

Energy drinks as a trigger factor for seizures in paediatric patients: A case report[☆]



Bebidas energéticas como desencadenante de crisis convulsivas en pacientes pediátricos: a propósito de un caso

Dear Editor:

Consumption of energy drinks has increased in recent years, mainly among preadolescents, adolescents, and young adults; 30%-70% of adolescents are frequent consumers.^{1,2} Energy drinks frequently contain high doses of caffeine, combined with such other substances as taurine, guarana, and sugar.³ These beverages have no proven therapeutic effects. On the contrary, consumption of energy drinks is associated with a number of adverse reactions, especially in children and young adults; these include seizures, cardiovascular problems, diabetes, behavioural alterations, and even sudden death.³⁻⁸ Knowing the possible adverse effects of energy drinks and ruling out their consumption is essential for proper differential diagnosis of neurological symptoms and to prevent recurrences and potential worsening of secondary symptoms.

We present the case of an 8-year-old boy with no relevant personal or family history who began to experience paraesthesia around the oral commissure, followed by clonus and commissure deviation, resulting in difficulty speaking and retaining saliva. The patient showed no alterations in the level of consciousness and had no other symptoms. He had experienced similar isolated episodes in the previous 2 months; episode frequency had increased over the previous week, with 2 episodes occurring on the day of the consultation, leading to his visit to the emergency department. Symptoms alternately affected both commissures, with episodes occurring predominantly at night and resolving spontaneously within 5 minutes. The last episode was associated with paresis of the left arm and the right side of the face.

The patient did not present fever or any other symptom of infection, and was not taking any medication. No substance use was reported except for energy drinks, which he consumed on a nearly daily basis. According to the patient's parents, the episodes started when the patient began consuming energy drinks. Consumption had increased in the previous week (500-mL cans of Monster Energy[®], each containing 5 mg caffeine per kg of the patient's weight).

The baseline neurological examination revealed no alterations. Normal results were also obtained from a blood test, including a complete blood count, blood gas anal-

ysis, and biochemical analysis (ions were within normal ranges). A urine analysis tested negative for toxins, and the electrocardiogram showed no alterations. The patient was admitted for observation and to undergo further testing. He experienced no additional episodes during hospitalisation. An electroencephalography (Fig. 1) showed bihemispheric epileptiform activity, predominantly during non-REM sleep, compatible with Rolandic epilepsy. The patient was discharged; discontinuation of energy drink consumption was recommended. He was followed up at the neuropaediatric department, displaying no further episodes in the 2 months after admission. After that period, he began to experience isolated episodes; a brain MRI scan revealed no alterations, and the patient started treatment with oxcarbazepine.

Energy drink consumption is becoming more prevalent among adolescents and young adults. Consumption starts at increasingly younger ages, as in our patient.⁹

The main active components of these beverages are caffeine, taurine, and such herbal supplements as guarana or ginseng. Although data are scarce, several of these substances are thought to be involved in the pathogenesis of seizures. Caffeine, a natural stimulant and a non-selective adenosine receptor antagonist, has proconvulsant effects both in healthy individuals, when administered at high doses, and in caffeine-sensitive patients.^{10,11} Taurine, an essential amino acid, may also have proconvulsant properties as the seizure threshold decreases in cases of sustained interaction between taurine and GABA_A receptors.¹² Guarana is a plant that contains such methylxanthines as caffeine and theophylline.

