Central pontine myelinolysis (CPM) is a rare neurologic entity of varied etiology characterized by the destruction of the myelin sheath in pontine structures. Initially described in chronic alcohol abuse and malnourished patients, CPM has also been associated with the rapid correction of hyponatremia and alterations in blood glucose homeostasis.

Here we present a case of a man experiencing CPM suspected to be related to the overconsumption of commonly used commercial energy drinks.

Case report. A 36-year-old right-handed white man with no prior medical history presented with dizziness, upper extremity paresthesia, vertigo, impaired balance, diplopia, dysarthria, and pseudobulbar affect. He denied exposure to vaccinations, recent illnesses, or travel outside of the United States in the weeks to months prior to his symptom onset. However, he reported a change in his social history within the week of his presenting symptoms, consuming 6 16-oz Monster Energy drinks and 4 12-oz Red Bull drinks, averaging approximately 484 g of sugar (sucrose, glucose), 2,000 mg of caffeine, and 18,000 mg of taurine daily, in an effort to improve his occupational performance. He denied a history of excessive alcohol consumption or recreational drug use.

Complete blood count, comprehensive metabolic panel, antinuclear antibody, extractable nuclear antigen panel, vitamin B12 level, homocysteine, methylmalonic acid level, hepatitis screen, thyroid-stimulating hormone, free T3, hypercoagulable studies, and CSF studies were unremarkable.

Structural neuroimaging of the brain was significant for a large nonenhancing pontine lesion involving the corticospinal tract, pontocerebellar fibers, nucleus reticularis tegmenti pontis, and medial lemniscus, demonstrating symmetry (figure). Susceptibility-weighted imaging sequences demonstrated increased susceptibility within the observed anomaly.

The patient was treated with 1 g IV methylprednisolone daily for 3 days. Over the next few months, both his neurologic symptoms and longitudinal radiologic findings (figure) improved significantly, and he described experiencing only brief episodes of double vision at the end of the workday or following caffeine exposure. Following his seminal neurologic event, he discontinued his energy drink use.

Discussion. CPM is a noninflammatory demyelinating disorder localized primarily to the pons, with features present in extrapontine structures (e.g., thalamus, striatum, cerebellar cortices, lateral geniculate, etc.) on rare occasions.

The potential connection between the ingredients within the consumed energy drinks and the myelin injury is intriguing. Pontine structures may be particularly vulnerable to blood-brain barrier breakdown due to the topographical mixture of gray and white matter.2 As gray matter contains a 10-fold increase in capillary density compared to white matter, this specific region may be more susceptible to injury related to both arterial hypertension and osmolar shifts.2

There are numerous studies linking the ingredients of energy drinks to acute hypertension.3 Therefore, it is postulated that overconsumption led to alterations in the tight junctions from increased pressures or rapid shifts in serum osmolarity, possibly related to increased amounts of taurine, an important osmolyte in maintaining the osmotic gradient within the brain,4 or other osmolytes (e.g., sugar), resulting in a change in the integrity of vascular endothelial cells.5 Taurine has been shown to have a positive inotropic effect,6 strengthening the hypothesis of a hypertensive etiology. As taurine has been reported to act as a neutralizing agent for adverse effects associated with caffeine, another mechanism beyond hypertension is plausible.7 Effect modification from another ingredient contained within these drinks or a synergistic mechanism from the unique combination of contained ingredients is also possible. As with all seemingly safe agents, excessive use either acutely or chronically may result in unanticipated, paradoxical outcomes. A metabolic abnormality was suspected; however, it is likely that homeostasis was achieved prior to acquisition of the brain imaging data.

The accompanying radiologic data, demonstrating increased susceptibility within the pons, supported the presence of paramagnetic substances...
within the lesion, specifically macrophages containing iron. A reduction in the degree of susceptibility over time was correlated with improvements in the T2-hyperintense lesion and the patient’s clinical symptomatology.

