Purpose: There have been many reports of toxicity associated with use of dietary supplements; some of these cases have lead to fatal outcomes. We report a rare case of fulminant liver failure assumed to be from ingestion of SlimQuick™ (Wellnx Life Sciences, Wilmington, DE) weight loss supplement containing green tea extract (GTE).

Case Report: A 52-year-old woman presented to the Emergency Department with one week of vomiting and progressive jaundice. On further questioning, the patient reported two days of ingesting the weight loss supplement SlimQuick™ while fasting 3 weeks before presentation. Past medical, surgical and family histories were unremarkable. Physical examination showed normal mental status, icteric sclera, mild abdominal distension and lower extremity edema. Initial workup showed total bilirubin of 16.5 mg/dL, AST 1507 IU/L, ALT 945 IU/L, alkaline phosphatase 210 IU/L and INR 2.82. CT abdomen revealed nodular liver with small amount of ascites. Serological tests for viral hepatitis, autoimmune hepatitis, Wilson disease, and primary biliary cirrhosis were negative. Liver biopsy was consistent with confluent hepatic necrosis with collapse. In the unlikely possibility that there was an autoimmune etiology to her acute liver injury, Prednisone 60mg was initiated but discontinued two days later due to worsening liver function. A day later, the patient’s mental status began to deteriorate. The patient was expeditiously evaluated and listed for liver transplant. She underwent liver transplantation two days later. The patient was discharged home post-operative day eight.

Discussion: To our knowledge, this is the first reported case of fulminant liver due to ingestion of SlimQuick™. The major ingredient in SlimQuick™ is GTE. GTE is a common ingredient in several dietary supplements, some of which have been withdrawn from the market due to safety concerns. An example of this is Exolise® (Arkopharma, France), a weight loss supplement containing GTE that was withdrawn from the market due to 13 cases of liver injury. Since 1966, 34 case reports of liver toxicity with GTE were identified by the United States Pharmacopeia. The majority of cases present with an acute hepatocellular injury pattern and most recover with cessation of use. An idiosyncratic or an immune-allergic mechanism appears to be the likely mechanism of injury. Animal studies with high doses of GTE have described dose dependent hepatotoxicity resulting in severe morbidity and mortality. This demonstrates the importance obtaining herbal and dietary supplement history in previously healthy subjects who develop liver injury.
Secondary Analyses: Not Applicable
Study Results: Yes
Submit:
Supported by Industry Grant: No
Purpose: Herbal supplements are commonly used by patients for various problems. It is a well-known fact that most patients do not tell their physicians regarding use of herbal supplements unless specifically asked. As a result, sometimes important points from drug side effects are missed in history taking. In this context, we present a rare case of Black Cohosh-induced hepatotoxicity leading to early cirrhosis.

Case Description: A 44-year-old female with no PMH presented with complaints of painless jaundice for one month. She went to her PCP, where initial work-up revealed that she had elevated LFTs. She was noted to have normal LFTs on her prior lab works. Work-up for viral and autoimmune hepatitis was negative. She was given a trial of steroids on outpatient basis without much improvement. She was referred to inpatient evaluation because of gradual progression of her symptoms. She denied history of alcohol intake, IV drug use, unprotected sex, recent travel outside the U.S., NSAID ingestion or blood transfusions. She reported no abdominal pain, fever, chills, nausea vomiting or diarrhea. She did report generalized itching, arthralgia and fatigue. She interestingly reported that she started taking Black Cohosh for alleviation of her menstrual symptoms about one month back. Her exam was remarkable for marked scleral icterus and jaundiced skin. On admission, her LFT showed Tbil =20, AST=420, ALT=215, AlkPhos=201, Platelets=135, INR 1.2 and Albumin=2.4. Ultrasound abdomen showed nodular contour of liver consistent with cirrhosis. Further work-up ruled out Wilson’s disease, Hemochromatosis, AMA negative PBC and autoimmune hepatitis. Liver biopsy was performed, which showed histologic pattern consistent with cholestasis, hepatocellular injury and early cirrhosis. Given patient’s history of Black Cohosh use and the timing of her abnormal liver chemistries, it was clinically evident the culprit agent was Black Cohosh. Her symptoms improved, and her LFT’s normalized after she stopped taking Black Cohosh.

Discussion: Black Cohosh, also named as Cimicifuga racemosa, is among commonly used herbal supplements in the United States for menstrual symptoms. There are few case reports available in literature that attribute Black Cohosh to liver injury. In most instances, the liver injury ranges from jaundice, mild transaminasemia to rare cases of fulminant hepatic failure. Our case is unique, since it represents development of accelerated cirrhosis in our patient for a relatively short period of time. This case is to bring awareness amongst clinicians about this potentially unexpected outcome in the backdrop of much-expected drug induced hepatitis.
Study Results: Yes
Submit:
Supported by Industry Grant: No
Purpose: A 36-year-old Caucasian male without prior medical history initially presented to another facility with one week of right upper quadrant (RUQ) abdominal pain, jaundice and fatigue. After abnormal lab values (Table 1) were found, he left that facility and presented to this hospital. He admitted to weekend binge drinking and drank 10 beers three hours prior to symptom onset. He denied taking herbal remedies, homeopathic medications or other supplements. He drank up to three energy drinks (Figure 1) on a daily basis for the past year.