Although caffeine tolerance varies, most individuals experience toxic effects with doses higher than 200 mg, or 3 mg/kg in children. Caffeine consumption in children and adolescents should not exceed 2.5 mg/kg/day or 100 mg/day, respectively; the maximum recommended dose for adults is 400 mg/day.²

In the United States, 73% of children and adolescents are frequent caffeine consumers.¹³ Adolescents consume a mean of 60-70 mg/day,³ mostly from soft drinks; caffeine obtained from energy drinks has increased in recent years and amounts to up to 10% of total caffeine intake.¹³

Energy drinks contain a mean of 70-80 mg caffeine per can (3 times more than cola); caffeine content may be even higher, as in the energy drink consumed by our patient (Table 1). Caffeine content is not specified on the packaging of many of these beverages. Furthermore, each gram of guarana extract contains 40-80 mg caffeine, but manufacturers are not obliged to specify this additional caffeine content.³ Therefore, a single can of these beverages is very likely to contain more caffeine than the maximum recommended daily dose.¹⁴

Consumption of energy drinks is associated with a wide range of severe adverse reactions,⁷ including first-time seizures,⁶⁻⁸ also in paediatric patients¹⁵; seizures resolve after discontinuation of energy drink consumption. The adverse effects of these beverages may be underestimated, as patients are rarely asked about energy drink consumption. It is also difficult to differentiate between dose-dependent episodes in healthy individuals and episodes triggered by decreased seizure threshold in caffeine-sensitive patients.

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Figure 1 Video-EEG recording revealing epileptiform activity, frequently independent and in the centrotemporal region of both hemispheres, becoming more marked when the patient fell asleep and during non-REM sleep, with high persistence. Centrotemporal epileptiform activity is mixed with slow, irregular activity. Sensitivity: 15 μ V/mm; high-frequency filter: 70 Hz; time constant: 0.3 s.

Although we cannot confirm a causal relationship between consumption of energy drinks and onset of partial seizures in the context of benign childhood epilepsy, these beverages may have triggered the episodes. Identifying the involvement of energy drink consumption in episodes of seizures may be a diagnostic challenge; a high level of clinical suspicion is therefore necessary. Children and adolescents are frequent consumers of these beverages but are not fully aware of the associated risks.

Raising awareness of the adverse effects of these substances among consumers and healthcare professionals is essential. Further studies should aim to determine the safety of energy drink consumption.

References

1. Babu KM, Church RJ, Lewander W. Energy drinks: the new eye-opener for adolescents. *Clin Pediatr Emerg Med*. 2008;9:35–42.
2. Bedi N, Dewan P, Gupta P. Energy drinks: potions of illusion. *Indian Pediatr*. 2014;51:529–33.
3. Seifert SM, Schaechter JL, Hershorin ER, Lipshultz SE. Health effects of energy drinks on children, adolescents, and young adults. *Pediatrics*. 2011;127:511–28.
4. Broderick P, Benjamin AB. Caffeine and psychiatric symptoms: a review. *J Oklahoma State Med Assoc*. 2004;97:538–42.
5. Clauson KA, Shields KM, McQueen CE, Persad N. Safety issues associated with commercially available energy drinks. *J Am Pharm Assoc* (2003). 2008;48:e55–63.
6. Iyadurai SJP, Chung SS. New-onset seizures in adults: possible association with consumption of popular energy drinks. *Epilepsy Behav*. 2007;10:504–8.
7. Calabrò RS, Italiano D, Gervasi G, Bramanti P. Single tonic-clonic seizure after energy drink abuse. *Epilepsy Behav*. 2012;23:384–5.
8. Matuszkiewicz E, Łukasik-Głębocka M, Sommerfeld K, Tezyk A, Zielińska-Psujka B, Zaba C. Energy drinks as a cause of

Table 1 Caffeine content of various widely consumed beverages.

Beverage/brand	Volume	Caffeine content per unit
<i>Tea</i>	125-250 mL	30-60 mg
<i>Standard coffee</i>	100 mL coffee	120 mg
<i>Espresso coffee</i>	30 mL coffee	40-75 mg
<i>Cola drink</i>	330 mL	25-40 mg
<i>Energy drink</i>	200 mL	60 mg
Monster®	500 mL	145 mg
Red Bull®	250 mL	75 mg
Burn®	250 mL	74 mg
Burn Day®	500 mL	92 mg

seizures – real or possible danger? Case report. *Przegląd Lek.* 2015;72:42–4.

