Acute Liver Failure Following One Year of Daily Consumption of a Sugar-Free Energy Drink

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Abstract

We report a 36-year-old man who presented with 1 week of right upper quadrant abdominal pain, jaundice, and fatigue. He consumed 3 sugar-free energy drinks daily for the past year with binge alcohol use. His liver function progressively deteriorated, requiring orthotopic liver transplantation. Submassive hepatic necrosis with eosinophilic infiltrate was seen on pathology, consistent with drug-induced liver injury. Further investigation is warranted into identifying which individuals are susceptible to liver failure from energy drink consumption.

Introduction

Since the early 2000s, energy drinks have become an increasingly popular beverage choice in the United States, especially among adolescents and young adults.\textsuperscript{1,2} In recent years, public concerns regarding increasing complications of these energy drinks has led to heightened media scrutiny of the safety profile of these products.\textsuperscript{3}

Case Report

A 36-year-old healthy white male presented with 1 week of right upper quadrant abdominal pain and jaundice. The patient reported that he was in good health until initial symptom onset 2 weeks prior. Since then, he reported fatigue, decreased appetite, and a 10-pound weight loss over the past year. The patient admitted to weekend binge alcohol drinking for the past 15 years, and drank 10 beers 3 hours prior to symptom onset. The patient denied taking herbal remedies, medications, or supplements. He noted that he was drinking 3 Rockstar energy drinks (Rockstar, Inc., Las Vegas, NV) per day for the past year. On physical exam, the patient was alert and coherent. He was jaundiced, had a single spider nevus on his back, and had a benign abdomen without ascites.

He was found to have abnormal liver tests with aspartate transaminase (AST) 1,541 U/L, alanine transaminase (ALT) 2,995 U/L, alkaline phosphatase 231 U/L, total bilirubin 16.1 μmol/L, and international normalized ratio (INR) 1.0. Computed tomography (CT) showed a normal-appearing liver and no biliary dilatation. Liver values subsequently increased to AST 2,160 U/L, ALT 2,566 U/L, and total bilirubin 1.3 μmol/L (Table 1). Laboratory investigation was negative for HIV, herpes simplex virus, varicella zoster virus, \textit{Cytomegalovirus}, Epstein-Barr virus, hepatitis A, B, C, and E serologies, anti-LKM antibody, anti-soluble liver antigen, antinuclear antibody, anti-mitochondrial antibody, anti-smooth muscle antibody, acetaminophen/salicylate level, alpha-1 antitrypsin, and ceruloplasmin. Liver biopsy showed severe active hepatitis, bridging necrosis, and lymphocytic infiltrate with eosinophils, consistent with an herbal/drug-toxicity pattern.
One week later, labs showed deteriorating liver function with INR 3.2, total bilirubin 23.1 μmol/L, and creatinine 1.3 mg/dL. The patient began to show signs of hepatic encephalopathy, which was treated with lactulose and rifaximin. He had episodes of hypoglycemia requiring intravenous dextrose infusion. He was given a short course of steroids empirically for seronegative autoimmune hepatitis, but this was discontinued given lack of any therapeutic response. He underwent successful orthotopic liver transplant. Histologic evaluation of the explanted liver showed massive hepatocellular necrosis and parenchymal collapse in the context of extensive lymphocytic infiltrate with eosinophils, neutrophils, and plasma cells. No fibrosis was seen on trichrome stain.

**Discussion**

Energy drinks contain many additives including caffeine, taurine, B-vitamins, and other ingredients (Box 1). Niacin (vitamin B3) has been shown to cause hepatotoxicity ranging from mild elevations in the aminotransferases to fulminant hepatic failure. Available data suggest a greater than 50% chance of hepatotoxicity when doses of niacin exceed 2,000 mg/day. Vivekanandarajah et al described a young woman who drank 10 cans of an energy drink over 2 weeks, resulting in acute hepatitis. She had consumed 300 mg per day of niacin, and they concluded this was most likely the cause of her acute hepatitis. Our patient drank three 8-oz cans of the sugar-free Rockstar energy drink every day for 1 year, equivalent to 120 mg of niacin per day. This is lower than previously reported dosages of niacin causing hepatotoxicity, suggesting that perhaps a cumulative effect may have been involved in the development of liver failure, or that the patient’s binge alcohol use made his liver more vulnerable to further injury. Previous studies have suggested that chronic alcohol ingestion induces CYP2E1, while niacin inhibits its activity. The Maria and Victorino scoring system is used to determine causality in drug-induced liver injury and was applied to our patient, who presented with a score of 10 points, implying a “possible” causality for our patient’s drug-induced liver injury. Further investigation is warranted into identifying the mechanism and effects that these ingredients have on cytochrome activity, liver metabolism, and drug-induced liver injury.

