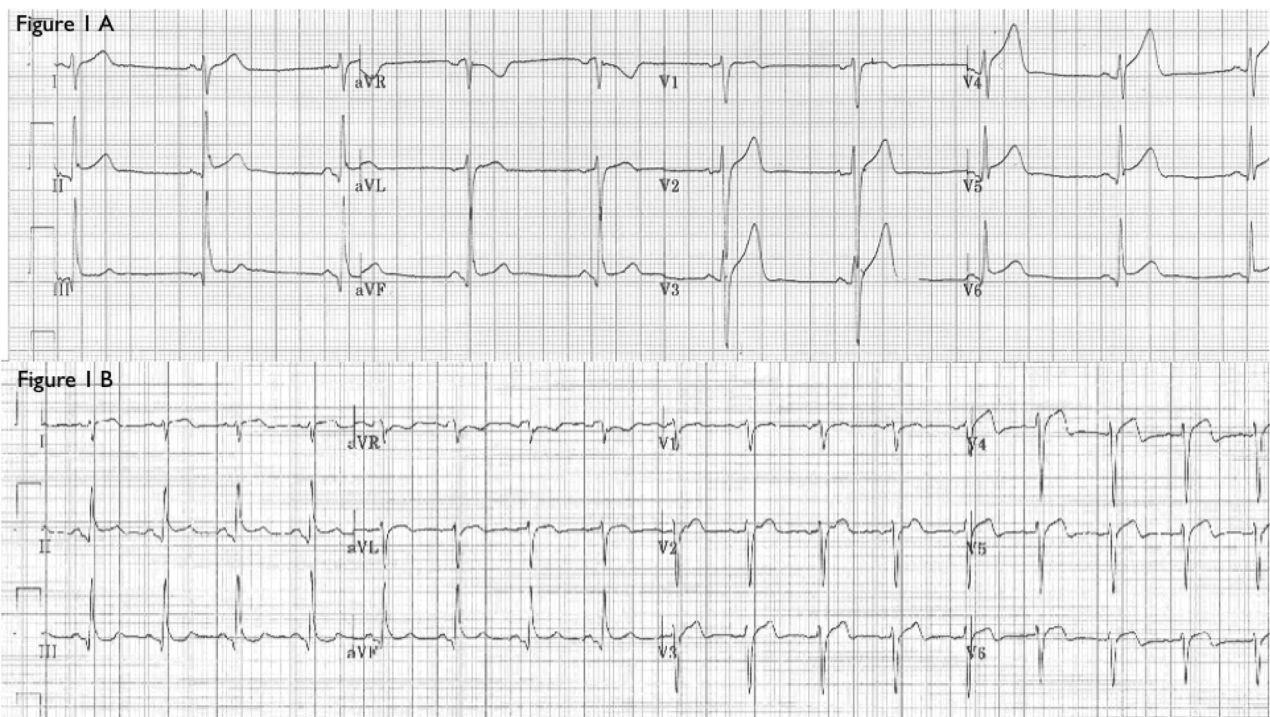


Figure 1. A, Initial 12-lead ECG demonstrating hyperacute T-wave changes and concave J-point elevation up to 4 mm at the precordial leads and up to 2 mm at the inferior limb leads. **B,** ECG shows progression to decreased R-wave amplitude in the anterolateral wall associated with a now convex sustained ST elevation and a terminal T-wave inversion.

From the Mount Sinai Medical Center, Miami Beach, FL (A.M.B., A.M.P., F.O.N., C.Z.) and Columbia University Division of Cardiology at Mount Sinai Medical Center, Miami Beach, FL (G.A.L., E.E.).

*Drs Benjo and Pineda contributed equally to this article.

The online-only Data Supplement is available with this article at <http://circ.ahajournals.org/lookup/suppl/doi:10.1161/CIRCULATIONAHA.111.086017/-DC1>.

Correspondence to Esteban Escolar, MD, FACC, Mount Sinai Medical Center, 4300 Alton Rd, Miami Beach, FL 33140. E-mail esteban.escolar@msmc.com

(*Circulation*. 2012;125:1447-1448.)

© 2012 American Heart Association, Inc.