9. Reissig CJ, Strain EC, Griffiths RR. Caffeinated energy drinks – a growing problem. *Drug Alcohol Depend.* 2009;99:1–10.
10. Chu NS. Caffeine-induced and aminophylline-induced seizures. *Epilepsia.* 1981;22:85–94.
11. Kaufman KR, Sachdeo RC. Caffeinated beverages and decreased seizure control. *Seizure.* 2003;12:519–21.
12. El Idrissi A, Trenkner E. Taurine as a modulator of excitatory and inhibitory neurotransmission. *Neurochem Res.* 2004;29:189–97.
13. Branum M. Trends in caffeine intake among US children and adolescents. *Pediatrics.* 2014;133:386–93.
14. Malinauskas BM, Aeby VG, Overton RF, Carpenter-Aeby T, Barber-Heidal K. A survey of energy drink consumption patterns among college students. *Nutr J.* 2007;6:35.
15. Babu KM, Zuckerman MD, Cherkes JK, Hack JB. First-onset seizure after use of an energy drink [corrected]. *Pediatr Emerg Care.* 2011;27:539–40.

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Peripheral nerve stimulation: An effective treatment alternative for refractory pain[☆]



Estimulación de nervio periférico: una alternativa terapéutica eficaz para el dolor refractario

Dear Editor:

Chronic neuropathic pain can be highly refractory to both conventional and percutaneous analgesic treatment. Clinical management of these patients is a challenge due to the limiting nature of their pain, loss of confidence after successive consultations with different specialists, and low expectations of improvement given the apparent lack of effective treatment options. We should note the availability of an alternative treatment which merits greater recognition than it currently receives, despite advances made in recent years.

We present the case of a 43-year-old patient diagnosed with the Samter triad with severe nasal polyps, complicated by recurrent sinusitis, which she had experienced since childhood. The patient had undergone 17 procedures at her local hospital's otorhinolaryngology department, including endoscopic procedures and frontal osteoplasty, which was reviewed several times by the surgical team following

onset of inflammation and local pain. She was referred to our clinic due to a 2-year history of chronic, disabling pain involving the supraorbital notch and the right lateral frontal region. The patient reported constant, burning pain. The physical examination revealed the Tinel sign and areas of primary mechanical hyperalgesia and allodynia; the patient was diagnosed with secondary supraorbital neuralgia. The patient's symptoms were refractory to both pharmacological and percutaneous treatment (radiofrequency supraorbital nerve block). The patient scored the maximum possible on the pain disability index (PDI) and visual analogue scale (VAS). The presurgical psychiatric evaluation only indicated an adaptive emotional response to pain. A cylindrical electrode (Precision[®] SC-2352-50, Boston Scientific) was implanted into the right frontal region under local anaesthesia (Fig. 1). During the procedure, the patient reported paraesthesia in the area receiving stimulation; 7 days later, pain had improved significantly (PDI, 32; VAS, 4). A generator (Precision[®] SC-1110-02, Boston Scientific) was subsequently implanted under general anaesthesia. Pain had resolved completely at 6 months (PDI, 0; VAS, 0) and continued to be absent at one year of follow-up.

Peripheral nerve field stimulation (PNFS) has for decades been a therapeutic alternative meriting consideration for use in patients with various types of refractory pain. This technique is more easily reversed and less invasive than other surgical options,¹ and has been used successfully in patients with neuropathic pain (postherpetic neuralgia, trigeminal neuralgia, occipital neuralgia, traumatic neuralgia), complex regional pain syndrome, axial back pain, headache, and even musculoskeletal pain, fibromyalgia, and visceral pain.² Clinical use of the technique, based on the gate control theory of pain, was first described in 1967 by Patrick D. Wall and William H. Sweet,³ who had confirmed its effect by testing it on themselves.⁴ However, it should be

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