**Disclosures**

Author contributions: B. Huang performed the chart review, wrote the first draft of the manuscript, and is the article guarantor. D. Kunkel and M. El Kabany conceptualized and supervised this case report and made edits on subsequent drafts.

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**Table 1. Laboratory Values**

| Value                        | Reference Range   | 8 Days Prior to First Admission | First Admission | Second Admission | Discharge 6 Days After Procedure |
|------------------------------|-------------------|--------------------------------|-----------------|-----------------|---------------------------------|
| White blood cells, 1000/µL   | 4.16–9.95         | 4.3                            | 3.6             | 4               | 7.5                             |
| Hemoglobin, g/dL             | 13.5–17.1         | 15.6                           | 14.5            | 15.3            | 11.8                            |
| Platelets, 1000/µL           | 143–398           | 185                            | 159             | 207             | 113                             |
| Creatinine, mg/dL            | 0.5–1.3           | –                              | 1               | 1.3             | 0.6                             |
| AST, U/L                     | 7–36              | 1541                           | 2253            | 1250            | 26                              |
| ALT, U/L                     | 4–45              | 2995                           | 2432            | 1004            | 114                             |
| Alkaline phosphatase, U/L    | 31–103            | 231                            | 183             | 116             | 66                              |
| Total bilirubin, mg/dL       | 0.2–1.1           | 16.1                           | 15.6            | 23.1            | 3.4                             |
| INR                          | N/A               | 1                              | 1.6             | 3.7             | 1.1                             |

*ALT = alanine transaminase; AST = aspartate transaminase; INR = international normalized ratio.

aAt an outside hospital.

In the third week following transplantation, the patient was discharged with INR 3.2, total bilirubin 23.1 μmol/L, and creatinine 1.3 mg/dL. One week later, labs showed deteriorating liver function with INR 3.2, total bilirubin 23.1 μmol/L, and creatinine 1.3 mg/dL. The patient began to show signs of hepatic encephalopathy, which was treated with lactulose and rifaximin. He had episodes of hypoglycemia requiring intravenous dextrose infusion. He was given a short course of steroids empirically for seronegative autoimmune hepatitis, but this was discontinued given lack of any therapeutic response. He underwent successful orthotopic liver transplant. Histologic evaluation of the explanted liver showed massive hepatocellular necrosis and parenchymal collapse in the context of extensive lymphocytic infiltrate with eosinophils, neutrophils, and plasma cells. No fibrosis was seen on trichrome stain.

**Discussions**

Energy drinks contain many additives including caffeine, taurine, B-vitamins, and other ingredients (Box 1). Niacin (vitamin B3) has been shown to cause hepatotoxicity ranging from mild elevations in the aminotransferases to fulminant hepatic failure. Available data suggest a greater than 50% chance of hepatotoxicity when doses of niacin exceed 2,000 mg/day. Vivekanandarajah et al described a young woman who drank 10 cans of an energy drink over 2 weeks, resulting in acute hepatitis. She had consumed 300 mg per day of niacin, and they concluded this was most likely the cause of her acute hepatitis. Our patient drank three 8-oz cans of the sugar-free Rockstar energy drink every day for 1 year, equivalent to 120 mg of niacin per day. This is lower than previously reported dosages of niacin causing hepatotoxicity, suggesting that perhaps a cumulative effect may have been involved in the development of liver failure, or that the patient’s binge alcohol use made his liver more vulnerable to further injury. Previous studies have suggested that chronic alcohol ingestion induces CYP2E1, while niacin inhibits its activity. The Maria and Victorino scoring system is used to determine causality in drug-induced liver injury and was applied to our patient, who presented with a score of 10 points, implying a “possible” causality for our patient’s drug-induced liver injury. Further investigation is warranted into identifying the mechanism and effects that these ingredients have on cytochrome activity, liver metabolism, and drug-induced liver injury.

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