Severe rhabdomyolysis in a sickle cell trait patient associated with consumption of an energy drink

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Abstract

Rhabdomyolysis is a syndrome characterized by muscle injury involving the release of potentially toxic intracellular contents into plasma, which in turn may cause extreme enzyme elevations, electrolyte imbalances and acute kidney injury. The typical causes for rhabdomyolysis include but not limited to muscle trauma or crush injury, prolonged immobilization, inflammatory myopathies, medications, drug use and severe physical exertion. We present a 24-year-old African American male with a past medical history significant for thalassemia minor and sickle cell trait admitted for severe rhabdomyolysis with a creatinine kinase (CK)> 650,000 precipitated by the consumption of energy drinks. Energy drinks are known to contain higher levels of caffeine whose diuretic effect can lead to hypokalemia, elevation of CK and renal impairment. Patients with sickle-cell trait are known to be more predisposed to dehydration secondary to a renal concentrating defect. This in turn can exacerbate a state of dehydration caused by caffeine, infections or excessive perspiration. Our case is the only reported presentation of severe non-traumatic rhabdomyolysis in a patient with sickle cell trait precipitated by the use of caffeine containing energy drink. Therefore, it may be prudent to avoid the consumption of energy drinks in patients with sickle cell trait.

Keywords

Rhabdomyolysis; energy drinks; sickle cell trait; creatine kinase; caffeine

Abbreviations

CK: Creatine kinase; ED: Emergency Department; PCP: Primary care physician; IV: Intravenous; HIV: Human immunodeficiency virus; EBV: Ebstein-Barr Virus; CMV: Cytomegalovirus; CO: carbon-monooxide; TSH: Thyroid stimulating hormone; LFTs: Liver Function tests; MRI: Magnetic resonance imaging; BUN: Blood urea nitrogen

Introduction

Rhabdomyolysis is a condition which involves skeletal muscle breakdown, resulting in the release of intramuscular contents into the general circulation. Creatine kinase (CK) levels are typically markedly
elevated to more than five times the upper limit of normal. There are multiple probable causes of rhabdomyolysis [1-4]. These are broadly divided into traumatic or muscle compression and non-traumatic categories (Table 1). Patients can initially be asymptomatic but with severe elevation of CK, life-threatening electrolyte imbalances, and acute kidney injury [1] can develop. We present a case of a young African American male with a history of Thalassemia minor and sickle cell trait who consumed energy drinks and subsequently developed severe rhabdomyolysis.

Case Presentation

A 24-year-old African American male with a history of thalassemia minor and sickle cell trait who presented to our facility from an outside hospital for the management of severe rhabdomyolysis. He reported one week history of diffuse myalgias, back pain as well as the development of dark colored urine. He reported travelling approximately 400 miles by car a week ago and consuming energy drinks. Patient did not remember the exact type and quantity of energy drink consumed but reported taking significant amounts. The reason for the consumption of energy drinks was not mentioned by the patient. He denied any recent increase in physical activity, drug abuse, alcohol intake, fever, jaundice, pruritus, rash, nausea, vomiting, urinary symptoms and any proximal muscle weakness or trauma. On obtaining further history he denied any Raynaud’s symptoms, dysphagia, photosensitivity, sicca symptoms or weight loss. There was no history of similar episodes in the past or a family history of metabolic myopathies.

In the outside hospital ED, his CK was found to be 330,437; subsequent measurement at presentation to our hospital showed CK levels of 622,674. BUN/Creatinine was normal and LFTs were moderately elevated likely secondary to Rhabdomyolysis. Urine drug screen was notable for opiates which were administered by ED. Dipstick was positive for large blood consistent with myoglobinuria. On presentation, his hemoglobin was 13.1 and Reticulocyte count was within normal limits. On arrival at our facility he reported no acute complaints. His physical examination was unremarkable except for the presence of discomfort on palpation of bilateral lower extremity muscles. Patient had sickle cell trait which was discovered about a year and half ago but he had never been hospitalized in the interim. The patient was given IV fluids and labs were redrawn. On the first day of his hospitalization, repeat laboratory evaluation showed improvement in CK levels to 452,794 which subsequently down trended to 330,000.

On the second day of his hospitalization his CK levels started rising and were noted to be 547,696. His intravenous fluids were switched from normal saline to 5% dextrose with sodium bicarbonate. Rheumatology was consulted and advised to check hepatitis C, HIV and TSH, which were unremarkable. Myositis panel was not ordered as per Rheumatology recommendations. Given the persistent elevation of his CK, a bilateral lower extremity MRI was ordered showing diffuse nonspecific muscular edema in both lower extremities consistent with rhabdomyolysis (Figure 1). The patient’s renal function remained stable and creatinine kinase slowly trended down. The patient’s CK levels at discharge was 9800. He was advised to continue good oral intake with fluids following discharge. The patient was able to ambulate and perform activities of daily living. He was counselled about the harmful effects of energy drinks and to avoid consuming them. He was requested to follow up with his primary doctor and hematologist.
Discussion

We present a unique case of severe rhabdomyolysis in a young African American male with sickle cell trait precipitated by the use of caffeine containing energy drink. We ruled out common differentials as the etiology of this presentation. Normal Reticulocyte count on presentation and stable hemoglobin suggested against a Sickle cell crisis picture. There was no history of trauma, endocrinopathy, electrolyte imbalance or recent drug usage.