Physical exam showed stable vital signs, jaundice, spider nevi and RUQ tenderness. Initial work-up was negative for viral, ischemic or autoimmune hepatitis. On HD #6, a liver biopsy showed severe active hepatitis with bridging necrosis consistent with an herbal/drug-toxicity pattern. On HD #9, the patient was discharged home, but was soon re-admitted for worsening LFTs. With an INR of 4.2, he was placed on the UNOS waiting list with a MELD score of 41. On HD #14 of this 2nd hospitalization, a suitable donor became available and the patient underwent successful liver transplantation. Gross liver pathology showed massive hepatocellular necrosis and parenchymal collapse consistent with drug-induced liver injury. He was discharged on post-operative day number seven.

Discussion: A similar case report by Vivekanandarajah et al. described a young woman who drank 10 cans of an energy drink for two weeks, resulting in acute hepatitis. The authors concluded that Vitamin B3 (niacin) was the culprit ingredient.

Our case report details a patient who drank a sugar-free energy drink over a longer period and had a more serious clinical course. The patient’s prior alcohol use may have provided a “first hit” on the liver, making it more susceptible to further insults. The energy drink in this case report contains many ingredients, some of which do not have a well-established safety profile. While drinking modest amounts of energy drinks appears to be relatively safe, frequent consumption over an extended period of time has recently been linked with hepatic injury.

Methods: N/A

Results: N/A

Conclusion: N/A

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<th>Table 1: Laboratory Findings</th>
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**TABLE TITLE:** Table 1: Laboratory Findings

**Ingredients of the Energy Drink (serving size & references):**
- Taurine
- Caffeine
- Vitamin B2 (Riboflavin)
- Vitamin B3 (Niacin)
- Vitamin B5 (Pantothenic Acid)
- Vitamin B6 (Pyridoxine)
- Vitamin B12 (Cobalamin)
- Guarana Seed Extract
- Panax Ginseng Root Extract
- L-Carnitine
- Inositol
- Milk Thistle Extract

**Figure 1:** Ingredients of Sugar Free Energy Drink

**IMAGE CAPTION:** Figure 1: Ingredients of Sugar Free Energy Drink

**Video Submission Confirmation:** No

**Video Upload:**
- **Abstract Author:** Investigator
- **Commercial Products or Services:** No
- **Designed Study:** Investigator
- **FDA Approval:** No
- **Financial Relationships:** No
- **Initiated Research:** Investigator
- **Investigator Contribution:** No
- **Performed Analysis:** Investigator
- **Secondary Analyses:** Not Applicable
- **Study Results:** Yes
- **Submit:**
- **Supported by Industry Grant:** No
Purpose: Ripped Fuel is an advanced weight loss and fat-burning formula, containing herbal extract with 60% flavonoids, caffeine, and cacao. Although it has been known to have a good safety profile, we present a case that illustrates drug-induced liver injury secondary to its use. A 36-year-old female with history of depression and no prior liver disease presented with 1-week history of abdominal pain, anorexia, and nausea. She began to take Ripped Fuel 3 weeks prior to developing these symptoms to loose weight. She denied use of other herbal medicine, supplements, or acetaminophen. For her depression, she had been taking venlafaxine for years, with no recent changes in medications. On physical examination, she had scleral icterus and mild jaundice. Abdominal examination revealed mild RUQ tenderness with no hepatosplenomegaly, and no stigmata of chronic liver disease. Initial laboratory findings suggested fulminant hepatic failure, with AST 2152 U/L, ALT 2711 U/L, ALP 290 U/L, total bilirubin 3.4 mg/dL, direct bilirubin 2.4 mg/dL, INR 1.2, BUN 10 mg/dL, and creatinine 0.7 mg/dL. Acute hepatitis panel was consistent with previous immunization to hepatitis A and hepatitis B, and was negative for hepatitis C. Autoimmune work-up with anti-smooth muscle antibody, anti-mitochondrial antibody, and immunoglobulin G, A, and M levels were all within normal limits. ANA was 1:80. As the liver enzymes continued to escalate with AST of 2,511 U/L, and ALT of 2,925 U/L, liver biopsy was performed on hospital day three. Findings of the liver biopsy was consistent with marked portal inflammation, with circumferential interface activity and bridging hepatocyte necrosis consistent with drug-induced liver injury (DILI). She was started on methylprednisone 60 mg/day and ursodeoxycholic acid (UDCA) 1,000 mg/day, and her AST, ALT levels began to improve instantly after beginning treatment. There was a lag of improvement in the level of bilirubin, which began to trend down 3 days after steroid treatment. She continued to improve clinically, with no evidence of hepatic failure during hospitalization, and was safely discharged.

Discussion: Flavonoids have been described to cause significant liver injury in several case reports. Treatment after development of DILI has been poorly defined, besides discontinuing the triggering substance. Previously, steroids have been studied to prevent tissue damage from inflammatory response, which failed to show beneficial effects. Usage of UDCA has been shown to be favorable, due to protection of hepatocytes against cytotoxic effects of bile acids, and stimulating hepatobiliary secretion. Recently, combination of steroids and UDCA proved to benefit the outcome of patients with severe DILI.

Methods: N/A
Results: N/A
Conclusion: N/A

Video Submission Confirmation: No
Video Upload:
Abstract Author: Investigator
Commercial Products or Services: Yes
Designed Study: Investigator
FDA Approval: No
Financial Relationships: No
Initiated Research: Investigator
Investigator Contribution: No
Performed Analysis: Investigator
Secondary Analyses: Not Applicable
Study Results: Yes
Submit:
Supported by Industry Grant: No