Circulation is available at <http://circ.ahajournals.org>

DOI: 10.1161/CIRCULATIONAHA.111.086017

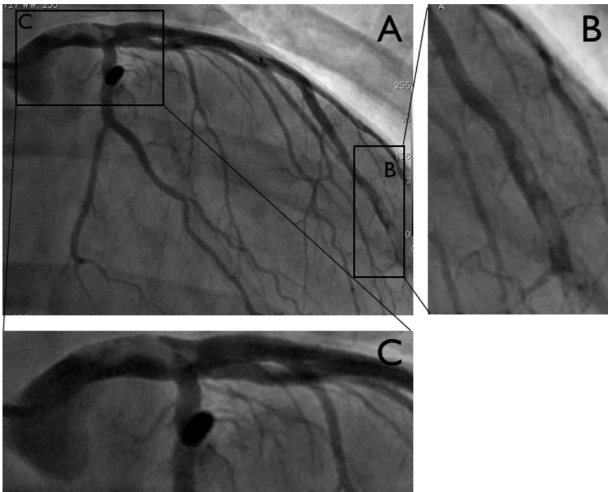


Figure 2. **A**, Right anterior oblique projection of the left coronary system. **B**, At the distal LAD, occlusive embolization is seen. **C**, Thrombi occupying mid through distal left main involving the takeoff of the circumflex and left anterior descending arteries.

An emergent coronary angiogram was performed demonstrating a large thrombus occupying most of the length and approximately 70% of the diameter of the left main coronary artery and involving the origin of the circumflex, with almost 90% occlusion. A second thrombus that occluded the distal left anterior descending coronary artery was seen (Figure 2). No atherosclerotic lesions or coronary malformations were identified (online-only Data Supplement Movies I and II). The patient developed congestive heart failure after coronary angiography.

An intra-aortic balloon pump was placed. Emergent coronary bypass graft surgery was ultimately performed, with the left internal mammary artery grafted to the left anterior descending coronary artery and a saphenous vein graft to the left circumflex artery to ensure antegrade perfusion because of the risk of total left main thrombosis. Troponin I before CABG was 38 ng/mL.

Further laboratory tests, including a lipid profile and coagulation panel, were within normal limits. Anticardiolipin

antibodies, homocysteine level, β_2 -glycoprotein antibodies, plasminogen activator inhibitor activity, and protein C and protein S activity were negative or within normal ranges. The patient was discharged home on warfarin.

Energy drinks have become very popular. In 2007, 51% of college students had consumed at least 1 energy drink in the prior month, and 54% of those had mixed it with alcohol while partying. Previous case reports had linked energy drinks with sudden cardiac death, coronary vasospasm, reversible postural tachycardia syndrome, and serious arrhythmias, including ventricular fibrillation.^{1–3} Most of the cases were related to overuse or concomitant alcohol intake. We could not find prior documented cases of coronary thrombosis associated with energy drink use.

A recently published article demonstrated that 250 mL of energy drink can acutely cause endothelial dysfunction and significantly increase platelet aggregation.⁴ Almost all such commercially available drinks have the same basic stimulants, caffeine, glucuronolactone, taurine, and vitamins, and it is difficult to know which component is responsible for the effect in platelet aggregation and endothelial function. Caffeine has not been shown to affect platelet function by itself, and no studies are available for the other components.⁴ Although its function in platelet aggregation is not clear, taurine has been found in high concentrations in platelets.

Energy drinks effects should be better scrutinized because of their increasing consumption by the public and their potentially lethal effects.

Disclosures

None.

References

- Berger AJ, Alford K. Cardiac arrest in a young man following excess consumption of caffeinated energy drinks. *Med J Aust.* 2009;190:41–43.
- Terlizzi R, Rocchi C, Serra M, Solieri L, Cortelli P. Reversible postural tachycardia syndrome due to inadvertent overuse of Red Bull. *Clin Auton Res.* 2008;18:221–223.
- Higgins JP, Tuttle TD, Higgins CL. Energy beverages: content and safety. *Mayo Clin Proc.* 2010;85:1033–1041.
- Worthley MI, Prabhu A, De Sciscio P, Schultz C, Sanders P, Willoughby SR. Detrimental effects of energy drink consumption on platelet and endothelial function. *Am J Med.* 2010;123:184–187.