In a time when the modern culture of energy drink consumption exists, caution should be exercised with excessive use, as the acute and chronic health consequences are unclear. Despite ingredients within these beverages being “generally regarded as safe,” we are unaware of the potential adverse events when used disproportionately or in combination with other products. A clear association between the temporal profile of the abrupt change in our patient’s social history and the observed striking radiologic features, along with the lack of classical biomarkers present, highlight both the importance of a comprehensive medical history in CPM and the complexities of its underlying biology.

From the Department of Neurology & Neurotherapeutics, Clinical Center for Multiple Sclerosis, Multiple Sclerosis & Neuroimmunology Imaging Program, UT Southwestern Medical Center, Dallas, TX.

Author contributions: B.D. Newton: drafting/revising the manuscript for content, including medical writing for content, study concept or design, analysis or interpretation of data. D.T. Okuda: drafting/revising the manuscript for content, including medical writing for content, study concept or design, analysis or interpretation of data.

Study funding: No targeted funding reported.

Disclosure: B.D. Newton reports no disclosures. D.T. Okuda has served on the scientific advisory boards for Genzyme and Teva Neuroscience; has received travel funding and/or speaker honoraria from Acorda Therapeutics, Genzyme, MS Association of America, MS Care Fund, and Teva Neuroscience; has consulted for Genzyme and Teva Neuroscience; and is on the speakers’ bureaus for Acorda Therapeutics, Genzyme, and Teva Neuroscience. Go to Neurology.org/NN for full disclosure forms. The Article Processing Charge was paid by the authors.

This is an open access article distributed under the terms of the Creative Commons Attribution-Noncommercial No Derivative 3.0 License, which permits downloading and sharing the work provided it is properly cited. The work cannot be changed in any way or used commercially.

Received November 24, 2014. Accepted in final form January 27, 2015.

Correspondence to Dr. Okuda: darin.okuda@utsouthwestern.edu

4. Thurston JH, Hauhart RE, Nelson JS. Adaptive decreases in amino acids (taurine in particular), creatine,


Pontine myelinolysis following excessive consumption of commercial energy drinks
Braeden D. Newton and Darin T. Okuda

*Neurol Neuroimmunol Neuroinflamm* 2015;2;
DOI 10.1212/NXI.0000000000000091

This information is current as of March 12, 2015

<table>
<thead>
<tr>
<th>Updated Information &amp; Services</th>
<th>including high resolution figures, can be found at: <a href="http://nn.neurology.org/content/2/3/e91.full.html">http://nn.neurology.org/content/2/3/e91.full.html</a></th>
</tr>
</thead>
<tbody>
<tr>
<td>References</td>
<td>This article cites 7 articles, 0 of which you can access for free at: <a href="http://nn.neurology.org/content/2/3/e91.full.html#ref-list-1">http://nn.neurology.org/content/2/3/e91.full.html#ref-list-1</a></td>
</tr>
<tr>
<td>Subspecialty Collections</td>
<td>This article, along with others on similar topics, appears in the following collection(s): All Demyelinating disease (CNS) <a href="http://nn.neurology.org/cgi/collection/all_demyelinating_disease_cns">http://nn.neurology.org/cgi/collection/all_demyelinating_disease_cns</a> MRI <a href="http://nn.neurology.org/cgi/collection/mri">http://nn.neurology.org/cgi/collection/mri</a></td>
</tr>
<tr>
<td>Permissions &amp; Licensing</td>
<td>Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: <a href="http://nn.neurology.org/misc/about.xhtml#permissions">http://nn.neurology.org/misc/about.xhtml#permissions</a></td>
</tr>
<tr>
<td>Reprints</td>
<td>Information about ordering reprints can be found online: <a href="http://nn.neurology.org/misc/addir.xhtml#reprintsus">http://nn.neurology.org/misc/addir.xhtml#reprintsus</a></td>
</tr>
</tbody>
</table>