To our knowledge, this is the first reported case in medical literature of rhabdomyolysis in a sickle cell trait patient likely secondary to energy drink consumption. All previous cases of Rhabdomyolysis secondary to caffeine were reported in the general population.

Energy drinks are fortified beverages with different dietary supplements. The first energy drink was launched in Austria in 1987 while it was commenced in the United States (US) in 1997 [5]. The consumption of energy drinks has increased significantly ever since. The main consumers are young adults aged 18-34 years [6]. These drinks predominantly contain caffeine blended with other natural ingredients that increase attention, improve performance and concentration time; however, health professionals are concerned about the detrimental effects associated with these products [7-10]. Energy drink-related health consequences reported in studies include liver damage, kidney and respiratory problems, seizures, and agitation, cardiac arrhythmias, heart failure, high blood pressure and infrequently rhabdomyolysis [11-13]. Caffeine is rarely cited as a potential etiology but few case reports describe rhabdomyolysis secondary to caffeine overdose [14,15]. Caffeine's diuretic effect can lead to fluid-electrolyte imbalance and renal impairment [16]. Individuals who do not typically consume large amounts of caffeine may be more prone to increased diuresis leading to dehydration [17].

In our patient, on ruling out the common and uncommon causes of rhabdomyolysis, we concluded that his severe rhabdomyolysis was probably secondary to the consumption of energy drinks in the setting of sickle-cell trait. Sherry theorized that persons with sickle-cell trait might be innately more predisposed to dehydration due to their inability to concentrate their urine when deprived of water [18]. This renal concentrating defect in persons with sickle-cell trait could be easily exacerbated by a state of dehydration caused by caffeine (energy drinks), excessive perspiration or an infection. The primary risk factors for rhabdomyolysis in persons with sickle-cell trait include: extreme heat and dehydration, high altitude, exercise-induced asthma and pre-event fatigue (in athletes) due to illness or lack of sleep [19]. These risk factors can induce sickling leading to a “logjam” of blood vessel causing ischemic rhabdomyolysis with rapid breakdown of muscles. Furthermore, major metabolic problems from severe rhabdomyolysis can be life threatening.

Upon careful review of literature, the consumption of energy drinks appears to be a rising health problem in the US which can no longer be ignored. As physicians, it is very important to inform patients about the potential harmful effects of energy drinks and raise awareness regarding the FDA dietary supplement reporting system. Of particular note, is the concern of dehydration induced by energy drinks leading to rhabdomyolysis in patients with known sickle-cell trait. As clinicians, we need to be vigilant and educate patients with sickle-cell trait about the potential dangers related to energy drinks.
Conclusion

Excessive caffeine intake from energy drinks can cause rhabdomyolysis especially in patients with sickle cell trait. Such patients should be counseled about the harmful effects of energy drinks or caffeinated beverages which may lead to life threatening rhabdomyolysis.

Table

Table 1: Etiologies of Rhabdomyolysis:

<table>
<thead>
<tr>
<th>Non-Traumatic</th>
<th>Traumatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Extreme exertional physical activity</td>
<td>- Trauma and Crush Injuries</td>
</tr>
<tr>
<td>- Status asthmaticus</td>
<td>- Prolonged Immobilization or Coma</td>
</tr>
<tr>
<td>- Seizures</td>
<td>- Vascular or Orthopedic surgery</td>
</tr>
<tr>
<td>- Thermal extremes and dysregulation</td>
<td>- Extensive Third-Degree burns</td>
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<tr>
<td>- Drugs: cocaine, amphetamines, methadone, colchicine, statins.</td>
<td>- High Voltage Electric injury</td>
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<td>- Toxins: snake venoms, CO poisoning, Mushroom poisoning</td>
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<td>- Abrupt withdrawal of Gamma- amino butyric acid agonist</td>
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<tr>
<td>- Endocrinopathies: Thyroid diseases and Diabetes mellitus</td>
<td></td>
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<td>- Non-depolarizing muscle blocking agents in critically ill patients</td>
<td></td>
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<tr>
<td>- Electrolyte disorders: Hypokalemia, Hypophosphatemia</td>
<td></td>
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<tr>
<td>- Infections: HIV, EBV, CMV, Bacterial myositis, falciparum malaria</td>
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<tr>
<td>- Myopathies</td>
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<tr>
<td>- Sickle Cell Trait</td>
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</table>

Figure

Figure 1: T2 MRI-Transverse View of Right leg muscles demonstrating rhabdomyolytic changes with solid arrows.

References


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