A 24-year-old man presented to the emergency room with chest pain, acute respiratory failure, and palpitations shortly after the ingestion of small amounts of an energy drink in little cups one after another. He was afebrile, with frequent runs of supraventricular and ventricular tachycardia and underlying sinus tachycardia. The patient was treated with fluids and metoprolol but required intubation because of progressive hypoxia. Chest x-ray revealed bilateral fluffy pulmonary infiltrates. His ECG was free of ischemic changes, and an echocardiogram at that time showed hypokinesis of all basal left ventricular segments with apical sparing and an ejection fraction of 35% (Figure 1 and Movie 1 in the online-only Data Supplement). His troponin was mildly increased and serially measured brain natriuretic peptide was elevated at 8000 pg/mL.

After treatment with furosemide, nitroglycerine, heparin, and aspirin, the patient responded with clinical improvement of vital signs and chest x-ray. Episodes of agitation and delirium ensued, delaying extubation. Computerized tomography brain scan and electroencephalogram were negative, and toxicological testing demonstrated no cocaine, cannabis, or other drugs of abuse. The patient was extubated on hospital day 3. The psychiatric syndrome gradually resolved, and on hospital day 10, he underwent cardiac magnetic resonance with gadolinium administration. Moderate to severe hypokinesis of the basal segments of the left ventricle with apical sparing and globally increased myocardial wall thickness (reflecting the presence of edema) was observed without any late gadolinium enhancement (Figure 2A and 2B and Movie II in the online-only Data Supplement).

The patient was discharged on hospital day 14 on lisinopril and carvedilol. Two months after admission, a repeat echocardiogram revealed normalization of left ventricular function with an estimated ejection fraction of 55% and the absence of regional wall motion abnormalities (Movie III in the online-only Data Supplement), and a new cardiac magnetic resonance demonstrated complete normalization of ventricular function and wall motion and thickness (the latter

Figure 1. ECG recorded at admission showing sinus tachycardia and nonspecific T-wave inversion in leads I and aVL.
To the best of our knowledge, our case is the first report of reverse SCM triggered by consumption of an energy drink containing sympathomimetic substances, specifically caffeine and 1,3-dimethylamylamine (DMAA).

Caffeine acts as a competitive antagonist of adenosine receptors A1 and A2A in both the central nervous system and the myocardium, altering neurotransmitter release and increasing heart rate, respectively. Caffeine also induces catecholamine release and causes a rise in intracellular calcium in myocytes. Its role in triggering arrhythmias is well established.

DMAA is a non–Food and Drug Administration–approved psychoactive substance acting on the central nervous system that gives its users an adrenaline rush. This substance exhibits an obvious structural similarity to both amphetamine and propylhexedrine, possibly accounting for its stimulant and norepinephrinergic effects. There is no specific blood test to identify concentrations of DMAA in the blood acutely, and the only recommended tests include standard complete blood count and comprehensive metabolic panel, as well as creatine kinase-MB and cardiac troponin I, which were measured in our patient.

In our case, the reversed pattern of SCM was observed instead of the common apical ballooning. Indeed, in a recent retrospective analysis of all published cases of SCM in 2007 to 2008, the authors concluded that patients with the reverse type present at an early age compared with other types of SCM, pointing out that peak adrenoceptor density is at the base of the heart during youth and gradually shifts toward the apex at older age. Furthermore, the mode of stress could have an influence on the observed pattern of SCM, because there are a few reports of reverse SCM cases associated with the exogenous administration of catecholamines, as it is in our case.3

The importance of this report is highlighted by the rapidly increasing consumption of energy drinks among the healthy young. Physicians who practice cardiology, emergency medicine, and psychiatry should be aware of the potential relationship between energy drinks and cardiovascular morbidity.

Disclosures

None.

References

4. Ramaraj R, Movahed MR. Reverse or inverted takotsubo cardiomyopathy (reverse left ventricular apical ballooning syndrome) presents at a younger age compared with the mid or apical variant and is always associated with triggering stress. Congest Heart Fail. 2010;16:284–286.

Discussion

Since the first description of stress cardiomyopathy (SCM), there have been reported cases of variant or atypical forms of the disease with different patterns of contractile dysfunction. The most common of these forms is the reverse type with hyperdynamic apex and akinesis of the base of the left ventricle, which could be explained by regional variations in adrenergic sensitivity or innervation of myocardium among individuals.1

Reverse SCM, which can be encountered even in young male individuals, appears less strongly related to emotional stress, and is thought to be triggered by either hypercatecholaminemia associated with catecholamine-secreting tumors such as pheochromocytoma and paraganglioma or acute cerebral disorders such as subarachnoid hemorrhage and severe head injury. Importantly, intravenous administration of catecholamines and other β-receptor agonists also has been reported in association with reverse SCM.2,3

consistent with resolution of edema) and the absence of any late gadolinium enhancement (Figure 2C and 2D and Movie IV in the online-only Data Supplement). Laboratory evaluation for pheochromocytoma was negative. All medications were stopped because of patient preference and lack of clear ongoing clinical indication.

Figure 2. Cardiac magnetic resonance imaging with gadolinium administration on hospital day 10 (A, diastole; B, systole) showing moderate to severe hypokinesis of the basal segments of the left ventricle with apical sparing along with globally increased myocardial wall thickness (reflecting the presence of edema) without any late gadolinium enhancement. Repeat examination 2 months after admission revealed complete normalization of left ventricular function, wall motion, and wall thickness and the absence of any late gadolinium enhancement (C, diastole; D, systole).
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